Propaedeutics of Internal Medicine

Module: Respiratory Diseases

By Kristian Debretseni

TOPIC: RESPIRATORY SYSTEM

Title of the seminar: Common and concerning symptoms (chest pain, dyspnea, wheezing, cough). Examination techniques (palpation, percussion)

Name of the tutor: Kristian Debretseni
Duration: 90 min
Audience: 3rd course students

Goals:
General: To master skills in history-taking in patients with pathology of the respiratory system, in performing inspection, palpation and percussion of the chest.
Special goals:

- Knowledge: to learn the main symptoms in respiratory patients and the basics of history-taking.
- Practical skills: physical examination, chest palpation, tactile fremitus, chest percussion
- Changing attitude: History-taking in patients is the basic, informative method of examination. It is the first interaction between the patient and doctor. If it has been done correctly, it provides valuable information that cannot be obtained in any other way. It allows recognizing symptoms of disease and points, in most cases, the diagnostic search on the right way.

Methodology of the seminar:

5 minutes Short introduction

5 minutes Ice breaker

20 minutes Theory presentation (Power Point presentation)

35 minutes Interactive
Use of “OPEN” QUESTIONS and ROLE-PLAY TEACHING METHODS in the “history-taking” part of the class.
Practical training of palpation and percussion (on patients, on each other)

15 minutes Control – verbal interview

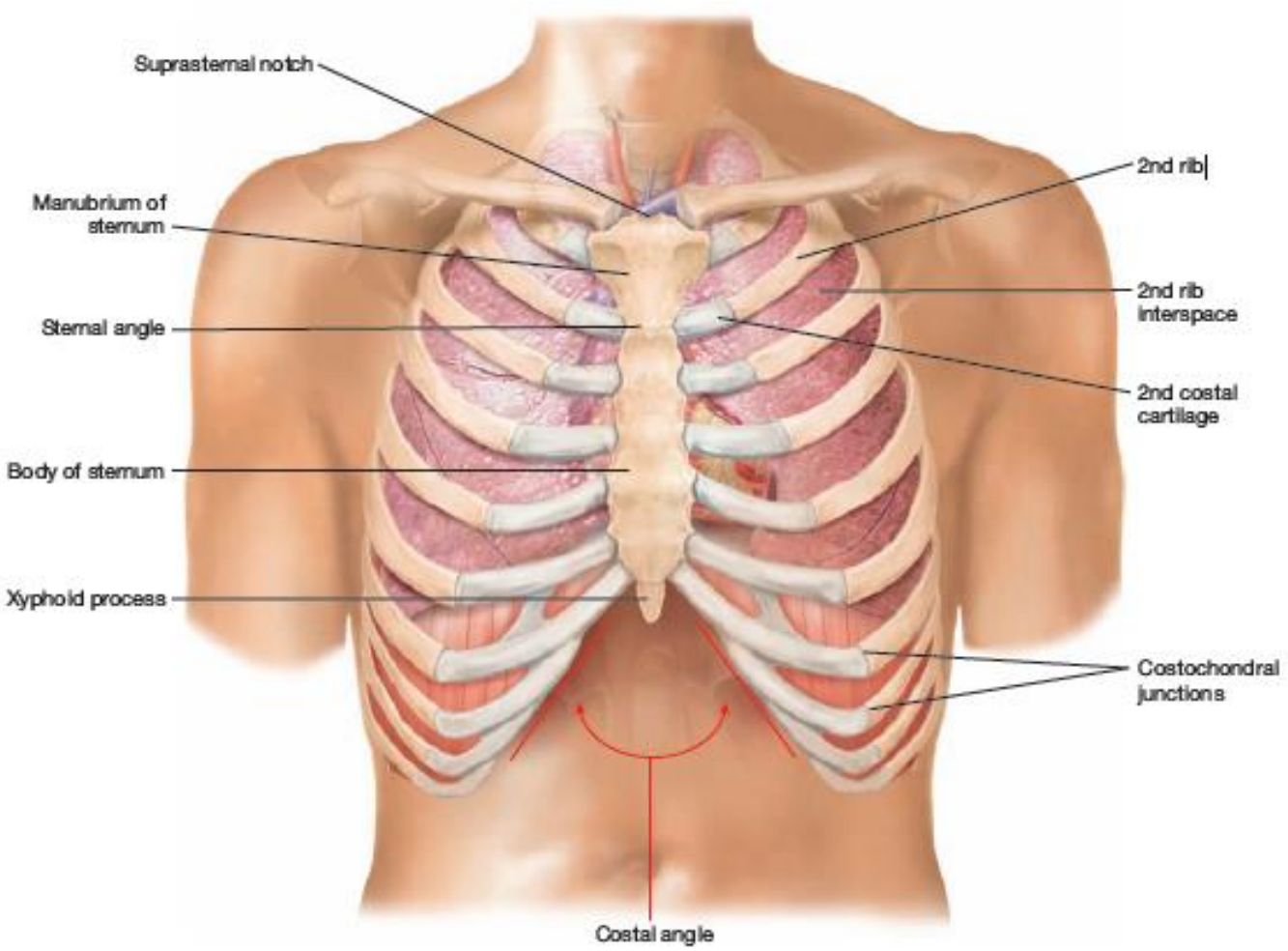
10 minutes Feedback from each participant: take home notes: “What was new for me today? What did I learn/practice? What will I introduce into my future practice?”

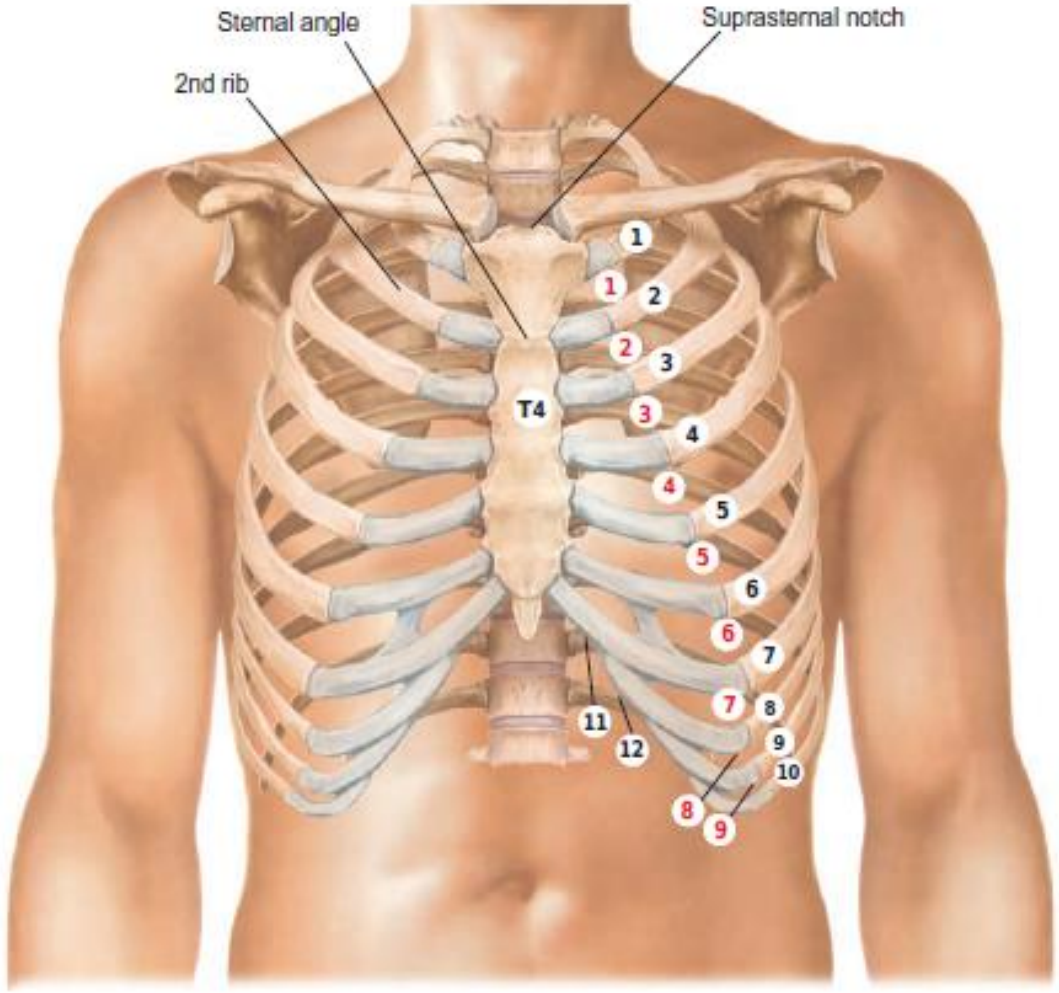
Results: we expect each participant:

- Master the basics of history-taking in respiratory patients
- Practice to perform physical examination, chest palpation, tactile fremitus, chest percussion.

Anatomy and Physiology

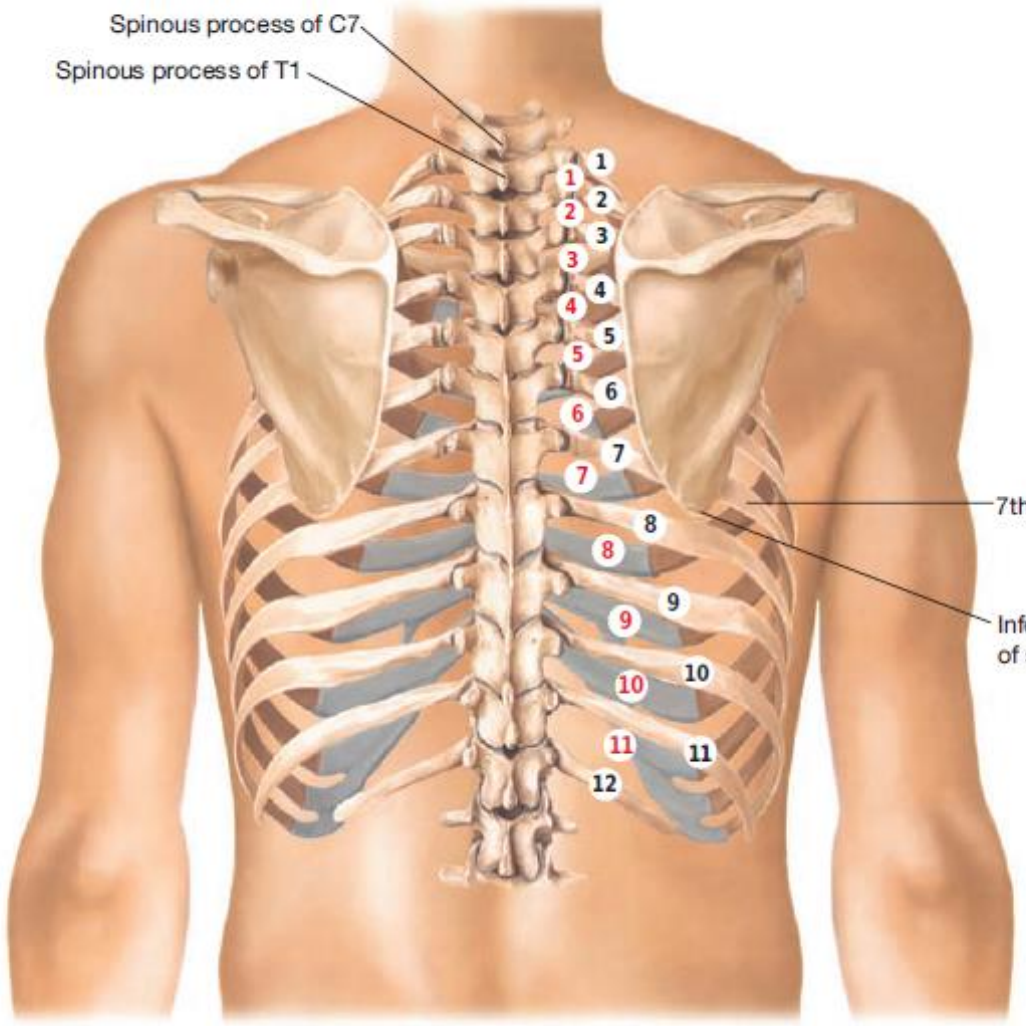
Study the *anatomy of the chest wall*, identifying the structures illustrated. Note that the interspace between two ribs is numbered by the rib above it.



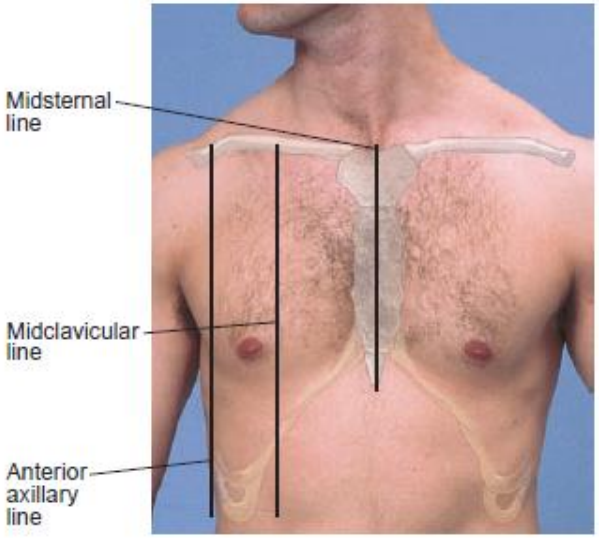


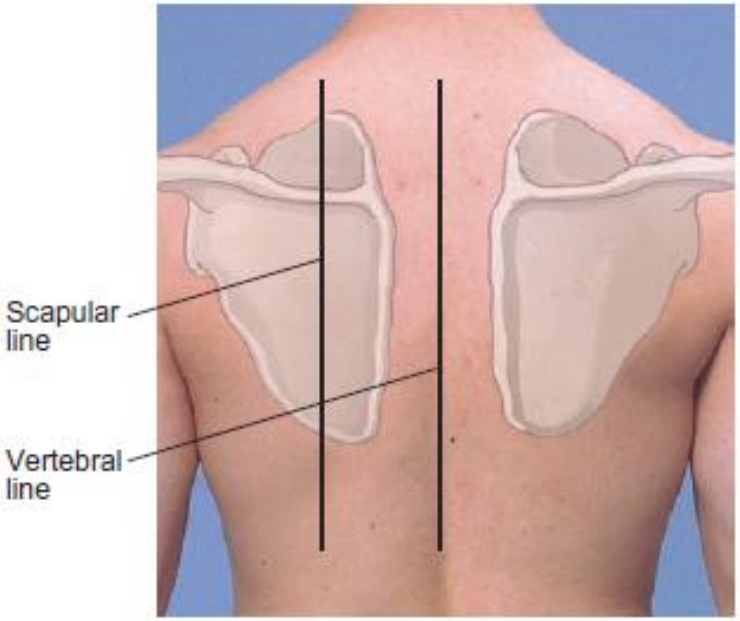
Note special landmarks: 2nd intercostal space for needle insertion for tension pneumothorax; 4th intercostal space for chest tube insertion; T4 for lower margin of endotracheal tube on a chest x-ray.

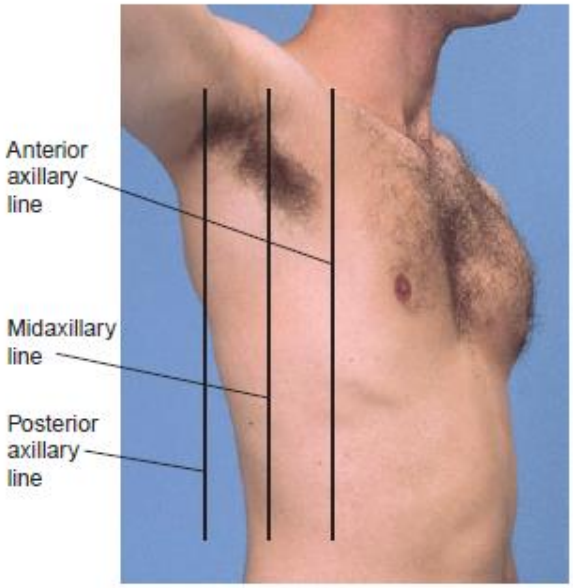
Neurovascular structures run under each rib, so needles and tubes should be placed just superior to the rib margins.



Note the T7–8 interspace as a landmark for thoracentesis.


Midsternal line
Midclavicular line
Anterior axillary line


Scapular line
Vertebral line


Anterior axillary line
Midaxillary line
Posterior axillary line

Patients complaints

1. Chest Pain. To assess this symptom, you must pursue a dual investigation of both thoracic and cardiac causes.

- “Do you have any discomfort or unpleasant feelings in your chest?”

Sources of Chest Pain and Related Causes

- The myocardium *Angina pectoris, myocardial infarction, myocarditis*
- The pericardium *Pericarditis*
- The aorta *Dissecting aortic aneurysm*
- The trachea and large bronchi *Bronchitis*
- The parietal pleura *Pericarditis, pneumonia, pneumothorax, pleural effusion, pulmonary embolus*
- The chest wall, including the musculoskeletal system and skin *Costochondritis, herpes zoster*
- The esophagus *Reflux esophagitis, esophageal spasm, esophageal tear*
- Extrathoracic structures such as the neck, gallbladder, and stomach *Cervical arthritis, biliary colic, gastritis*

2. Shortness of Breath (Dyspnea) and Wheezing.

“Have you had any difficulty breathing?”

3. Cough.

- For complaints of cough, a thorough assessment is in order. Duration of the cough is important: is the cough *acute, lasting less than 3 weeks; subacute,* lasting 3 to 8 weeks; or *chronic, more than 8 weeks?*

4. Hemoptysis.

Hemoptysis is the coughing up of blood from the lungs

Posterior chest examination

Inspection

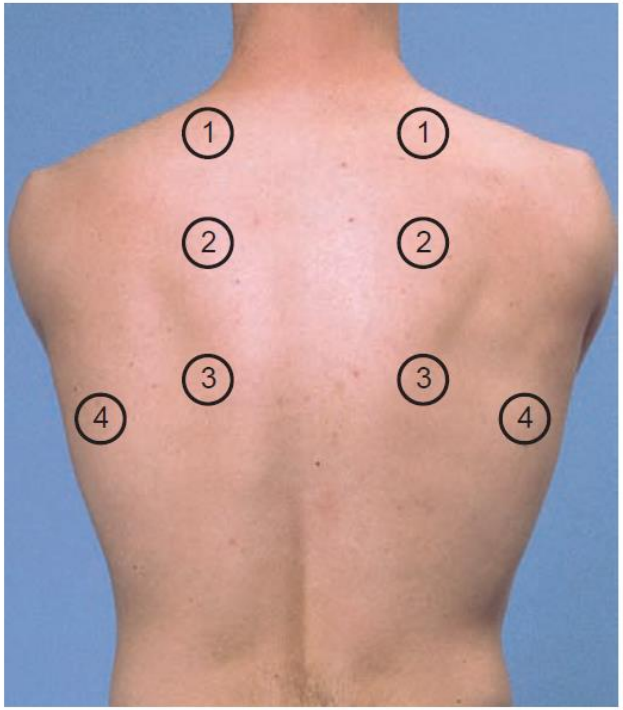
From a midline position behind the patient, note the *shape of the chest and how the chest moves, including:*

- Deformities or asymmetry in chest expansion
- Abnormal retraction of the interspaces during inspiration. Retraction is most apparent in the lower interspaces.
- Impaired respiratory movement on one or both sides or a unilateral lag (or delay) in movement.

- Palpation
- As you palpate the chest, focus on areas of tenderness and abnormalities in the overlying skin, respiratory expansion, and fremitus.
- Identify tender areas. Carefully palpate any area where pain has been reported or where lesions or bruises are evident.
- Assess any visible abnormalities such as masses or sinus tracts (blind, inflammatory, tubelike structures opening onto the skin).

Palpate and compare symmetric areas of the lungs in the pattern shown in the photograph. Identify and locate any areas of increased, decreased, or absent fremitus.

Fremitus refers to the palpable vibrations transmitted through the bronchopulmonary tree to the chest wall as the patient is speaking.



LOCATIONS FOR FEELING FREMITUS

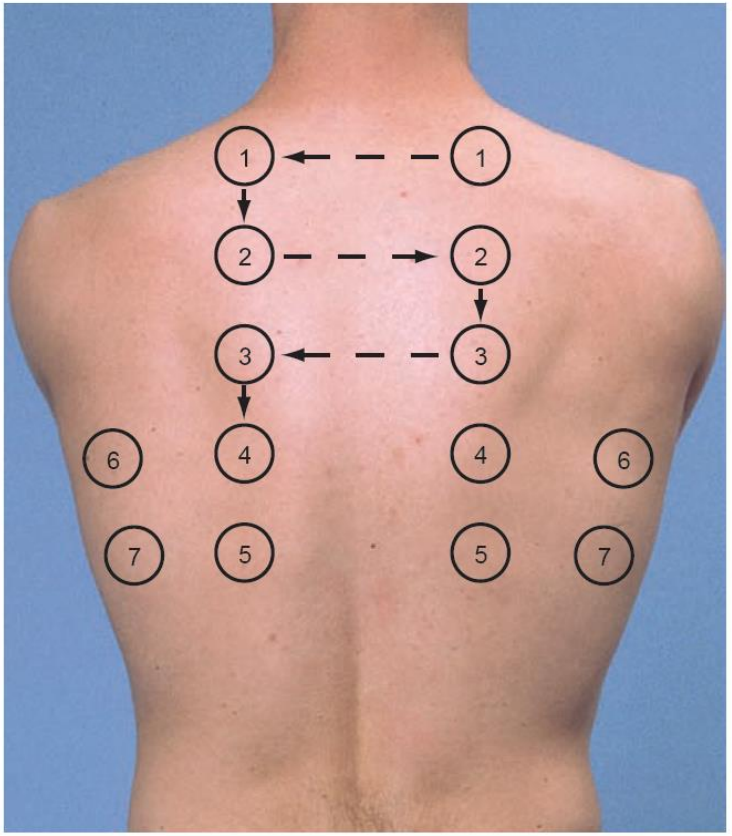
Percussion

- Percussion is one of the most important techniques of physical examination.
- Percussion sets the chest wall and underlying tissues in motion, producing audible sound and palpable vibrations.
- Percussion helps you establishwhether the underlying tissues are air-filled, fluid-filled, or solid.

Percussion Notes and Their Characteristics

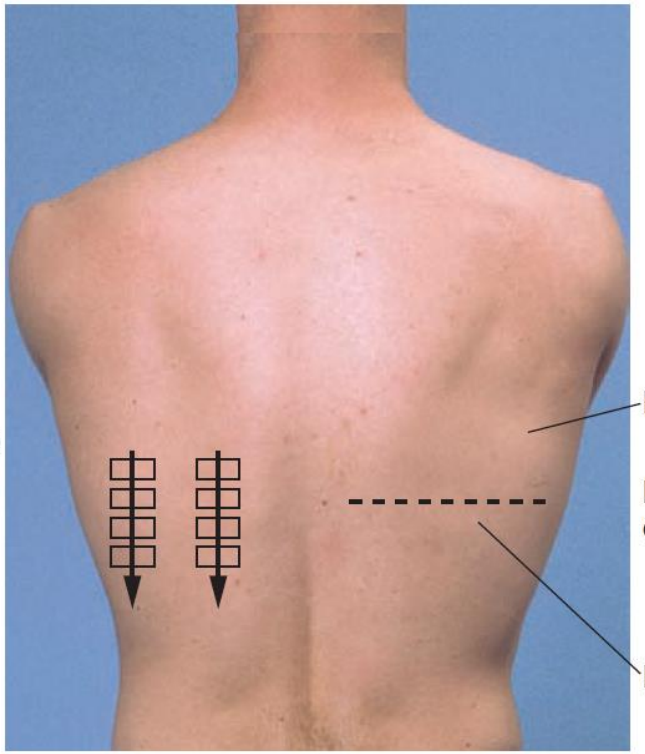
	Relative Intensity	Relative Pitch	Relative Duration	Example of Location	Pathologic Examples
Flat	Soft	High	Short	Thigh	Large pleural effusion
Dull	Medium	Medium	Medium	Liver	Lobar pneumonia
Resonant	Loud	Low	Long	Healthy lung	Simple chronic bronchitis
Hyperresonant	Very loud	Lower	Longer	Usually none	COPD, pneumothorax
Tympanitic	Loud	High*	Longer	Gastric air bubble or puffed-out cheek	Large pneumothorax

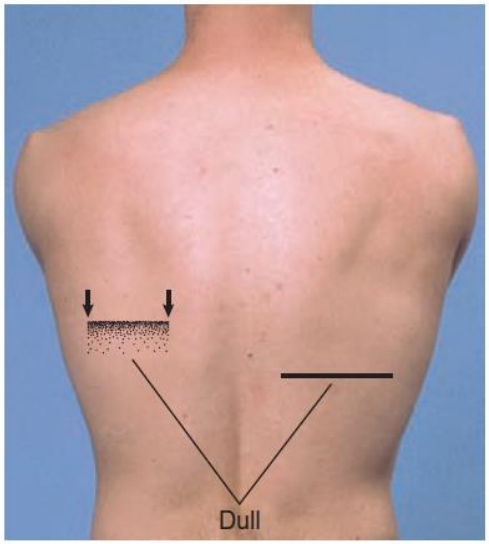
Percuss one side of the chest and then the other at each level in a ladderlike pattern, as shown by the numbers below.



"LADDER" PATTERN FOR PERCUSSION AND AUSCULTATION

Identify the descent of the diaphragm, or diaphragmatic excursion. First, determine the level of diaphragmatic dullness during quiet respiration.





An abnormally high level suggests *pleural effusion*, or a high diaphragm as in *atelectasis* or *phrenic nerve paralysis*.

Estimate the extent of diaphragmatic excursion by determining the distance between the level of dullness on full expiration and the level of dullness on full inspiration, normally about 3 to 5.5 cm

Anterior chest examination

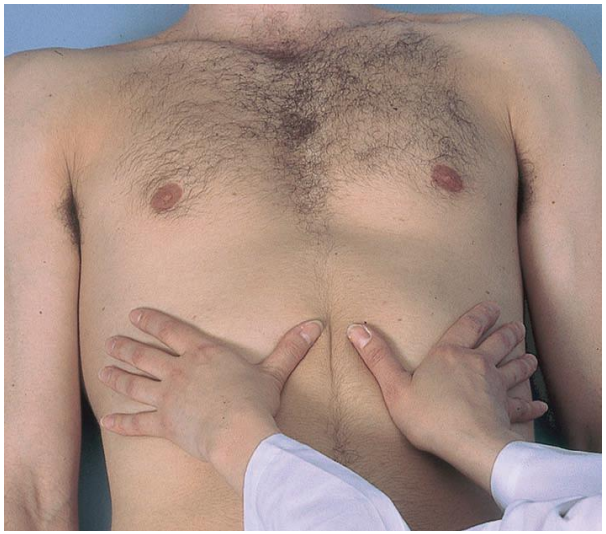
Inspection

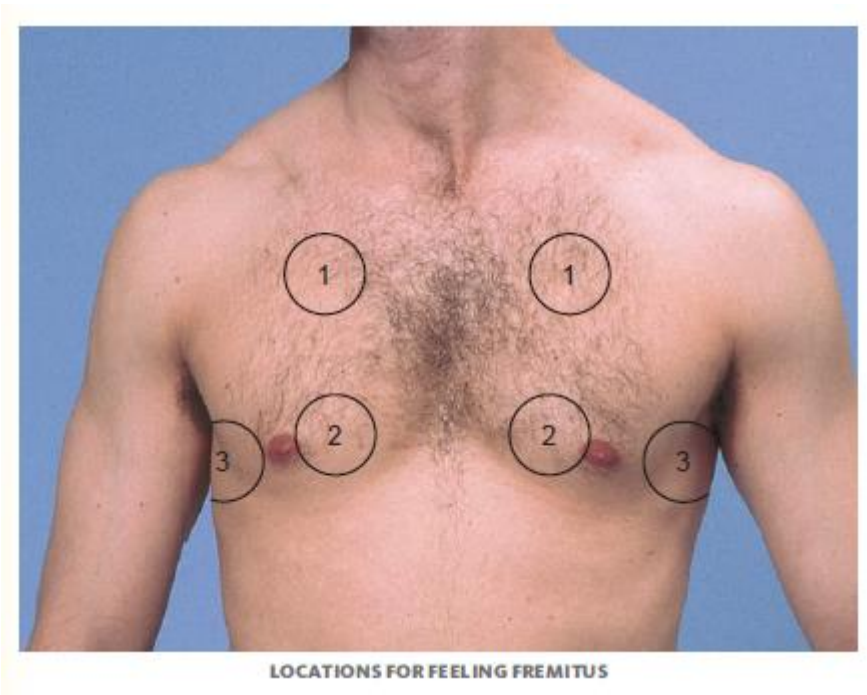
- Observe the shape of the patient's chest and the movement of the chest wall.

Note:

- Deformities or asymmetry
- Abnormal retraction of the lower interspaces during inspiration.
- Local lag or impairment in respiratory movement

Assessment of tactile fremitus.
Compare both sides of the chest, using the ball or ulnar surface of your hand. Fremitus is usually decreased or absent over the precordium.

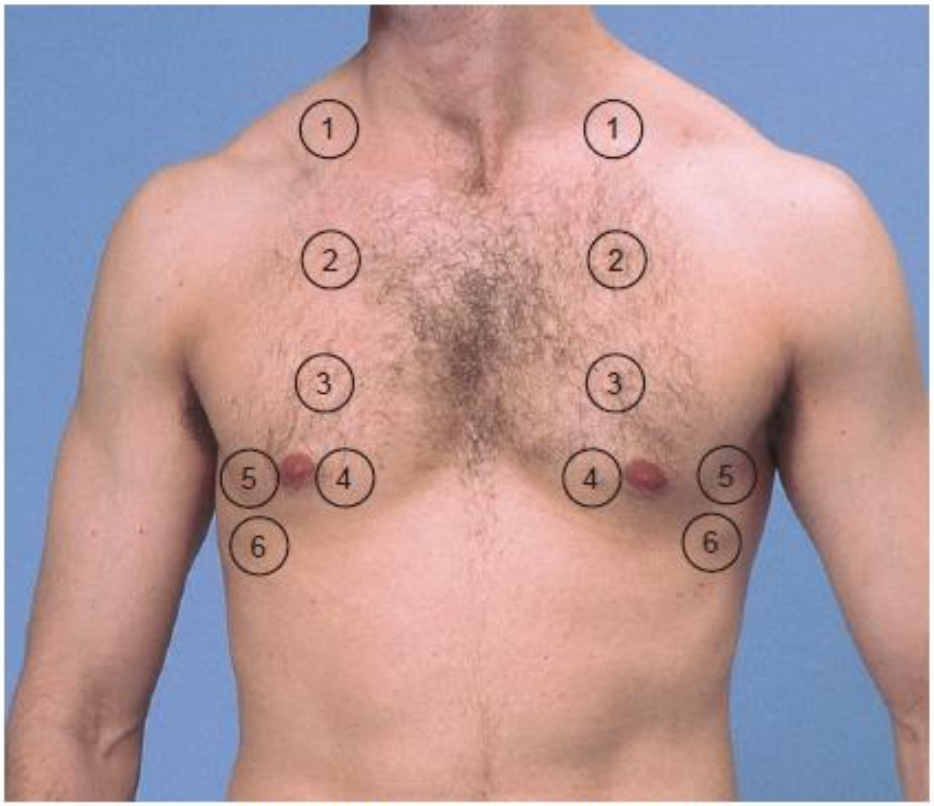




LOCATIONS FOR FEELING FREMITUS

Percussion

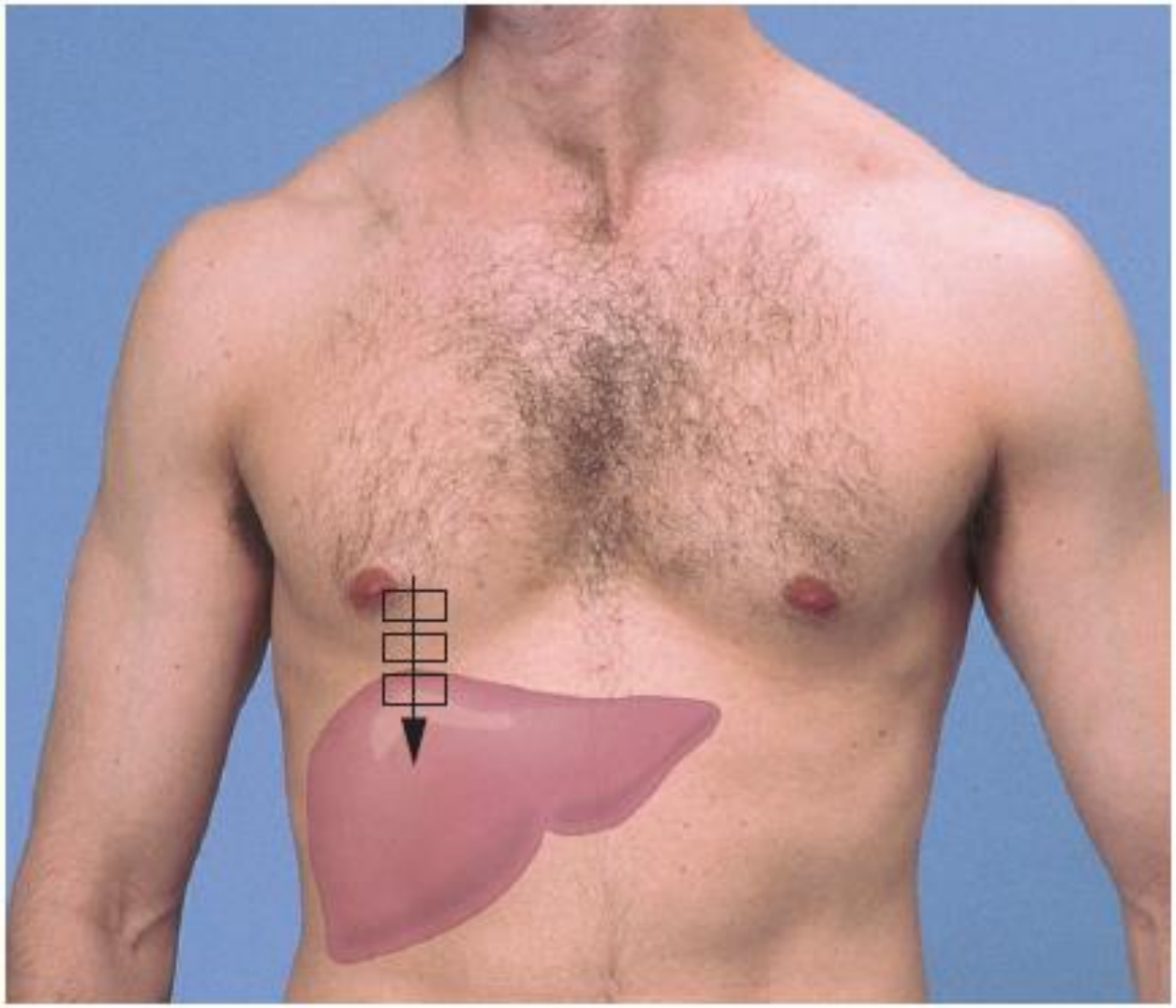
Percuss the anterior and lateral chest, again comparing both sides. The heart normally produces an area of dullness to the left of the sternum from the 3rd to the 5th interspaces. Percuss the left lung lateral to the area of dullness.



LOCATIONS FOR PERCUSSION AND AUSCULTATION

With your pleximeter finger above and parallel to the expected upper border of liver dullness, percuss in progressive steps downward in the right midclavicular line. Identify the upper border of liver dullness. Later, during the abdominal examination, you will use this method to estimate the size of the liver.

As you percuss down the chest on the left, the resonance of normal lung usually changes to the tympany of the gastric air bubble.



TOPIC: RESPIRATORY SYSTEM

Title of the seminar: Auscultation of lungs. Characteristic of breath sounds.

Name of the tutor: Kristian Debretseni
Duration: 90 min
Audience: 3rd course students

Goals:
General: To master skills in performing auscultation of lungs.
Special goals:

- Knowledge: to learn physical grounds of auscultation, mechanisms of production of normal and abnormal respiratory sounds, to learn the main types of normal and pathological breath sounds.

Learn the classification of the adventitious breath sounds (rales, crepitation and pleural friction rub).
Learn causes and mechanisms of the dry and wet rales producing and their types.

- Practical skills: to practice auscultation of lungs
- Changing attitude: Auscultation of the chest is one of the basic, informative methods of examination of the respiratory patients. It allows hearing normal and pathological changed sounds produced during act of breathing. Most of the respiratory diseases are accompanied with certain auscultation phenomena: changes of main sounds and appearance of the adventitious lung sounds, their determining is one of clues at the diagnostic process of the respiratory patients.

Methodology of the seminar:

5 minutes Short introduction

5 minutes Ice breaker

15 minutes Theory presentation (Power Point presentation)

40 minutes Interactive
Practical training of lungs auscultation (on patients, on each other)

15 minutes Control – verbal interview, audio samples of respirtatory sounds

10 minutes Feedback from each participant: take home notes: “What was new for me today? What did I learn/practice? What will I introduce into my future practice?”

Results: we expect each participant:

- Master the basic knowledge about the main breath sounds.
- Practice to perform lungs auscultation.

Auscultation

Auscultation is the most important examination technique for assessing air flow through the tracheobronchial tree. Together with percussion, it also helps the clinician assess the condition of the surrounding lungs and pleural space.

Auscultation involves

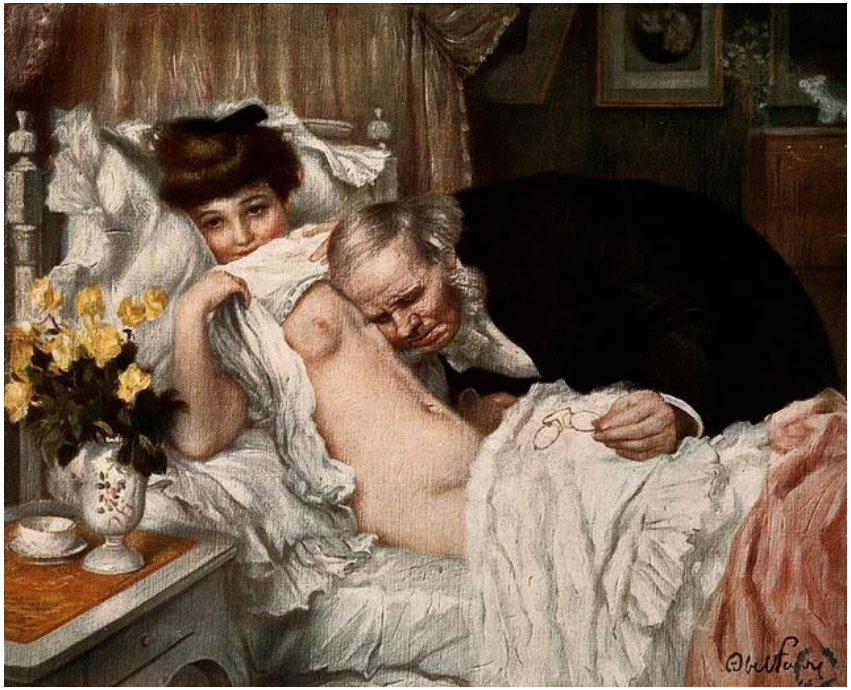
(1) listening to the sounds generated by breathing,

(2) listening for any adventitious (added) sounds,

and (3) if abnormalities are suspected, listening to the sounds of the patient's spoken or whispered voice as they are transmitted through the chest wall.

Auscultation

Auscultation, as an objective method of patient's examination developed by a French scientist Rene Laennec in 1816 and introduced in medical practice in 1819. Rene Laennek described sound phenomena while listening respiratory tract and mechanisms of their formation. He introduced basic terms: vesicular and bronchial breathing, rales, pleural rub.



Indirect auscultation

“In 1816, I was consulted by a young woman laboring under general symptoms of diseased heart, and in whose case percussion and the application of the hand were of little avail on account of the great degree of fatness. The other method just mentioned [direct auscultation] being rendered inadmissible by the age and sex of the patient. I rolled a quire of paper into a kind of cylinder and applied one end of it to the region of the heart and the other to my ear, and was not a little surprised and pleased to find that I could thereby perceive the action of the heart in a manner much more clear and distinct than I had ever been able to do by the immediate application of my ear.”

Indirect auscultation

Further indirect method of auscultation improved Pierre Piorri, Academician FG Yanovsky, and Professor NF Filatov first recommends binaural stethoscope while Y.Shkoda introduces the practice phonendoscope. However, they described the physical basis of the lungs and heart auscultation. The advantage and convenience of indirect auscultation, made it the most widely used and informative. Direct auscultation is currently used mostly at the hearing infants, or, sometimes, pleural rub.

Further development of auscultation - a graphical recording of sound effects – phonography. The first such record of the heart sounds was made in 1894.

Auscultation rules

1. The chest of the patient should be bare as the shuffling of the clothes may mix with the sound.

2. The examination room should be warm and silent.

3. The stethoscope should be applied to the body tightly, with the whole edge of the funnel Sliding of an untightly applied stethoscope may also produce accessory sounds.

4. It is not necessary to use excessive force to apply the stethoscope. It can cause pain and hinder vibrations of the chest wall in the studied area, in this way weakening conduction of the vibrations from the underlying tissues to the air and the ear.

5. It is not necessary to hold the tube of the stethoscope with the hand as the minute motions of the holding fingers can add sounds.

6. It is necessary to use one and the same stethoscope. Various stethoscopes and phonendoscopes are available for indirect auscultation. This method allows avoiding some disadvantages of direct auscultation mentioned above, besides the instrument aids conduction of the sound to the physician's ear and limits the studied area.

A few additional things worth noting

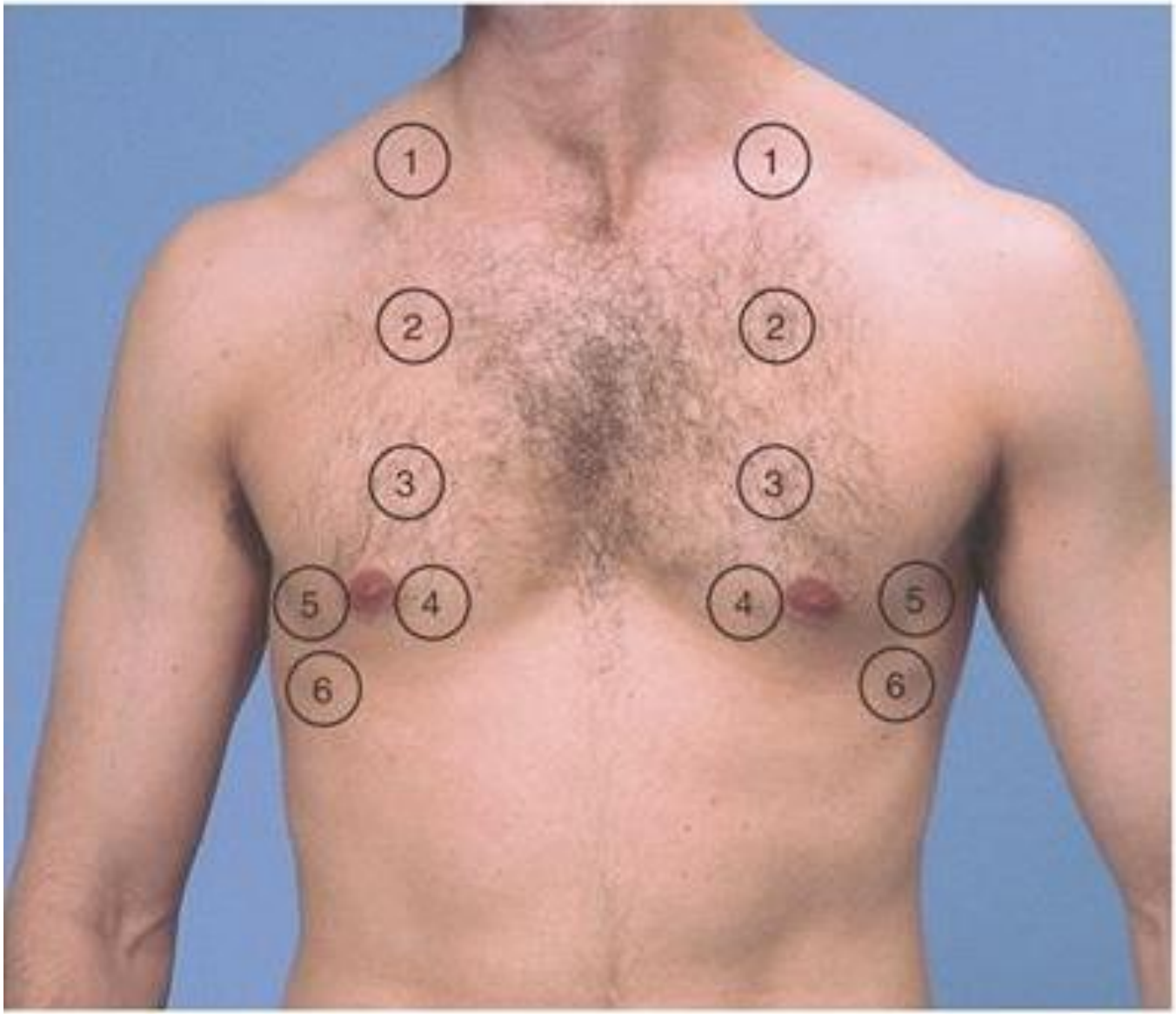
1. Don't get in the habit of performing auscultation through clothing.

2. Ask the patient to take slow, deep breaths through their mouths while you are performing your exam. This forces the patient to move greater volumes of air with each breath, increasing the duration, intensity, and thus detectability of any abnormal breath sounds that might be present.

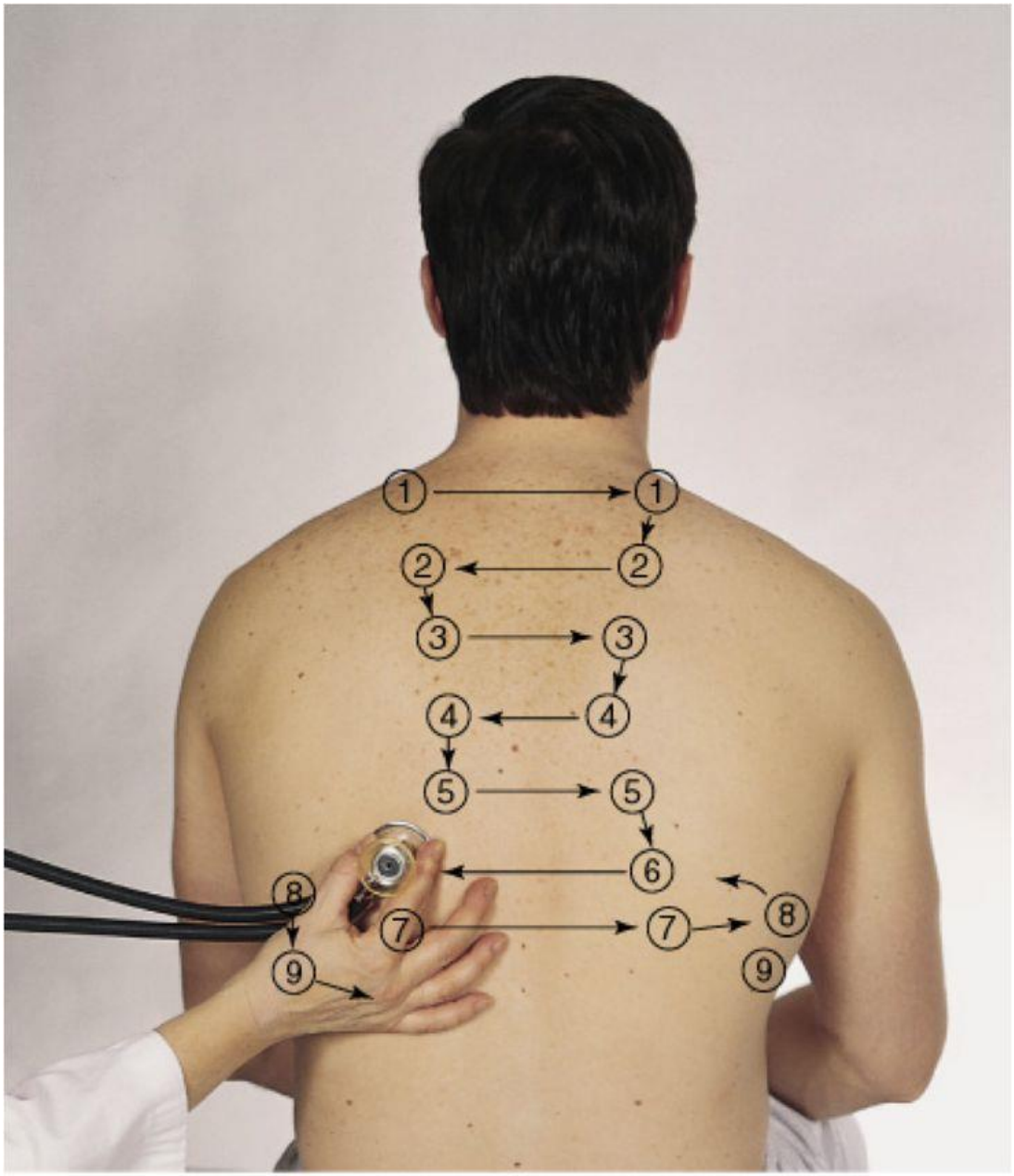
3. Sometimes it's helpful to have the patient cough a few times prior to beginning auscultation. This clears airway secretions and opens small atelectatic (i.e. collapsed) areas at the lung bases.

4. If the patient cannot sit up (e.g. in cases of neurologic disease, post-operative states, etc.), auscultation can be performed while the patient is lying on their side. Get help if the patient is unable to move on their own. In cases where even this cannot be accomplished, a minimal examination can be performed by listening laterally/posteriorly as the patient remains supine.

5. Requesting that the patient exhale forcibly will occasionally help to accentuate abnormal breath sounds (in particular, wheezing) that might not be heard when they are breathing at normal flow rates.



Locations for Percussion and Auscultation



Characteristics of Breath Sounds

	Duration of Sounds	Intensity of Expiratory Sound	Pitch of Expiratory Sound	Locations Where Heard Normally
Vesicular* 	Inspiratory sounds last longer than expiratory sounds.	Soft	Relatively low	Over most of both lungs
Broncho-vesicular 	Inspiratory and expiratory sounds are about equal.	Intermediate	Intermediate	Often in the 1st and 2nd interspaces anteriorly and between the scapulae
Bronchial 	Expiratory sounds last longer than inspiratory ones.	Loud	Relatively high	Over the manubrium, (larger proximal airways)
Tracheal 	Inspiratory and expiratory sounds are about equal.	Very loud	Relatively high	Over the trachea in the neck

*The thickness of the bars indicates intensity; the steeper their incline, the higher the pitch.

Main breath sounds

- Main breath sounds may be divided into two types: vesicular and bronchial breath sounds.

When listening over the larynx (lower portion of the neck), trachea and large bronchi (upper portion of the chest), respiratory sound resembling "h" sound is heard, expiration is louder and longer than inspiration. This sound is produced in the larynx when the air passes trough the fissure of the glottis due to air circulation above the vocal cords at breathing in and under the vocal cords at breathing out. As the fissure of the glottis is narrower at breathing out than at breathing in, the sound produced will be rougher and longer. This is the so-called laryngotracheal or **bronchial breath** sound (respiration).

Main breath sounds

The sound over the rest of the chest is completely different. It is a soft, blowing sound resembling "f' sound. This sound is stronger and longer on breathing in and weaker on breathing out. At the beginning of inspiration, in its first third, this is weak and poorly heard, later it becomes stronger and weakens with the beginning of breathing out and is heard only in the first third of expiration. This respiratory sound is called **vesicular breath** sound (respiration). Vesicular respiration is produced in the lung parenchyma when the air enters the alveoli and their walls get strained.

Normal Breath Sounds

- Created by turbulent air flow
- Inspiration
  - Air moves to smaller airways hitting walls
  - More turbulence, Increased sound
- Expiration
  - Air moves toward larger airways
  - Less turbulence, Decreased sound
- Normal breath sounds
  - Loudest during inspiration, softest during expiration

Normal Breath Sounds

- Tracheal
  - Very loud, high pitched sound
  - Inspiratory = Expiratory sound duration
  - Heard over trachea
- Bronchial
  - Loud, high pitched sound
  - Expiratory sounds > Inspiratory sounds
  - Heard over manubrium of sternum
  - If heard in any other location suggestive of consolidation

Normal Breath Sounds

- Bronchovesicular
  - Intermediate intensity, intermediate pitch
  - Inspiratory = Expiratory sound duration
  - Heard best 1st and 2nd ICS anteriorly, and between scapula posteriorly
  - If heard in any other location suggestive of consolidation
- Vesicular
  - Soft, low pitched sound
  - Inspiratory > Expiratory sounds
  - Major normal BS, heard over most of lungs

Adventitious Breath Sounds

- Crackles (Rales, Crepitation)
  - Discontinuous, intermittent, nonmusical, brief sounds
  - Heard more commonly with inspiration
  - Classified as fine or coarse
  - Normal at anterior lung bases
    - Maximal expiration
    - Prolonged recumbency
  - Crackles caused by air moving through secretions and collapsed alveoli
  - Associated conditions
    - pulmonary edema, early CHF, PNA

Crepitation

- *Crepitation* is an auscultation phenomenon that develops in the alveoli. It is close to the moist rales. It looks like a fine soft crack appearing when a small bundle of hairs is smoothed out above the ear. Crepitation appears when alveolar walls are imbued with exudates or transudes. In the phase of expiration the walls adhere, in the phase of inspiration they depart at the height of the maneuver. Therefore crepitation is heard at the height of inspiration.
- Crepitation suggests the presence of the changes in the alveoli and involvement of the lung tissue itself. Crepitation is heard at inflammation of the lung tissue (initial and final stages of lobular pneumonia), lung atelectasis, congestion, lung infarction.

Adventitious Breath Sounds

- Wheeze
  - Continuous, high pitched, musical sound, longer than crackles
  - Hissing quality, heard > with expiration, however, can be heard on inspiration
  - Produced when air flows through narrowed airways
  - Associated conditions
    - asthma, COPD

Adventitious Breath Sounds

- Stridor
  - Inspiratory musical wheeze
  - Loudest over trachea
  - Suggests obstructed trachea or larynx
  - Medical emergency requiring immediate attention
  - Associated condition
    - inhaled foreign body

Adventitious Breath Sounds

- Pleural Rub
  - Discontinuous or continuous brushing sounds
  - Heard during both inspiratory and expiratory phases
  - Occurs when pleural surfaces are inflamed and rub against each other
  - Associated conditions
    - pleural effusion, PTX

Pleural Rub

- Normal parietal and visceral pleura glide smoothly during respiration.
- If the pleura is roughened due to any reason, a scratching, grating sound, related to respiration is heard.
- You can hear the sound by compressing harder with the stethoscope and making the patient take deep breaths.

TOPIC: RESPIRATORY SYSTEM

Title of the seminar: Acute pulmonary embolism, pneumothorax, COVID-19

Name of the tutor: Kristian Debretseni

Duration: 90 min

Audience: 3rd course students

Goals:

General: To master skills in diagnostics of pulmonary embolism, pneumothorax, COVID-19.

Special goals:

- Knowledge: Study the main symptoms, signs and risk factors of pulmonary embolism, pneumothorax, COVID-19 and main diagnostic methods.
- Practical skills: history-taking in patients with life-threatening and infectious respiratory diseases.
- Changing attitude: Pulmonary embolism is a blockage in one of the pulmonary arteries in the lungs. Because the clots block blood flow to the lungs, pulmonary embolism can be life-threatening.

Methodology of the seminar:

5 minutes Short introduction

5 minutes Ice breaker

20 minutes Theory presentation (Power Point presentation)

30 minutes Interactive
Work in pairs (patient-doctor)
Practical training of lungs auscultation (on patients, on each other)

15 minutes Control

15 minutes Feedback from each participant: take home notes: “What was new for me today? What did I learn/practice? What will I introduce into my future practice?”

Results: we expect each participant:

- Master the basic knowledge about acute pulmonary embolism, pneumothorax, COVID-19.
- Practice to perform lungs auscultation.

Acute Pulmonary Embolism

Pulmonary embolism (**PE**) is a blockage of an artery in the lungs by a substance that has moved from elsewhere in the body through the bloodstream (embolism).

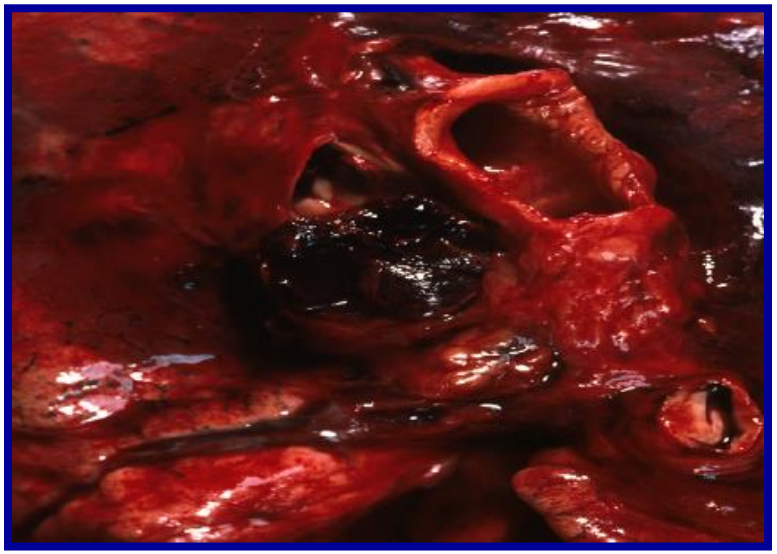
PE usually results from a blood clot in the leg that travels to the lung.

The risk of blood clots is increased by cancer, prolonged bed rest, smoking, stroke, certain genetic conditions, estrogen-based medication, pregnancy, obesity, and after some types of surgery.

A small proportion of cases are due to the embolization of air, fat, or amniotic fluid.

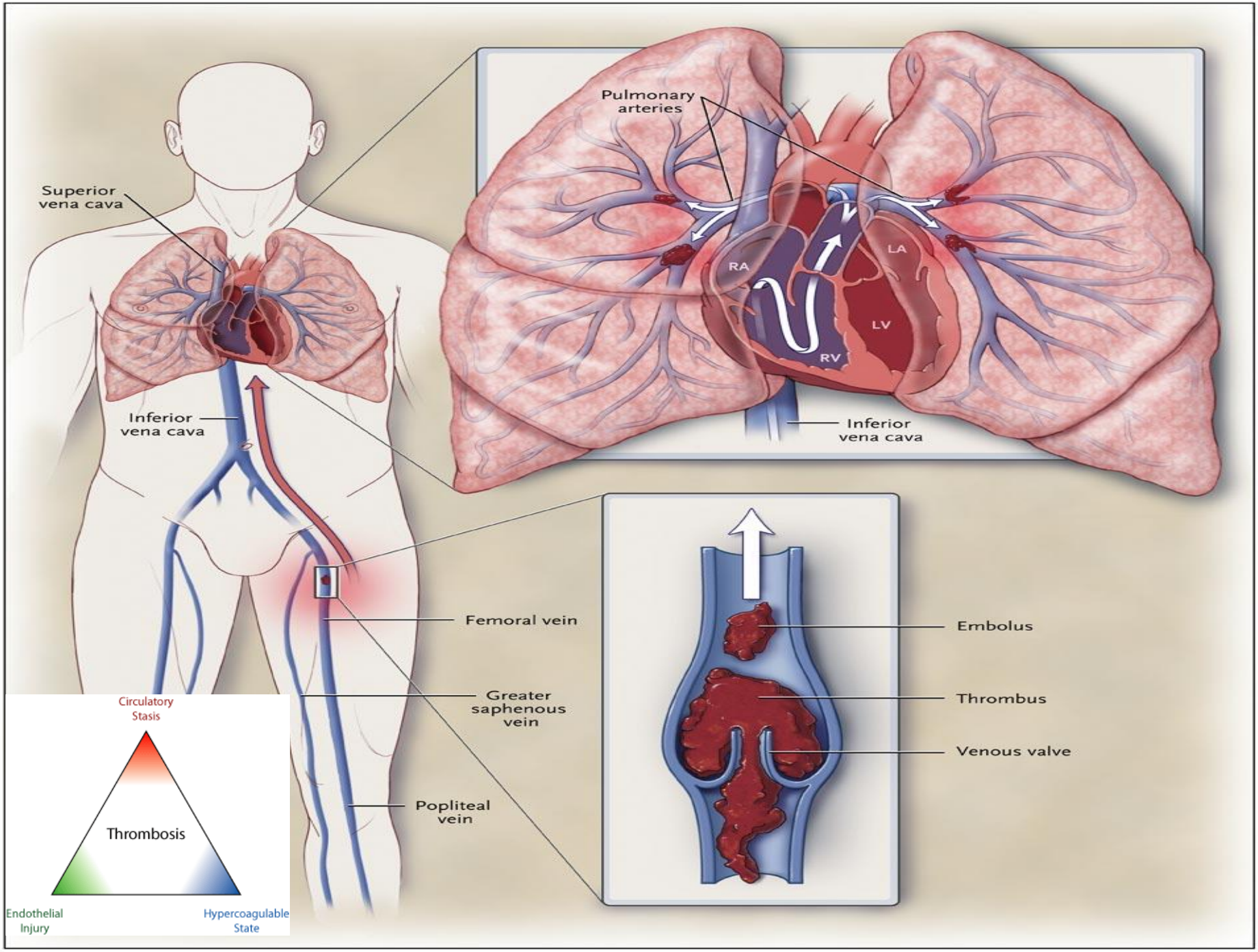
Why care?

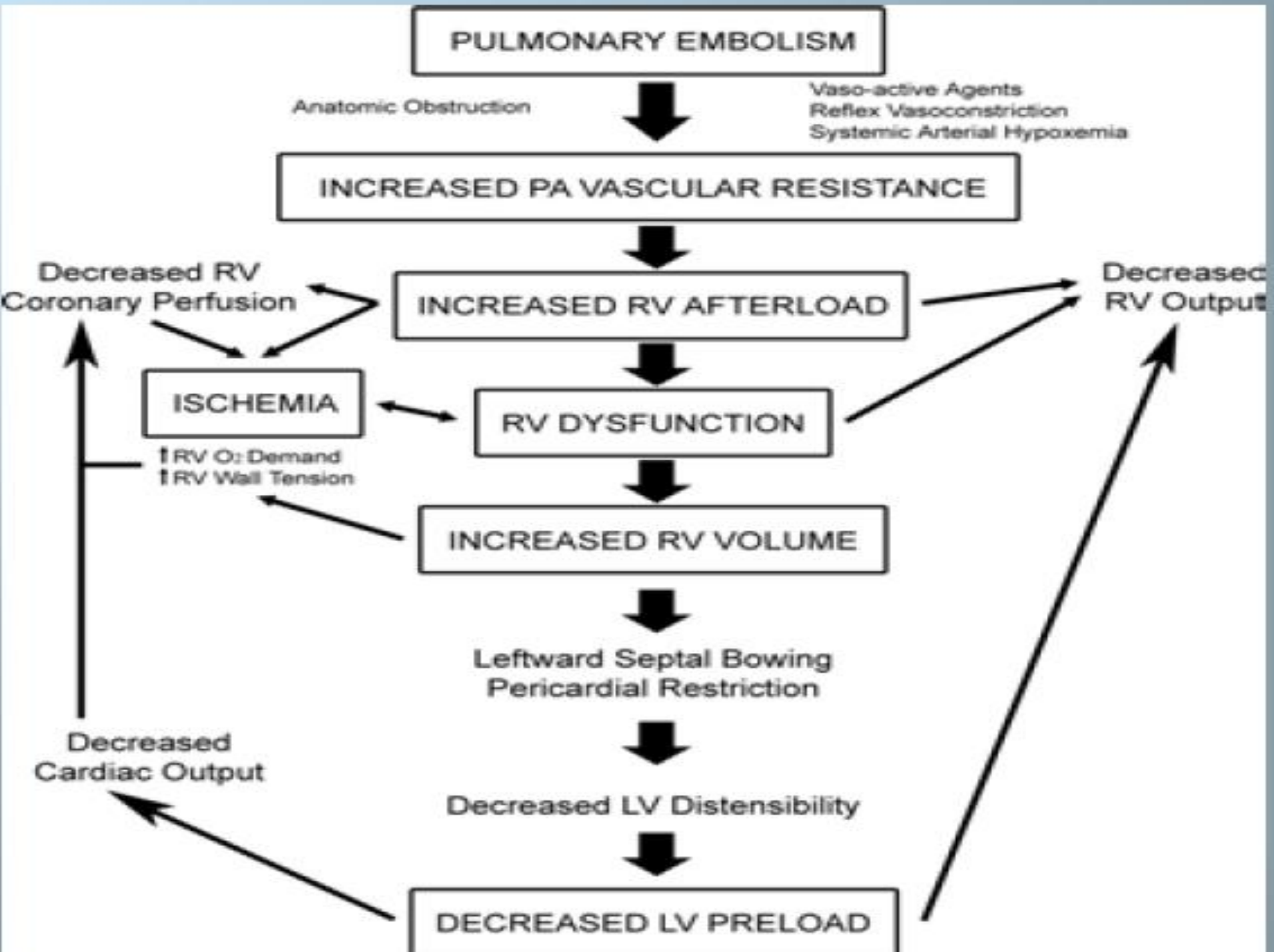
- PE is the most common preventable cause of death in hospitalized patients
- ~600,000 deaths/year
- 80% of pulmonary emboli occur without prior warning signs or symptoms
- 2/3 of deaths due to pulmonary emboli occur within 30 minutes of embolization
- Death due to massive PE is often immediate
- Diagnosis can be difficult
- Early treatment is highly effective



Pathology

At least 90% of pulmonary emboli originate from major leg veins.


Superior vena cava
Inferior vena cava
Pulmonary arteries
RA
LA
LV
RV
Inferior vena cava
Femoral vein
Greater saphenous vein
Popliteal vein
Embolus
Thrombus
Venous valve
Circulatory Stasis
Thrombosis
Endothelial Injury
Hypercoagulable State


PULMONARY EMBOLISM
Anatomic Obstruction
Vaso-active Agents
Reflex Vasoconstriction
Systemic Arterial Hypoxemia
INCREASED PA VASCULAR RESISTANCE
Decreased RV
Coronary Perfusion
INCREASED RV AFTERLOAD
Decreasec
RV Output
ISCHEMIA
RV DYSFUNCTION
↑RV O2 Demand
↑RV Wall Tension
INCREASED RV VOLUME
Leftward Septal Bowing
Pericardial Restriction
Decreased
Cardiac Output
Decreased LV Distensibility
DECREASED LV PRELOAD

Natural History of VTE

- 40-50% of pts with DVT develop PE, often “silent”
- PE presents 3-7 days after DVT
  - Fatal within 1 hour after onset of respiratory symptoms in 10%
  - Shock/persistent hypotension in 5-10% (up to 50% of patients with RV dysfunction)
- Most fatalities occur in untreated pts
- Perfusion defects completely resolve in 75% of all patients (who survive)

Diagnosis: Clinical Presentation

Symptoms of pulmonary embolism are typically sudden in onset and may include one or many of the following:

dyspnea (shortness of breath), tachypnea (rapid breathing), chest pain of a "pleuritic" nature (worsened by breathing), cough and hemoptysis (coughing up blood).

More severe cases can include signs such as cyanosis (blue discoloration, usually of the lips and fingers), collapse, and circulatory instability because of decreased blood flow through the lungs and into the left side of the heart.

About 15% of all cases of sudden death are attributable to PE.

Table 6 Prevalence of symptoms and signs in patients with suspected PE according to final diagnosis

	PE confirmed ($n = 219$)	PE excluded ($n = 546$)
Symptoms **Non-Specific!!**		
Dyspnoea	80%	59%
Chest pain (pleuritic)	52%	43%
Chest pain (substernal)	12%	8%
Cough	20%	25%
Haemoptysis	11%	7%
Syncope	19%	11%
Signs		
Tachypnoea (≥20/min)	70%	68%
Tachycardia (>100/min)	26%	23%
Signs of DVT	15%	10%
Fever (>38.5°C)	7%	17%
Cyanosis	11%	9%

Diagnosis: Chest X-Ray

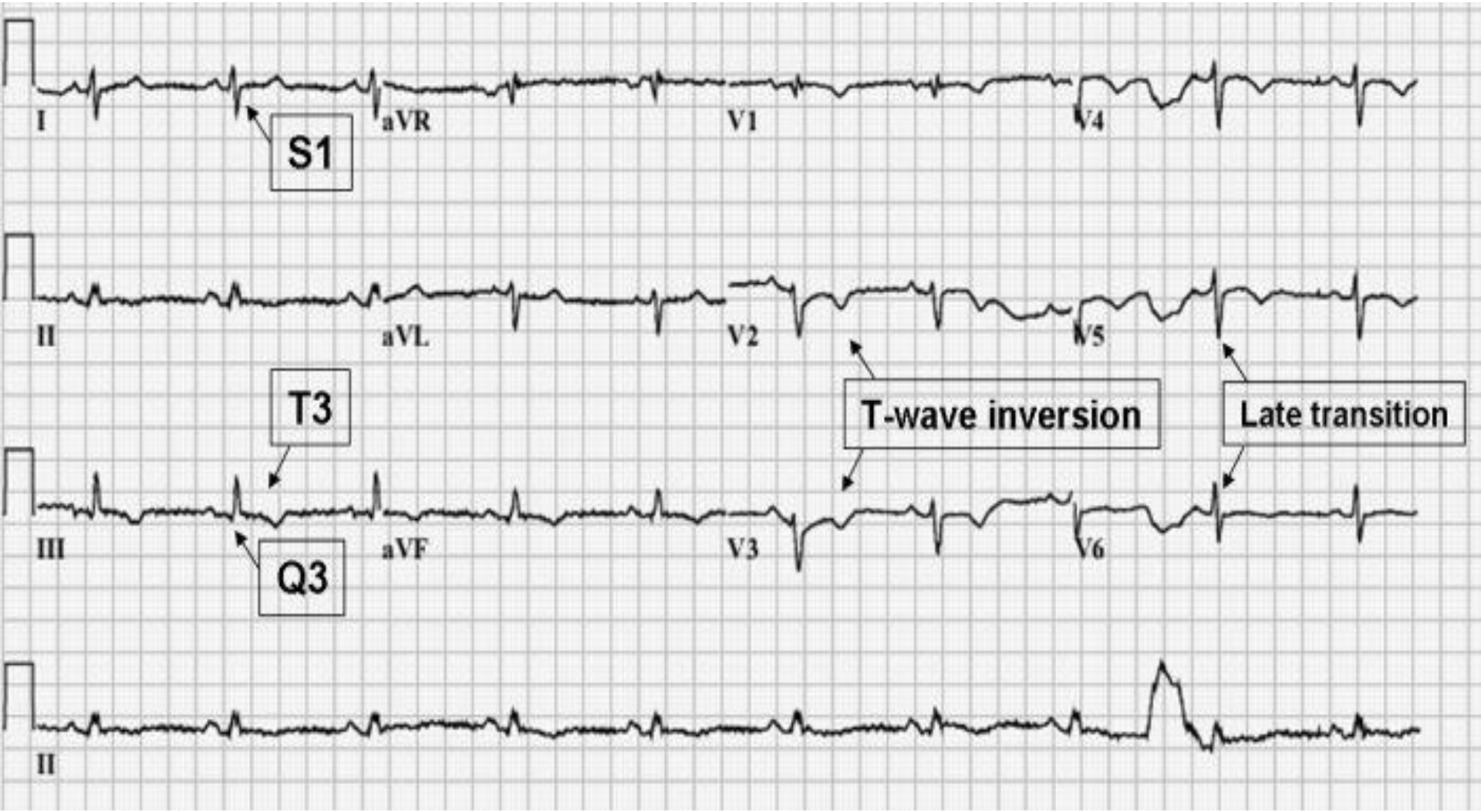
- Usually abnormal, but non-specific
- Study of 2,322 patients with PE:
  - Cardiac enlargement (27%)
  - Normal (24%)
  - Pleural effusion (23%)
  - Elevated hemidiaphragm (20%)
  - Pulmonary artery enlargement (19%)
  - Atelectasis (18%)
  - Parenchymal pulmonary infiltrates (17%)

Chest Radiographs in Acute Pulmonary Embolism: Results From the International Cooperative Pulmonary Embolism Registry. Chest July 2000 118:3338; 10.1378/chest.118.1.33

Diagnosis: ECG

- Usually non-specific ST/T waves changes and tachycardia
- RV strain patterns suggest severe PE
  - Inverted T waves V1-V4
  - QR in V1
  - Incomplete RBBB
  - S1Q3T3

S1Q3T3 and T wave changes



Diagnosis:Other tests

- Most patients with PE have a normal pulse oximetry

Clinical Diagnosis of PE

- In summary, clinical signs, symptoms and routine tests do not allow for the exclusion or confirmation of acute PE but may increase the index of its suspicion
- Consider PE in cases of unexplained tachycardia or syncope

Diagnosis-Probability Assessment

- Implicit clinical judgement is fairly accurate: “Do you think this patient has a PE?”
- Validated prediction rules standardize clinical judgement
    - Wells
    - Geneva

Modified Wells criteria: clinical assessment for pulmonary embolism

Clinical symptoms of DVT (leg swelling, pain with palpation)	3.0
Other diagnosis less likely than pulmonary embolism	3.0
Heart rate >100	1.5
Immobilization (≥3 days) or surgery in the previous four weeks	1.5
Previous DVT/PE	1.5
Hemoptysis	1.0
Malignancy	1.0
Probability	**Score**
Traditional clinical probability assessment	
High	>6.0
Moderate	2.0 to 6.0
Low	<2.0
Simplified clinical probability assessment*	
PE likely	>4.0
PE unlikely	≤4.0

Proportion with PE

65%

30

10%

Data from van Belle, A, et al. JAMA 2006; 295:172.

Revised Geneva Score*

Characteristic	Points
Age older than 65 years	1
Previous deep venous thrombosis or pulmonary embolism	3
Surgery or fracture within 1 month	2
Active malignant condition	2
Unilateral lower limb pain	3
Hemoptysis	2
Heart rate 75–94 beats/minute	3
Heart rate 95 beats/minute or more	5
Pain in response to lower-limb deep venous palpation, unilateral edema	4

0–3 points indicates low clinical probability of pulmonary embolism (8% in the original validation study*)

4–10 points indicates intermediate clinical probability of pulmonary embolism (28% in the original validation study*)

≥11 points indicates high clinical probability of pulmonary embolism (74% in the original validation study*)

**Le Gal G. Prediction of pulmonary embolism in the emergency department: The revised Geneva score. Ann Intern Med 2006;144:165.*

Diagnosis

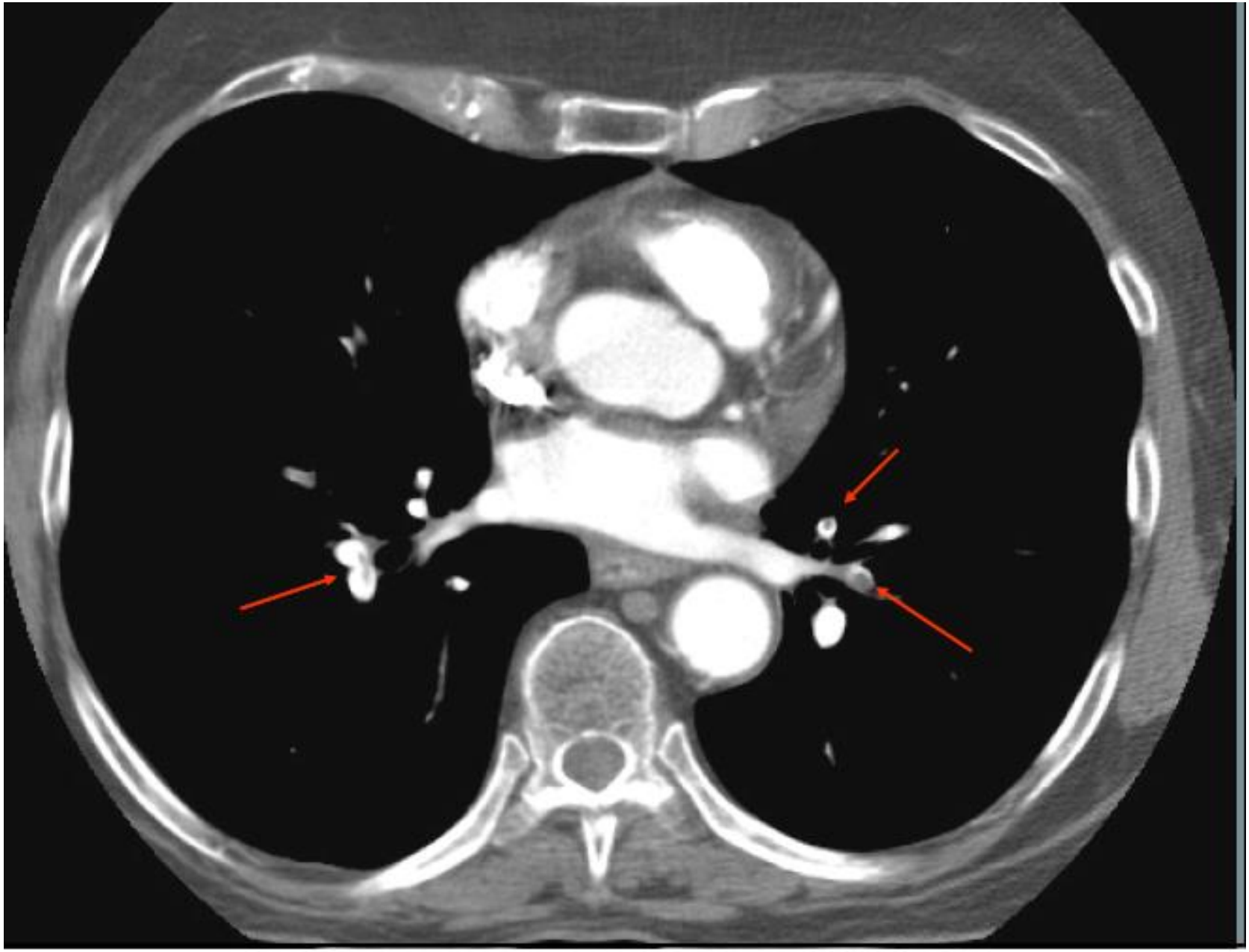
- D-Dimer
  - Fibrin degradation product
  - ELISA tests are highly sensitive (>95%)
  - Non specific (~40%): cancer, sepsis, inflammation increase d-dimer levels
- Sn*N*Out
  - Negative result excludes PE safely in **PE-unlikely** patients (using Clinical probability scores)

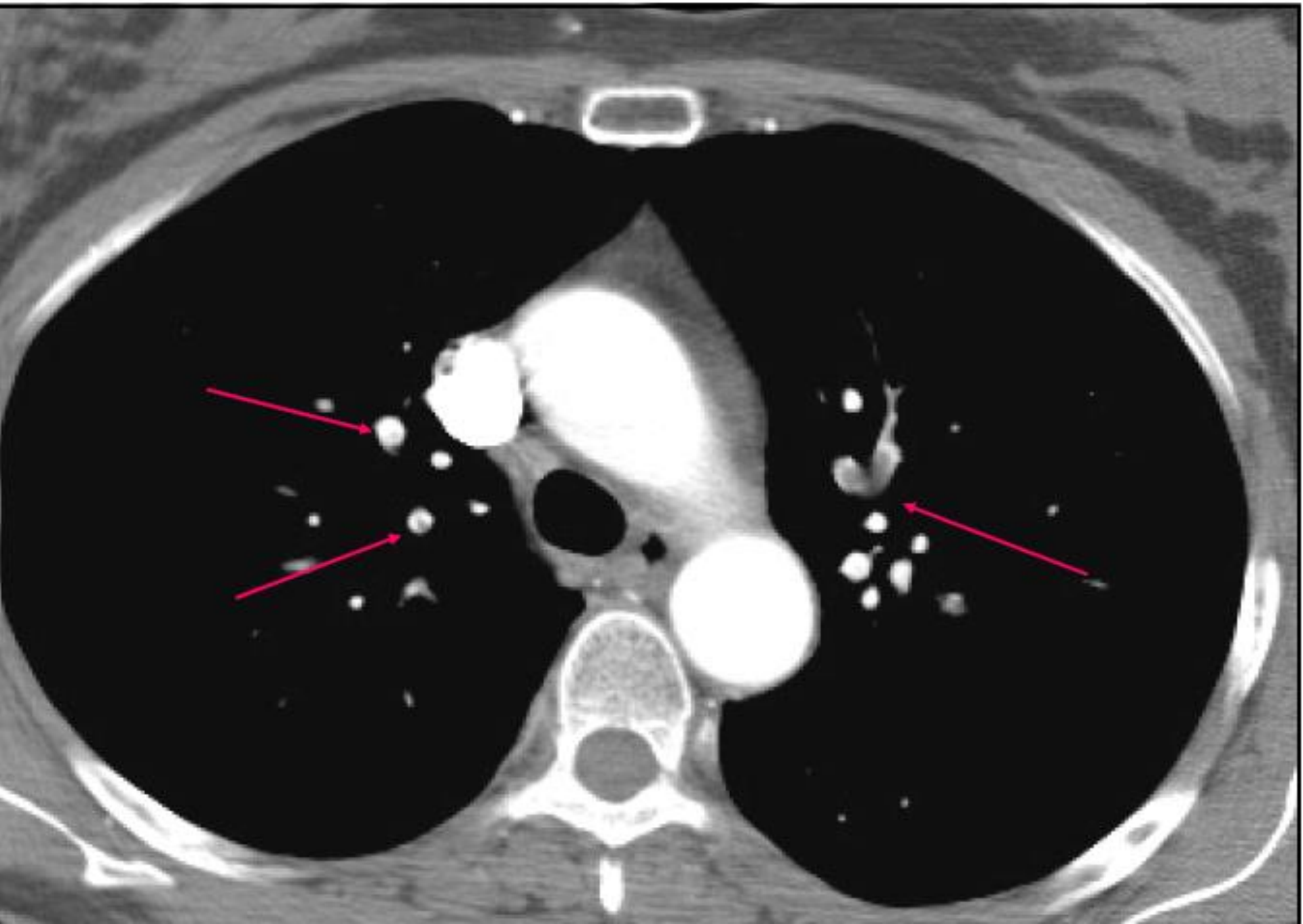
Spiral CT

- Direct visualization of emboli.
- Both parenchymal and mediastinal structures can be evaluated.
- Offers differential diagnosis in 2/3 of cases with a negative scan.

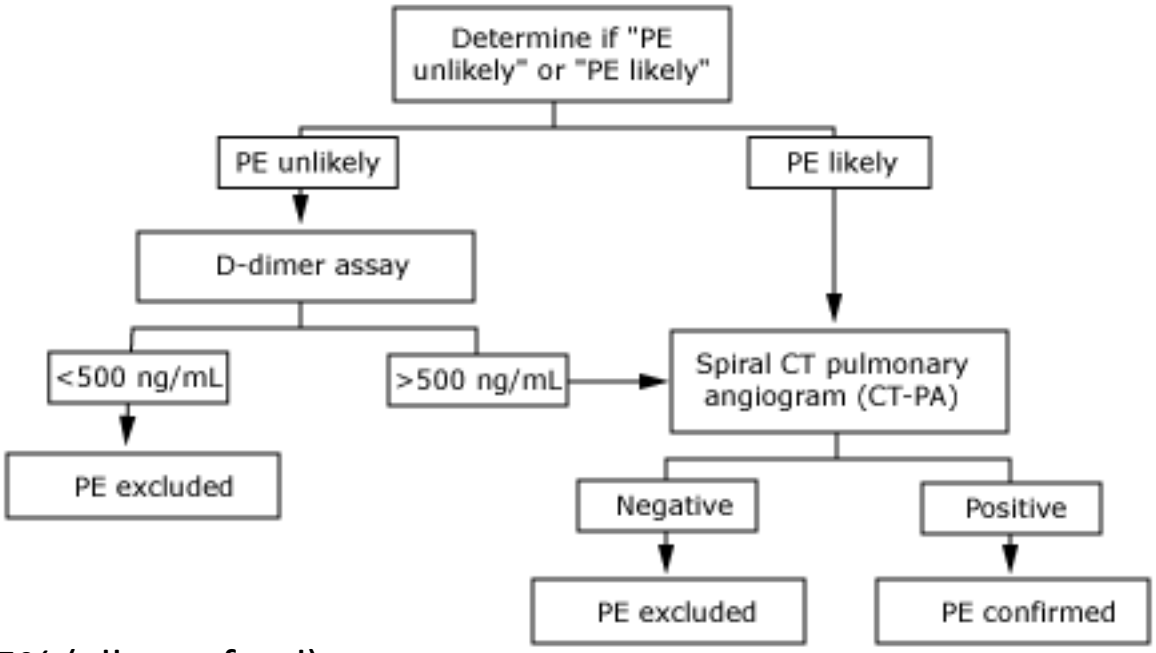
BUT…

- Dye load and large radiation dose
- Optimally used when incorporated into a validated diagnostic decision tree





CT-based diagnostic strategy used in patients with suspected pulmonary embolism



3 month VTE rate

0.5% (all non fatal)

1.3%

Adapted from van Belle, A, et al. JAMA 2006; 295:172.

UpToDate

This algorithm allowed for a management decision in 98% of patients presenting with symptoms suggestive of PE

Diagnosis- Summary

- History and physical examination
- Then 1,2,3 approach:
  1. Clinical decision score
  2. D-Dimer test
  3. Chest CT

Pneumothorax

What is a pneumothorax?

- Air within the pleural cavity (i.e. between visceral and parietal pleura)
- The air enters via a defect in the visceral pleura (e.g. ruptured bulla) or the parietal pleura (e.g. puncture following rib fracture)

A primary spontaneous pneumothorax (PSP) tends to occur in a young adult without underlying lung problems, and usually causes limited symptoms.

Chest pain and sometimes mild breathlessness are the usual predominant presenting features.

People who are affected by a PSP are often unaware of the potential danger and may wait several days before seeking medical attention.

A primary spontaneous pneumothorax is one that occurs without an apparent cause and in the absence of significant lung disease.

A secondary spontaneous pneumothorax occurs in the presence of existing lung disease.

Smoking increases the risk of primary spontaneous pneumothorax, while the main underlying causes for secondary pneumothorax are COPD, asthma, and tuberculosis.

A pneumothorax can also be caused by physical trauma to the chest or as a complication of a healthcare intervention, in which case it is called a traumatic pneumothorax.

Diagnosis of a pneumothorax by physical examination alone can be difficult (particularly in smaller pneumothoraces).

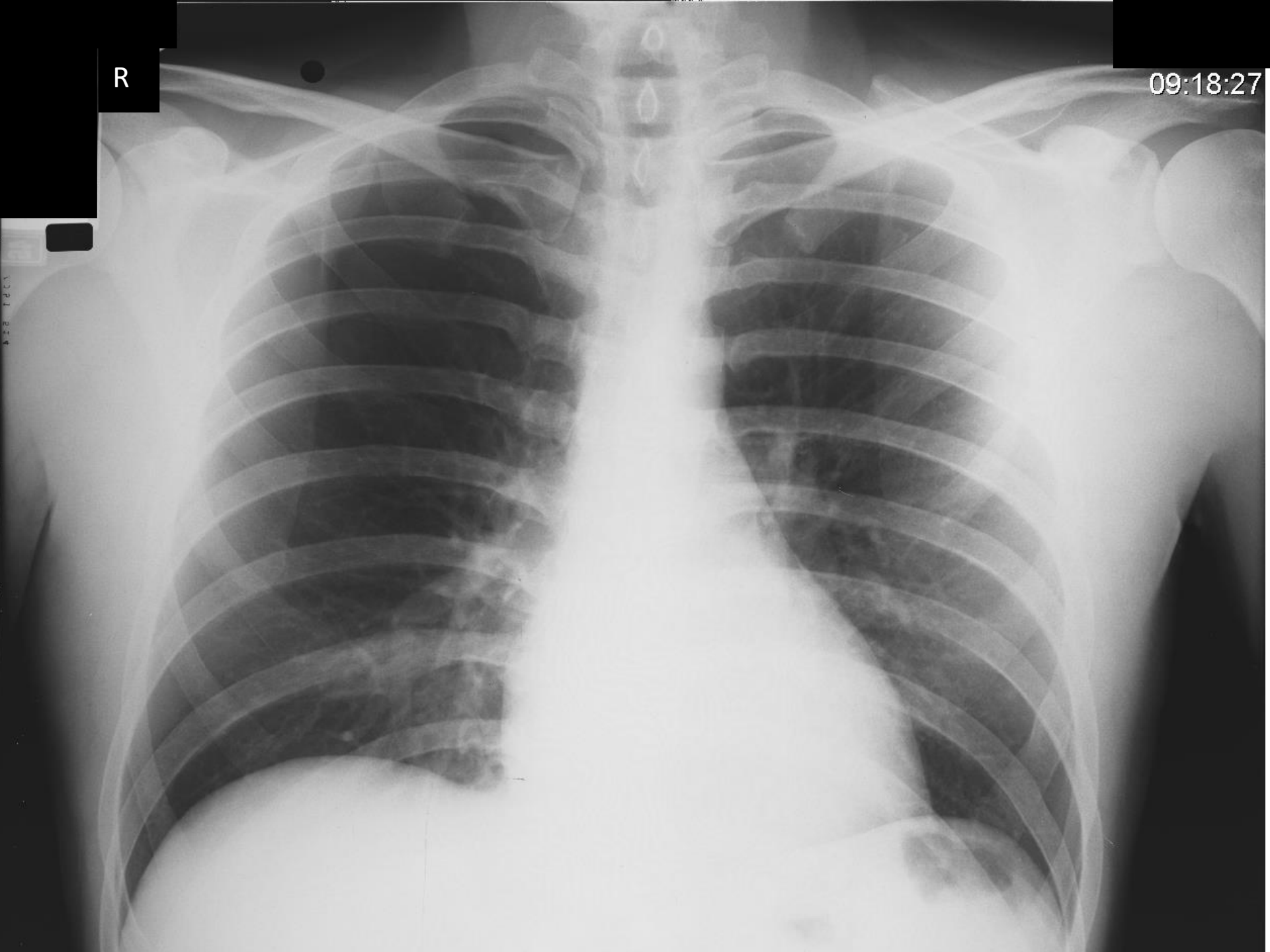
A chest X-ray, computed tomography (CT) scan, or ultrasound is usually used to confirm its presence.

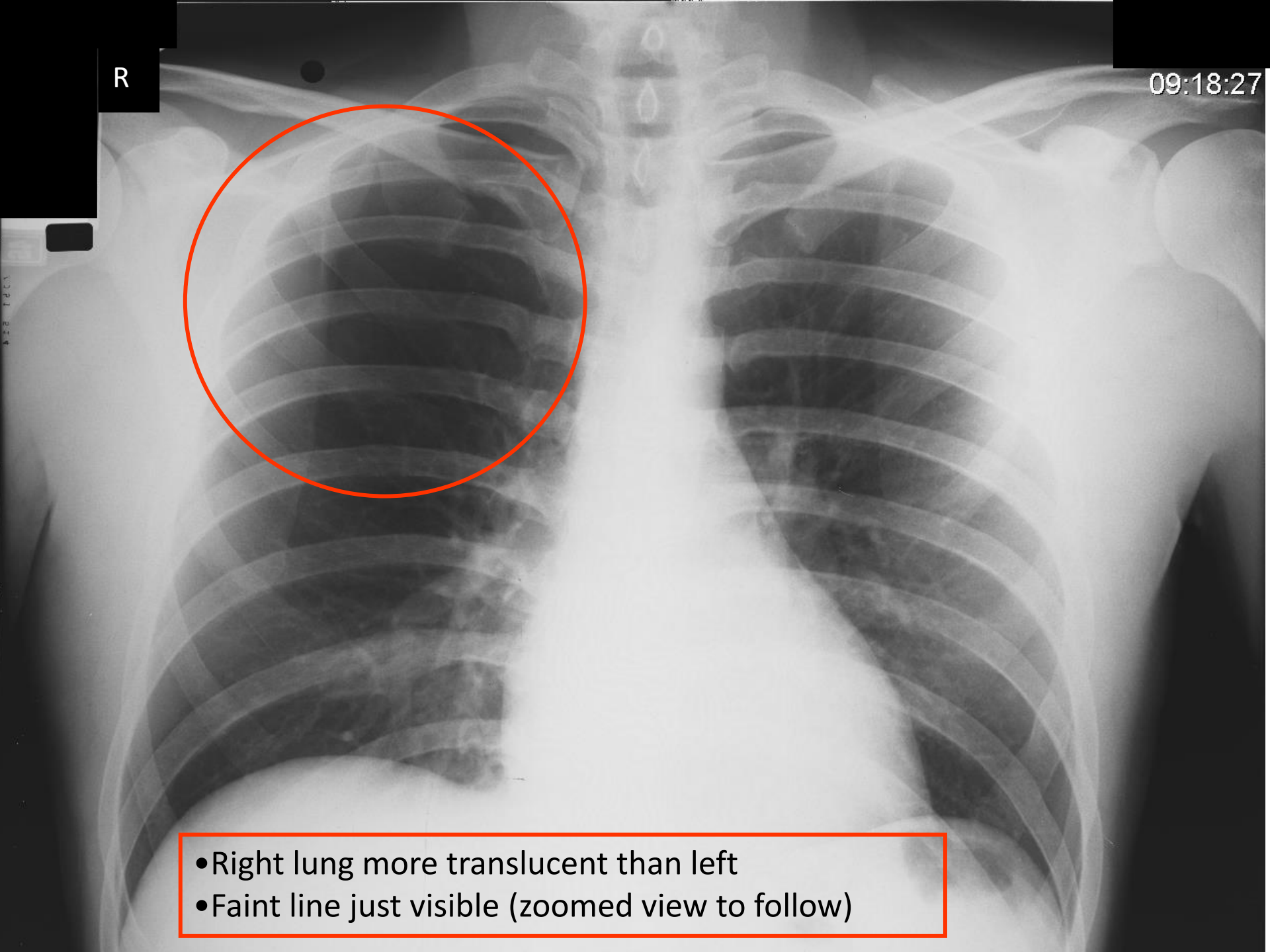
Other conditions that can result in similar symptoms include a hemothorax (buildup of blood in the pleural space), pulmonary embolism, and heart attack.

CXR features of pneumothorax

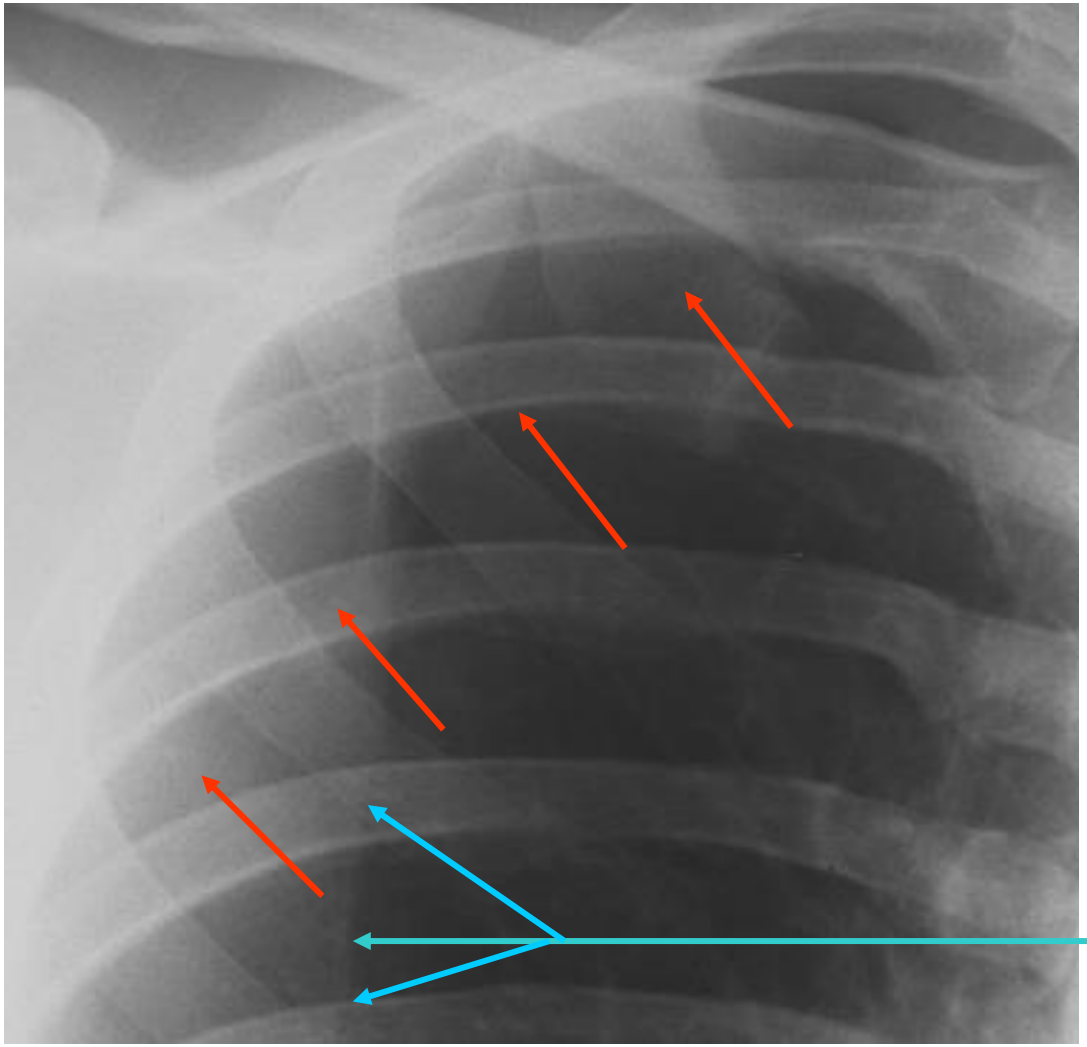
- White line of visceral pleura parallel to chest wall
- No lung markings lateral to the line
- There may be associated rib fractures
- Do not confuse the line with skin fold or with scapula
- The most sensitive test if in doubt is a CXR taken in expiration

Look at the CXR on the next slide. Where is the pneumothorax?


R
09:18:27


R
09:18:27
•Right lung more translucent than left
•Faint line just visible (zoomed view to follow)

Right pneumothorax



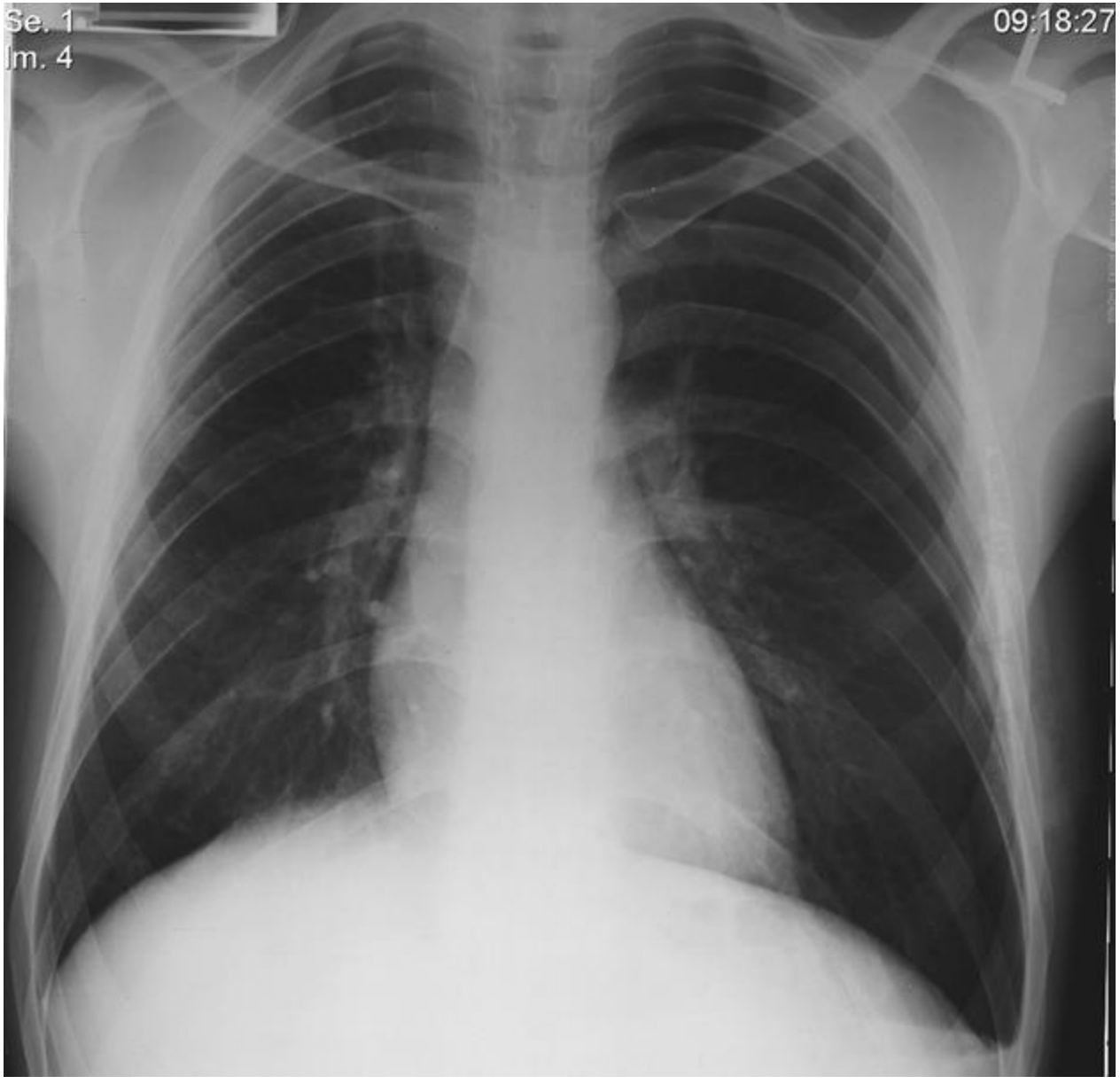
- Pencil-thin white line running parallel to chest wall
- No lung markings lateral to the line

Blade of right scapula

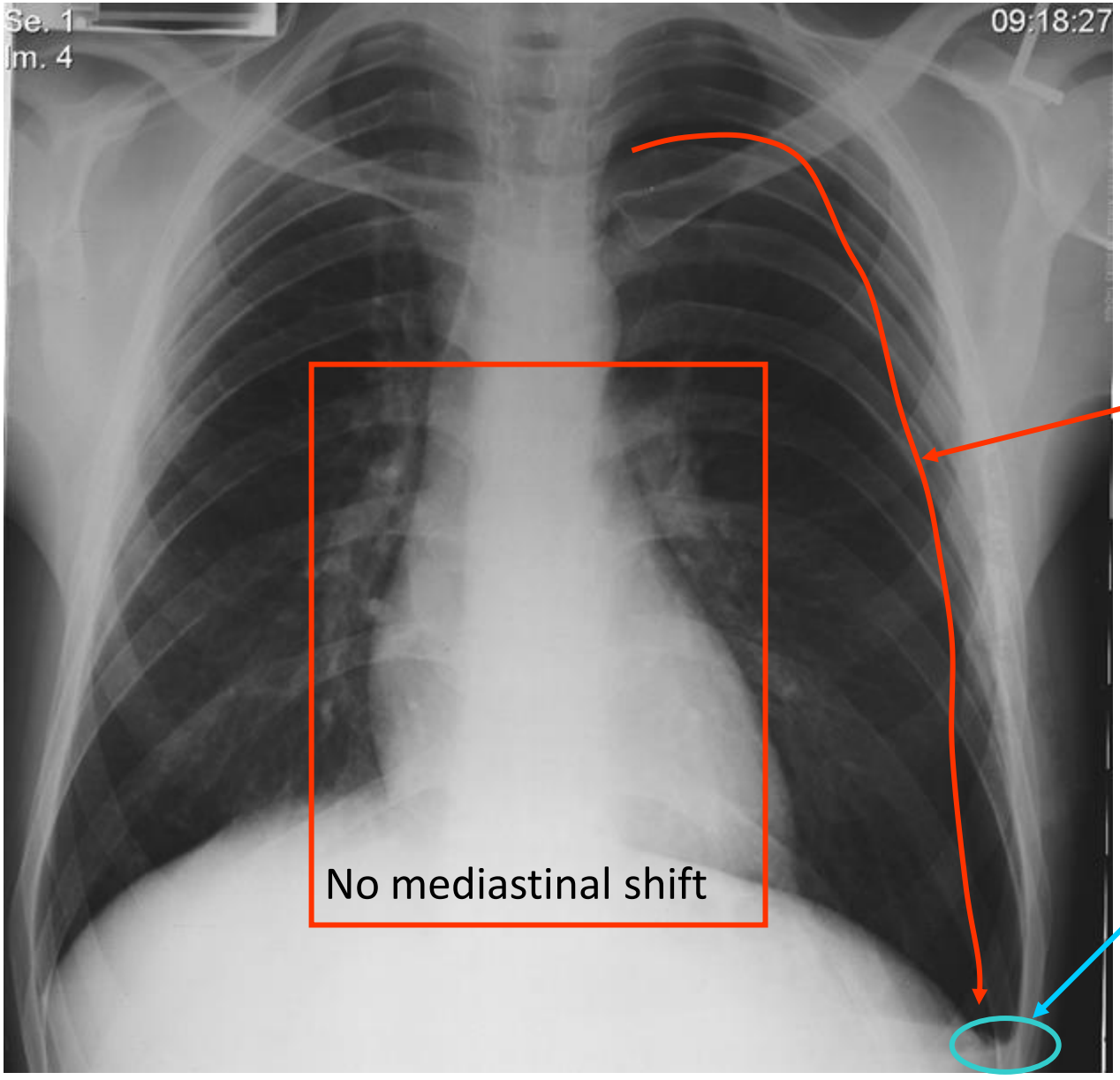
Types of Pneumothorax

- Simple
  - Mediastinum remains central
  - Clinical condition stable
  - Can wait for CXR to confirm diagnosis
- Tension
  - Progressive build up of air in the pleural space, causing a shift of the heart and mediastinal structures away from side of pneumothorax
  - This can cause a steadily worsening oxygen shortage and low blood pressure and unless reversed can be fatal.[
  - Clinical condition unstable
  - Do **not** wait for CXR to confirm diagnosis

Simple Left Pneumothorax

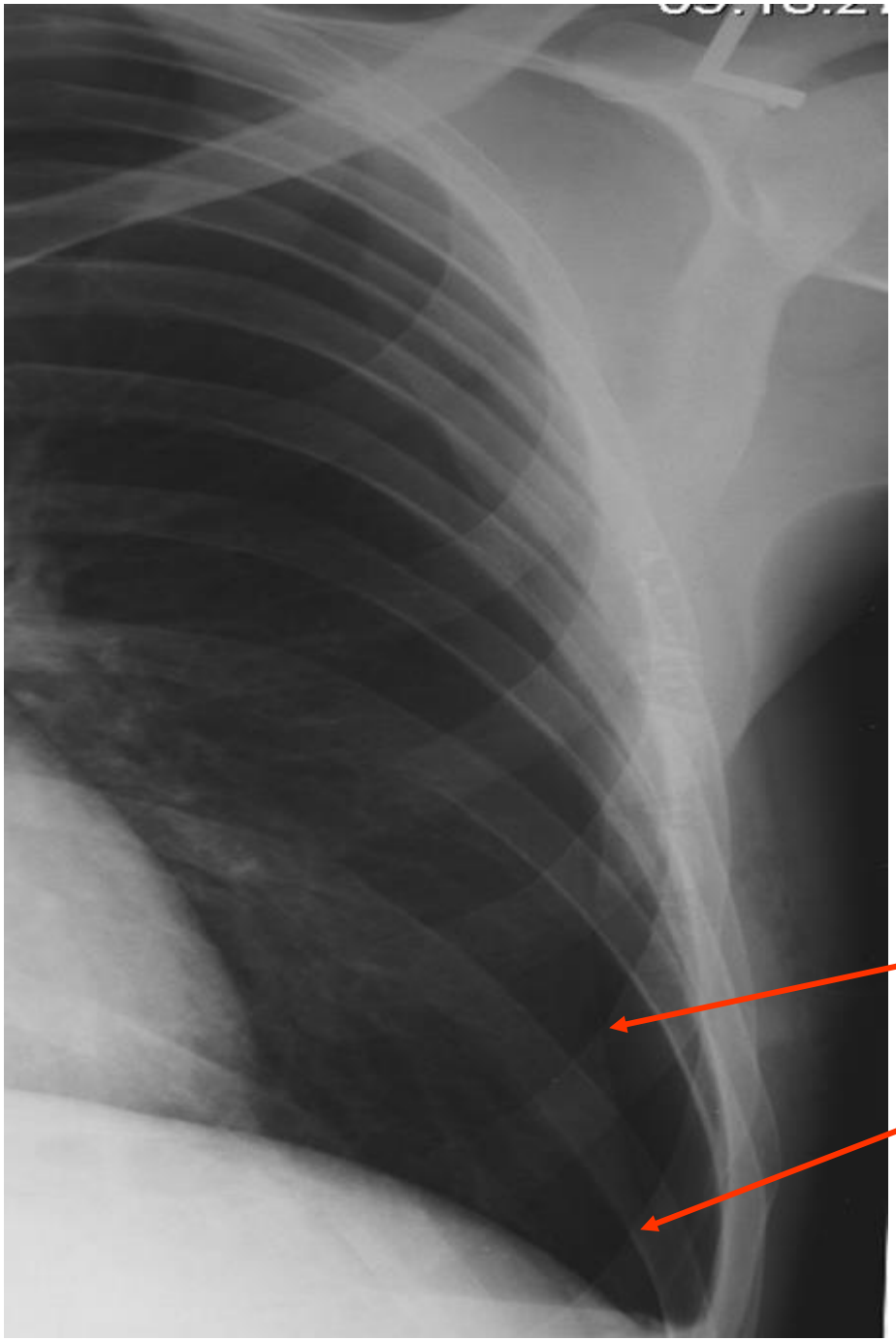


Simple Left Pneumothorax

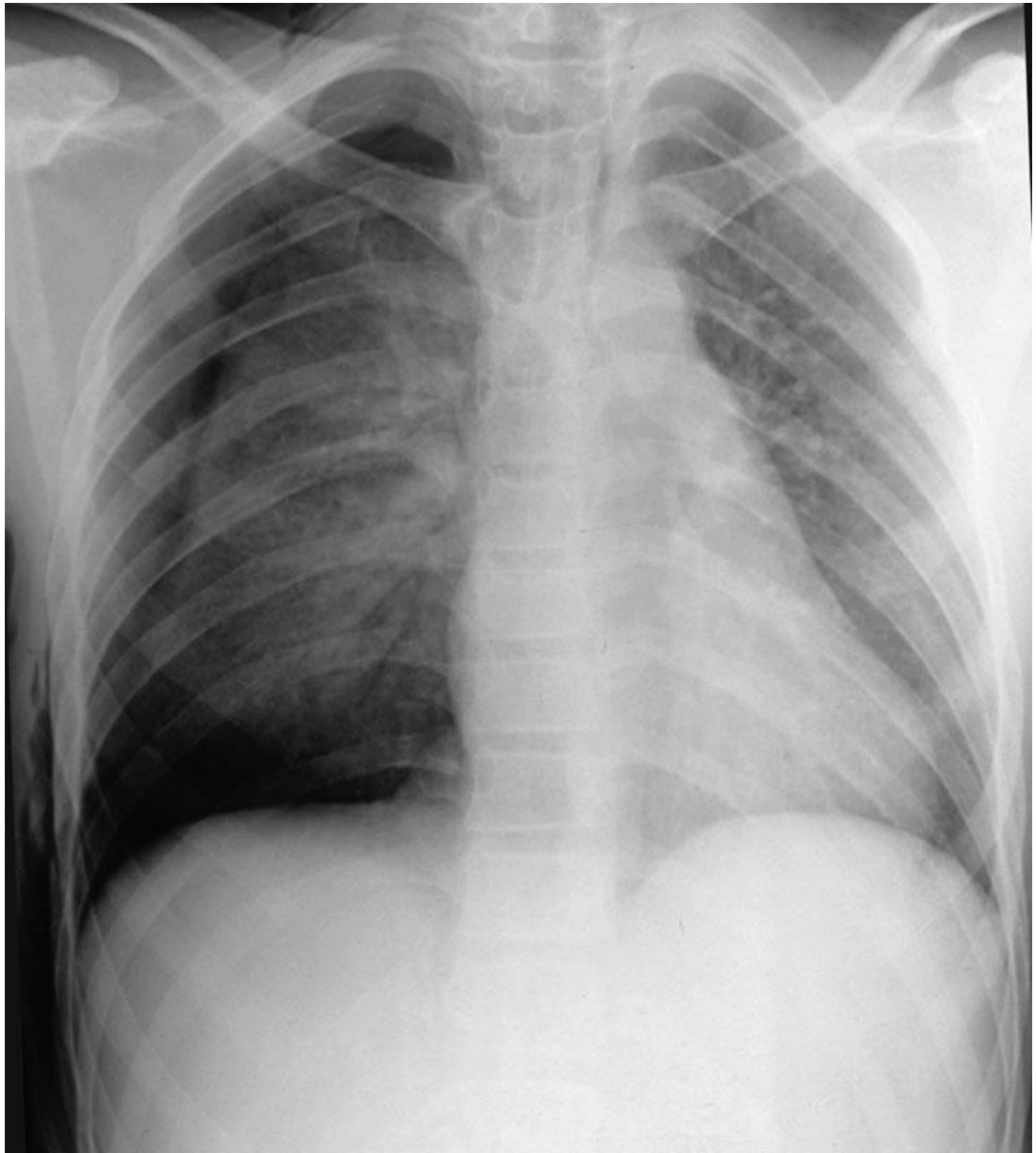


Visceral pleural line (zoomed view on next slide)

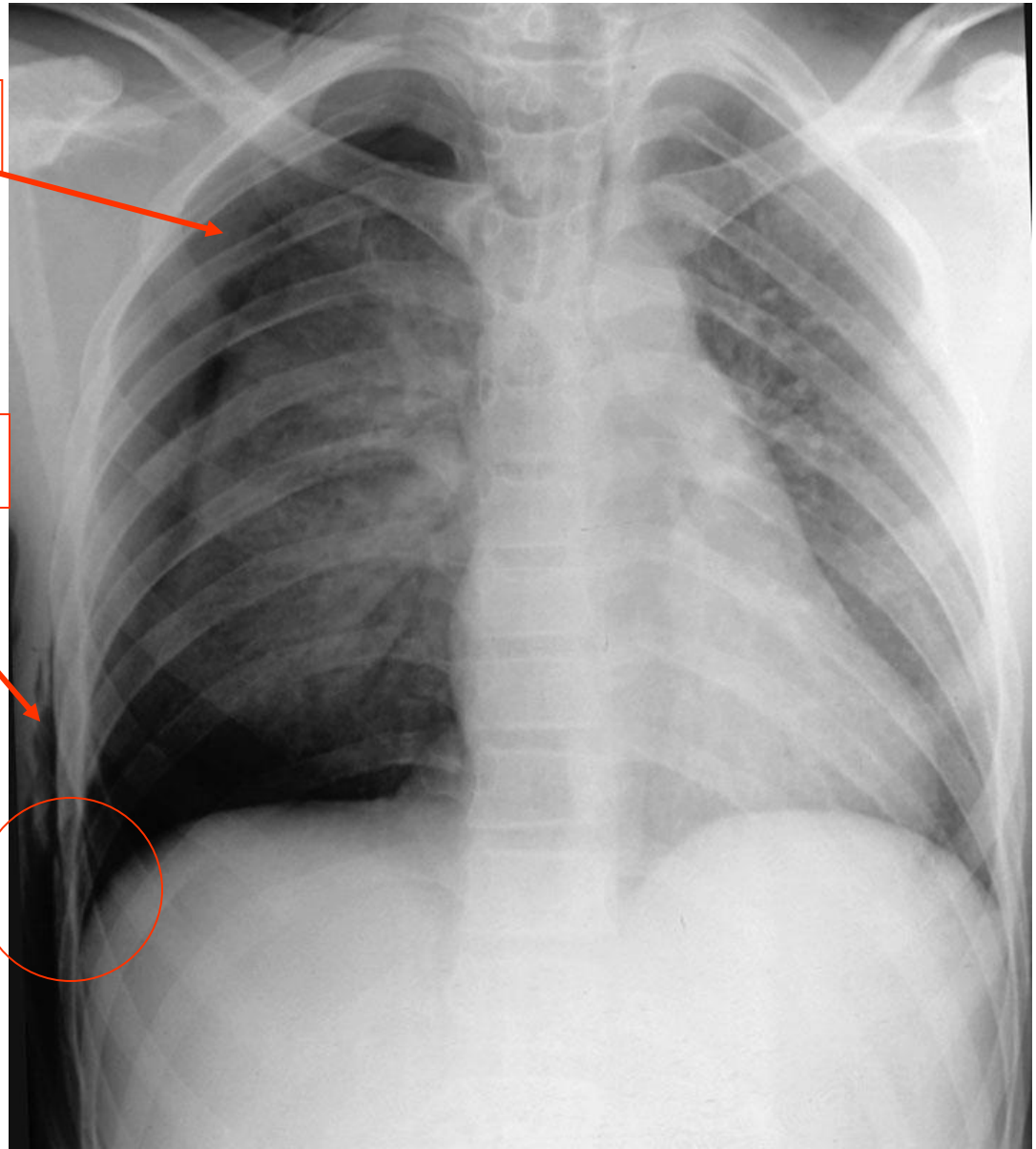
Small pleural effusion (common finding)


Note absence of lung markings lateral to this line

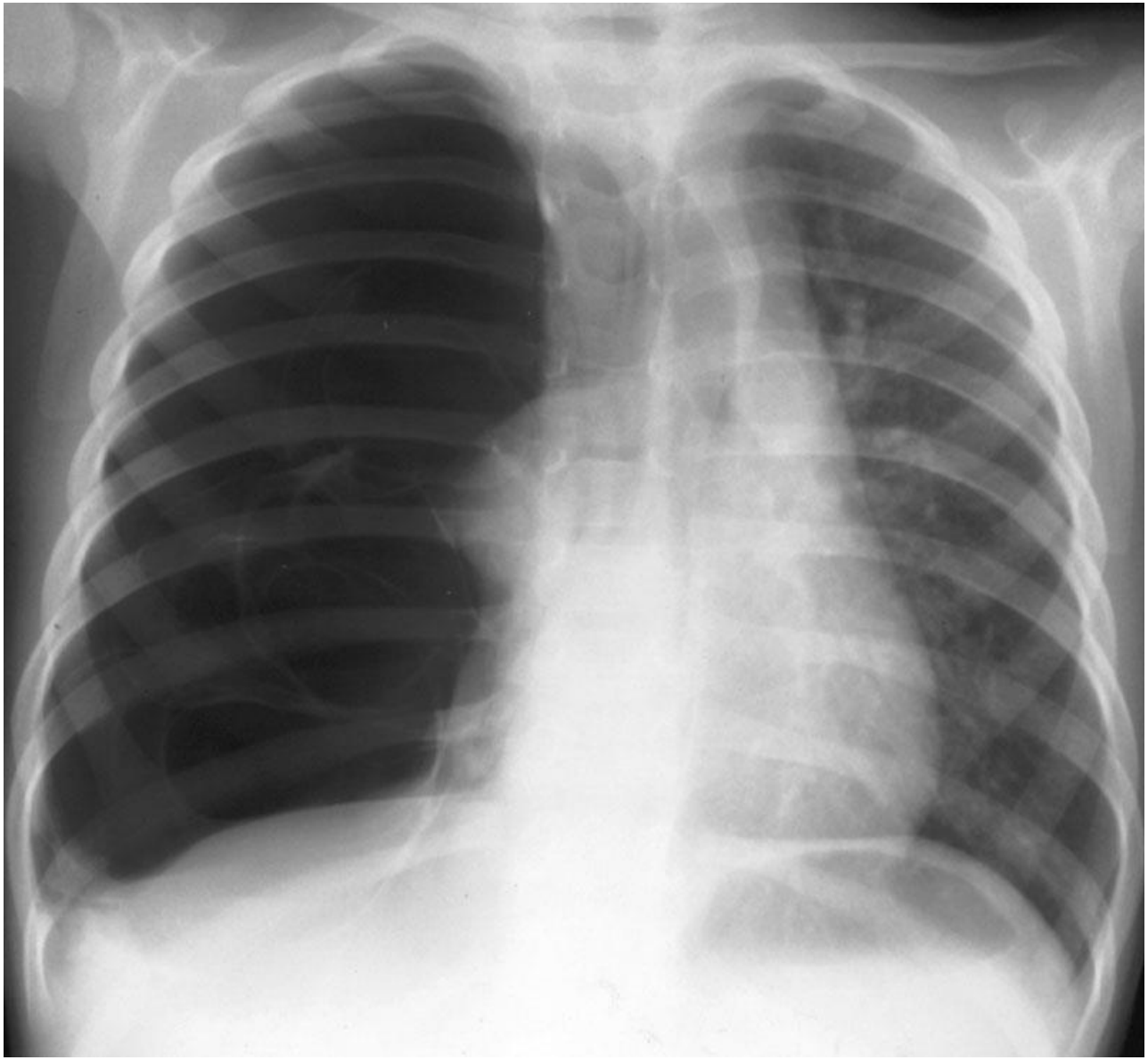
Pneumothorax with rib fractures



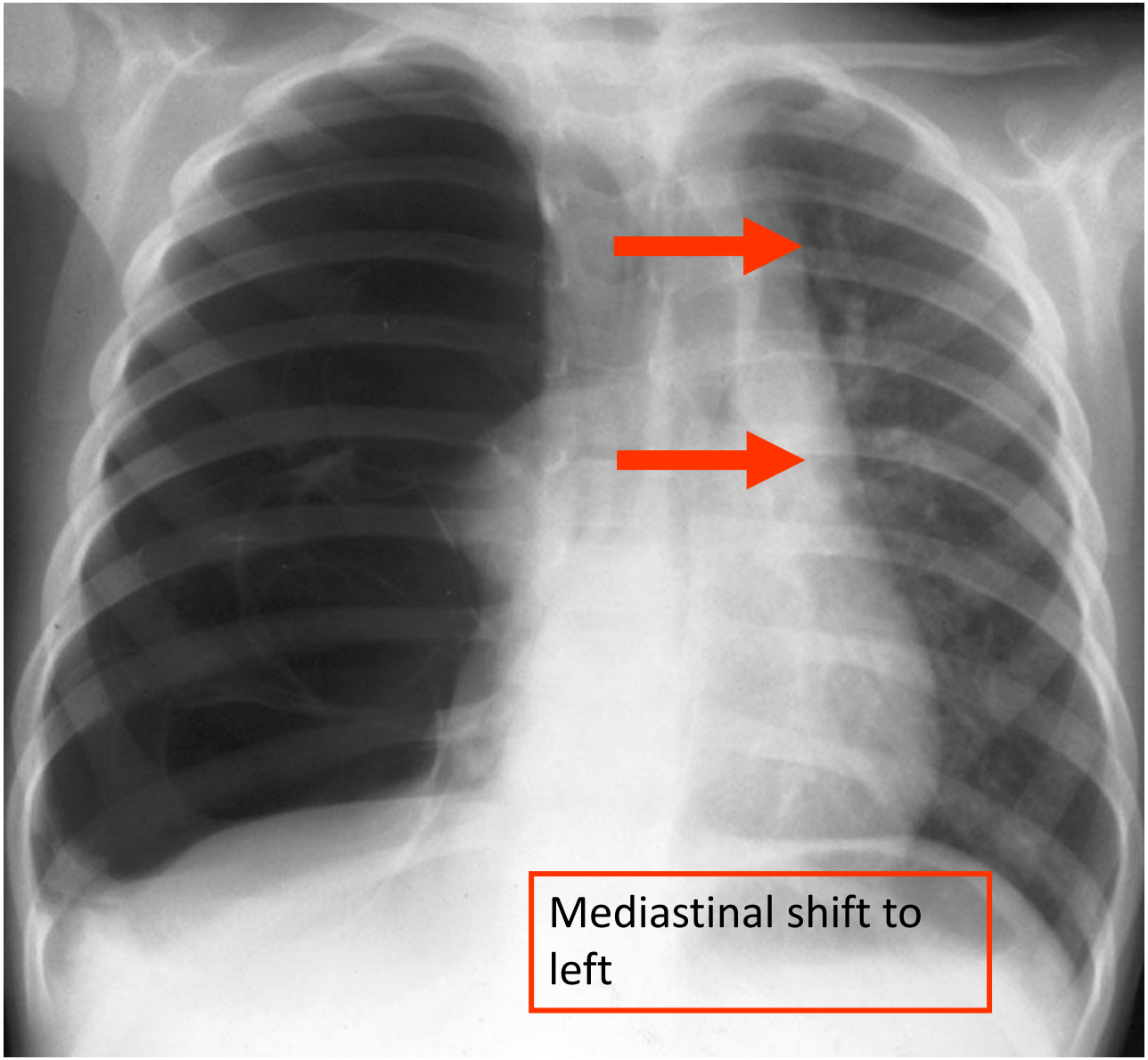
Pneumothorax with rib fractures



Tension right pneumothorax



Tension right pneumothorax



Causes of Pneumothorax

- Spontaneous
  - Rupture of an apical bleb
- Traumatic
  - With rib fractures
  - Penetrating chest trauma
- Pre-existing lung abnormality
  - Pulmonary fibrosis
  - Asthma
  - Vasculitis
  - Pulmonary metastases close to edge of lung

Other causes of absent lung markings

- Large emphysematous bullae
- Large lung cysts
- Pulmonary embolism

….but only pneumothorax has a white line parallel to the chest wall

Take Home Points

- Look for a pencil-thin white line parallel to the chest wall
- No lung markings lateral to the line
- Make sure the patient does not have another cause for absent lung markings before inserting a chest drain

TOPIC: RESPIRATORY SYSTEM

Title of the seminar: COPD, asthma, pneumonia

Name of the tutor: Kristian Debretseni

Duration: 90 min

Audience: 3rd course students

Goals:

General: To master skills in diagnostics of COPD, asthma, pneumonia.

Special goals:

- Knowledge: Study the main symptoms, signs and risk factors of COPD, asthma, pneumonia and main diagnostic methods.
- Practical skills: history-taking in patients with COPD, asthma, pneumonia.
- Changing attitude: Ability to recognizing COPD and bronchial asthma is very important for every doctor or student, because sometimes these diseases appear with emergency life-threatened condition that should be resolved immediately.

Methodology of the seminar:

5 minutes Short introduction

5 minutes Ice breaker

20 minutes Theory presentation (Power Point presentation)

35 minutes Interactive
Work in pairs (patient-doctor)
Practical training of lungs auscultation (on patients, on each other)

15 minutes Control – verbal interview

10 minutes Feedback from each participant: take home notes: “What was new for me today? What did I learn/practice? What will I introduce into my future practice?”

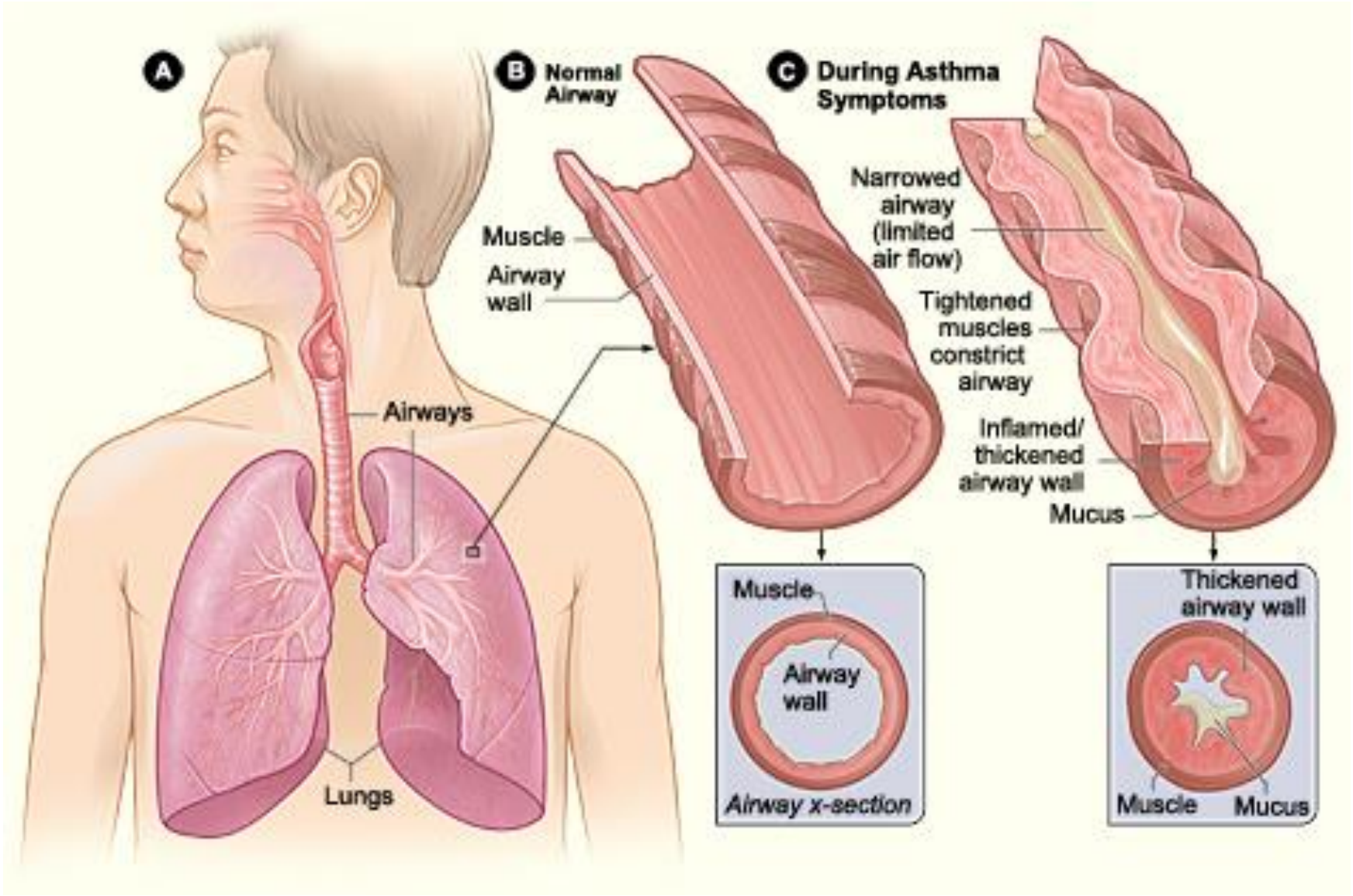
Results: we expect each participant:

- Master the basic knowledge about COPD, asthma, pneumonia.
- Practice to perform lungs auscultation.

Asthma

Definition (GINA 2016)

- Asthma is a heterogeneous disease, usually characterized by chronic airway inflammation. It is defined by the history of respiratory symptoms such as wheeze, shortness of breath, chest tightness and cough that vary over time and in intensity, together with variable expiratory airflow limitation.


A
B Normal Airway
C During Asthma Symptoms
Muscle
Airway wall
Airways
Lungs
Narrowed airway (limited air flow)
Tightened muscles constrict airway
Inflamed/ thickened airway wall
Mucus
Muscle
Airway wall
Airway x-section
Thickened airway wall
Muscle
Mucus

Epidemiology

As of 2011, 235–330 million people worldwide are affected by asthma, and approximately 250,000–345,000 people die per year from the disease. Rates vary between countries with prevalences between 1 and 18%. It is more common in developed than developing countries. One thus sees lower rates in Asia, Eastern Europe and Africa.[

- Global rates of asthma have increased significantly between the 1960s and 2008 with it being recognized as a major public health problem since the 1970s.

- Asthma affects approximately 7% of the population of the United States and 5% of people in the United Kingdom. Canada, Australia and New Zealand have rates of about 14–15%.

Worldwide Burden of Asthma

- 300 million people suffer from asthma worldwide.
  - 255,000 asthma deaths in 2005.
    - ~3,500 in the United States.
  - Over 80% of asthma deaths occur in low and lower-middle income countries.

Causes

- While the exact cause of asthma is not known, it is thought that a variety of factors interacting with one another, early in life, result in the development of asthma.

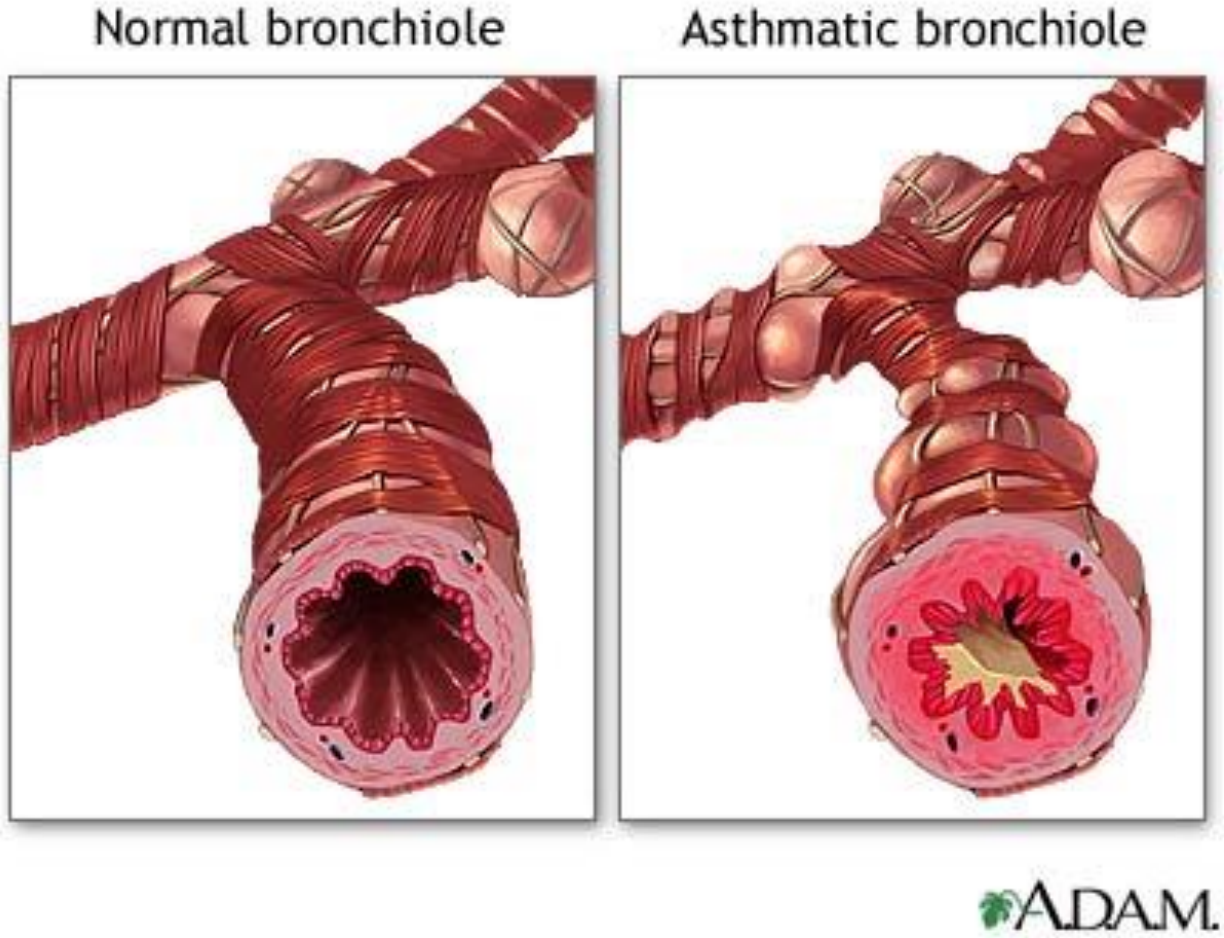
Causes

- Parents with asthma
- Atopy
- Childhood respiratory infections
- Exposure to allergens or infections while the immune system is developing

Pathophysiology

- The pathophysiology of asthma is complex and involves airway inflammation, intermittent airflow obstruction, and bronchial hyperresponsiveness. The mechanism of inflammation in asthma may be acute, subacute, or chronic, and the presence of airway edema and mucus secretion also contributes to airflow obstruction and bronchial reactivity. Varying degrees of mononuclear cell and eosinophil infiltration, mucus hypersecretion, desquamation of the epithelium, smooth muscle hyperplasia, and airway remodeling are present.

Normal bronchiole / Asthmatic bronchiole

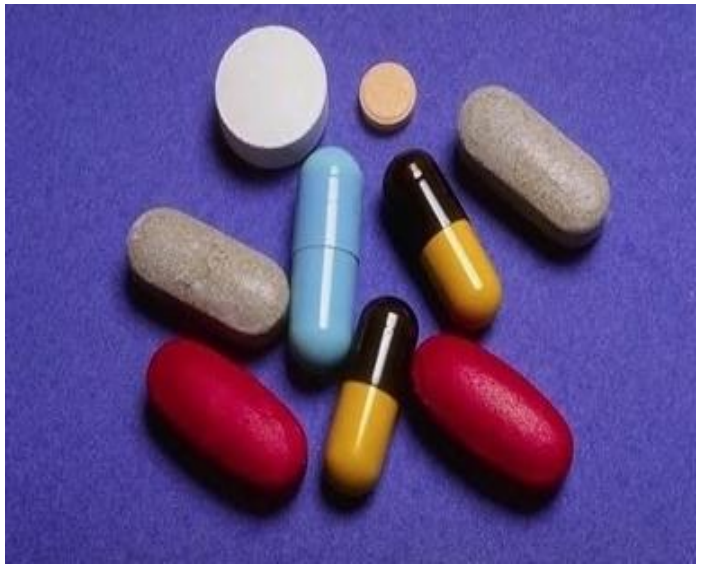


What are the Triggering Factors?

- Domestic dust mites
- Air pollution
- Tobacco smoke
- Occupational irritants
- Animal with fur
- Pollen

Triggering Factors (cont.)

- Respiratory (viral) infections
- Chemical irritants
- Strong emotional expressions
- Drugs (aspirin, beta blockers)



Classification

Severity	Symptom frequency	Night-time symptoms	%FEV_1 of predicted	FEV_1 variability	SABA use
Intermittent	≤2/week	≤2/month	≥80%	<20%	≤2 days/week
Mild persistent	>2/week	3–4/month	≥80%	20–30%	>2 days/week
Moderate persistent	Daily	>1/week	60–80%	>30%	daily
Severe persistent	Continuously	Frequent (7/week)	<60%	>30%	≥twice/day

Diagnosis

- Based on:
  - Medical history
  - Physical examination
  - Test results

MAKING THE INITIAL DIAGNOSIS

Making the diagnosis of asthma is based on identifying both a characteristic pattern of respiratory symptoms such as wheezing, shortness of breath (dyspnea), chest tightness or cough, and variable expiratory airflow limitation. The pattern of symptoms is important, as respiratory symptoms may be due to acute or chronic conditions other than asthma.

Patterns of respiratory symptoms that are characteristic of asthma

The following features are typical of asthma and, if present, increase the probability that the patient has asthma:

- More than one symptom (wheeze, shortness of breath, cough, chest tightness), especially in adults
- Symptoms often worse at night or in the early morning
- Symptoms vary over time and in intensity
- Symptoms are triggered by viral infections (colds), exercise, allergen exposure, changes in weather, irritants such as car exhaust fumes, smoke or strong smells.

Asthma

Clinical Manifestations

- Expiration may be prolonged from a inspiration-expiration ratio of 1:2 to 1:3 or 1:4

- Between attacks may be asymptomatic with normal or near-normal lung function

The following features decrease the probability that respiratory symptoms are due to asthma:

- Isolated cough with no other respiratory symptoms
- Chronic production of sputum
- Chest pain
- Exercise-induced dyspnea with noisy inspiration.

DIAGNOSTIC FEATURE	CRITERIA FOR MAKING THE DIAGNOSIS OF ASTHMA
1. History of variable respiratory symptoms	
Wheeze, shortness of breath, chest tightness and cough Descriptors may vary between cultures and by age, e.g. children may be described as having heavy breathing	• Generally more than one type of respiratory symptom (in adults, isolated cough is seldom due to asthma) • Symptoms occur variably over time and vary in intensity • Symptoms are often worse at night or on waking • Symptoms are often triggered by exercise, laughter, allergens, cold air • Symptoms often appear or worsen with viral infections

History and family history

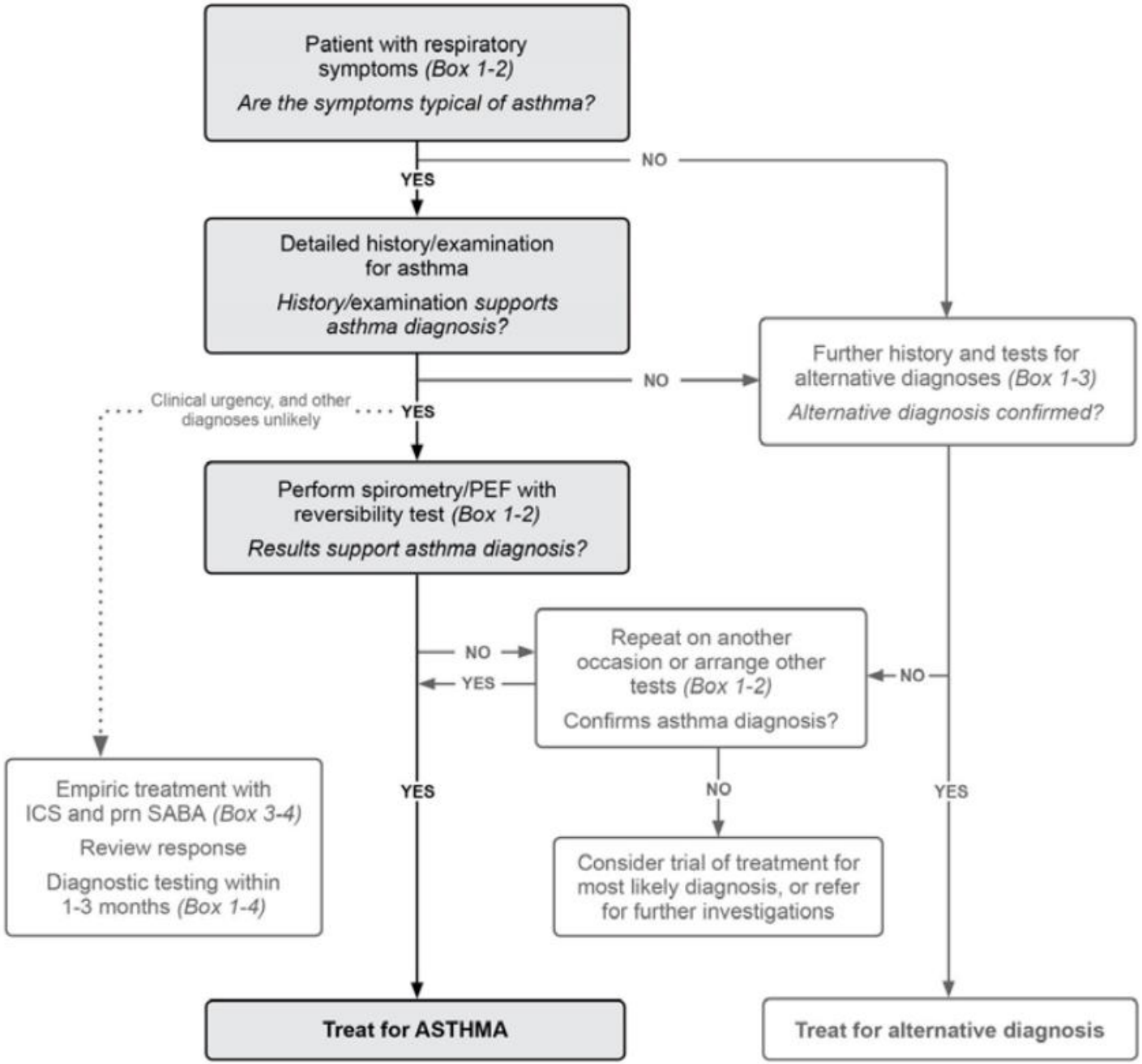
- Commencement of respiratory symptoms in childhood, a history of allergic rhinitis or eczema, or a family history of asthma or allergy, increases the probability that the respiratory symptoms are due to asthma. However, these features are not specific for asthma and are not seen in all asthma phenotypes. Patients with allergic rhinitis or atopic dermatitis should be asked specifically about respiratory symptoms.

Physical examination

Physical examination in people with asthma is often normal. The most frequent abnormality is expiratory wheezing (rhonchi) on auscultation, but this may be absent or only heard on forced expiration. Wheezing may also be absent during severe asthma exacerbations, due to severely reduced airflow (so called ‘silent chest’), but at such times, other physical signs of respiratory failure are usually present.

Physical examination 2

Wheezing may also be heard with upper airway dysfunction, chronic obstructive pulmonary disease (COPD), respiratory infections, tracheomalacia, or inhaled foreign body. Crackles (crepitations) and inspiratory wheezing are not features of asthma. Examination of the nose may reveal signs of allergic rhinitis or nasal polyposis.


Patient with respiratory symptoms (Box 1-2)
Are the symptoms typical of asthma?
NO
YES
Detailed history/examination for asthma
History/examination supports asthma diagnosis?
NO
Further history and tests for alternative diagnoses (Box 1-3)
Alternative diagnosis confirmed?
Clinical urgency, and other diagnoses unlikely
YES
Perform spirometry/PEF with reversibility test (Box 1-2)
Results support asthma diagnosis?
NO
YES
Repeat on another occasion or arrange other tests (Box 1-2)
Confirms asthma diagnosis?
NO
Empiric treatment with ICS and prn SABA (Box 3-4)
Review response
Diagnostic testing within 1-3 months (Box 1-4)
YES
NO
YES
Consider trial of treatment for most likely diagnosis, or refer for further investigations
Treat for ASTHMA
Treat for alternative diagnosis


F1
F2
F3
F4
Esc
Del
Micro
Medical

Lung function testing to document variable expiratory airflow limitation

Asthma is characterized by variable expiratory airflow limitation, i.e. expiratory lung function varies over time and in magnitude to a greater extent than in healthy populations. In asthma, lung function may vary between completely normal and severely obstructed in the same patient. Poorly controlled asthma is associated with greater variability in lung function than well-controlled asthma.

Lung function testing should be carried out by well-trained operators with well-maintained and regularly calibrated equipment.

Forced expiratory volume in 1 second (FEV1) from spirometry is more reliable than peak expiratory flow (PEF).

If PEF is used, the same meter should be used each time, as measurements may differ from meter to meter by up to 20%.1

Classification

Severity	Symptom frequency	Night-time symptoms	%FEV_1 of predicted	FEV_1 variability	SABA use
Intermittent	≤2/week	≤2/month	≥80%	<20%	≤2 days/week
Mild persistent	>2/week	3–4/month	≥80%	20–30%	>2 days/week
Moderate persistent	Daily	>1/week	60–80%	>30%	daily
Severe persistent	Continuously	Frequent (7/week)	<60%	>30%	≥twice/day

Peak Flow Results (PEF)

- Red zone
  - 50% or less of personal best
  - Indicates serious problem
  - Definitive action must be taken with health care provider

A reduced FEV1 may be found with many other lung diseases (or poor spirometric technique), but a reduced ratio of FEV1 to FVC indicates airflow limitation.

From population studies, the FEV1/FVC ratio is normally greater than 0.75 to 0.80, and usually greater than 0.90 in children. Any values less than these suggest airflow limitation. Many spirometers now include age-specific predicted values.

In clinical practice, once an obstructive defect has been confirmed, variation in airflow limitation is generally assessed from variation in FEV1 or PEF.

'Variability' refers to improvement and/or deterioration in symptoms and lung function. Excessive variability may be identified over the course of one day (diurnal variability), from day to day, from visit to visit, or seasonally, or from a reversibility test.

'Reversibility' generally refers to rapid improvements in FEV1 (or PEF), measured within minutes after inhalation of a rapid-acting bronchodilator such as 200–400 mcg salbutamol or more sustained improvement over days or weeks after the introduction of effective controller treatment such as ICS.

In a patient with typical respiratory symptoms, obtaining evidence of excessive variability in expiratory lung function is an essential component of the diagnosis of asthma. Some specific examples are:

- • An increase in lung function after administration of a bronchodilator, or after a trial of controller treatment.
- • A decrease in lung function after exercise or during a bronchial provocation test.
- • Variation in lung function beyond the normal range when it is repeated over time, either on separate visits, or on home monitoring over at least 1–2 weeks.

- in adults with respiratory symptoms typical of asthma, an increase or decrease in FEV1 of >12% and >200 mL from baseline, or (if spirometry is not available) a change in PEF of at least 20%, is accepted as being consistent with asthma.

Other tests

- ***Bronchial provocation tests***
- Airflow limitation may be absent at the time of initial assessment in some patients. As documenting variable airflow limitation is a key part of establishing an asthma diagnosis, one option is to refer the patient for bronchial provocation testing to assess airway hyperresponsiveness. This is most often established with inhaled methacholine.

- ***Exhaled nitric oxide***
- Exhaled nitric oxide is a biological marker that correlates with eosinophilic inflammation in asthma.
- Exhaled NO measurement can provide diagnostic and predictive value for a corticosteroid response.

Peak Flow Meters

Peak Flow Monitoring

- Provides objective information
- Documents personal best
- Detects worsening asthma before changes occur
- Useful only if breathing is monitored regularly
- Indicates need for quick-relief medications
- Assists in precipitant identification
- Aids in communication

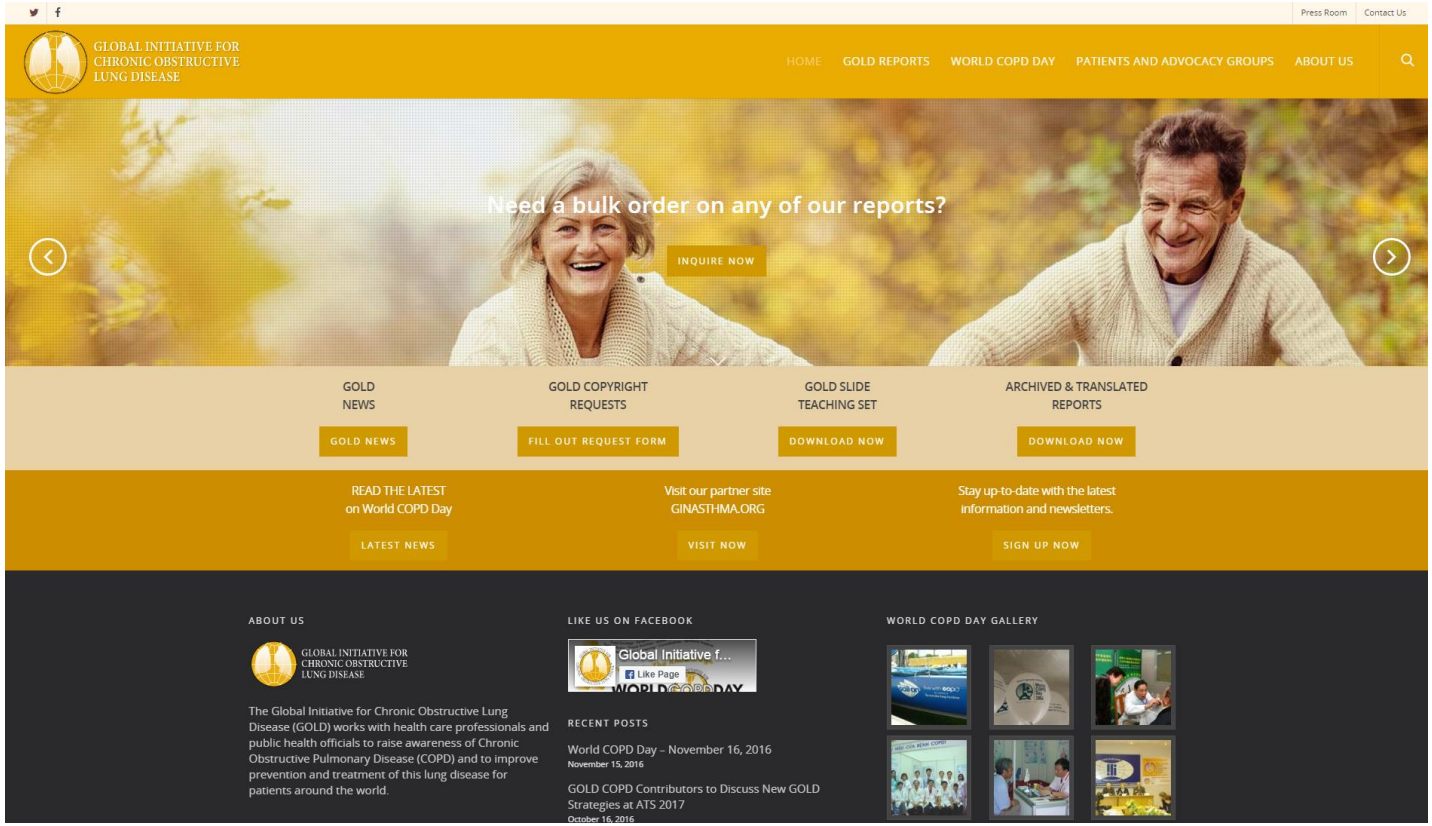
COPD

Chronic obstructive pulmonary disease

GLOBAL INITIATIVE FOR CHRONIC OBSTRUCTIVE LUNG DISEASE (GOLD)

GOLD Website Address

www.goldcopd.org



COPD

- **C**hronic **O**bstructive **P**ulmonary **D**isease

  Chronic Obstructive Pulmonary Disease (COPD) is a common, preventable and treatable disease that is characterized by persistent respiratory symptoms and airflow limitation that is due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases.

Chronic Obstructive Pulmonary Disease (COPD)

- COPD is currently the fourth leading cause of death in the world.[1]
- COPD is projected to be the 3rd leading cause of death by 2020.[2]
- More than 3 million people died of COPD in 2012 accounting for 6% of all deaths globally.
- Globally, the COPD burden is projected to increase in coming decades because of continued exposure to COPD risk factors and aging of the population.

1. Lozano R, Naghavi M, Foreman K, et al. Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet* 2012; **380**(9859): 2095-128.
2. Mathers CD, Loncar D. Projections of global mortality and burden of disease from 2002 to 2030. *PLoS Med* 2006; **3**(11): e442.

Prevalence

Prevalence of COPD

- Estimated 384 million COPD cases in 2010.
- Estimated global prevalence of 11.7% (95% CI 8.4%–15.0%).
- Three million deaths annually.
- With increasing prevalence of smoking in developing countries, and aging populations in high-income countries, the prevalence of COPD is expected to rise over the next 30 years.
- By 2030 predicted 4.5 million COPD related deaths annually.

Understanding COPD

- Critical to first understand normal lung function

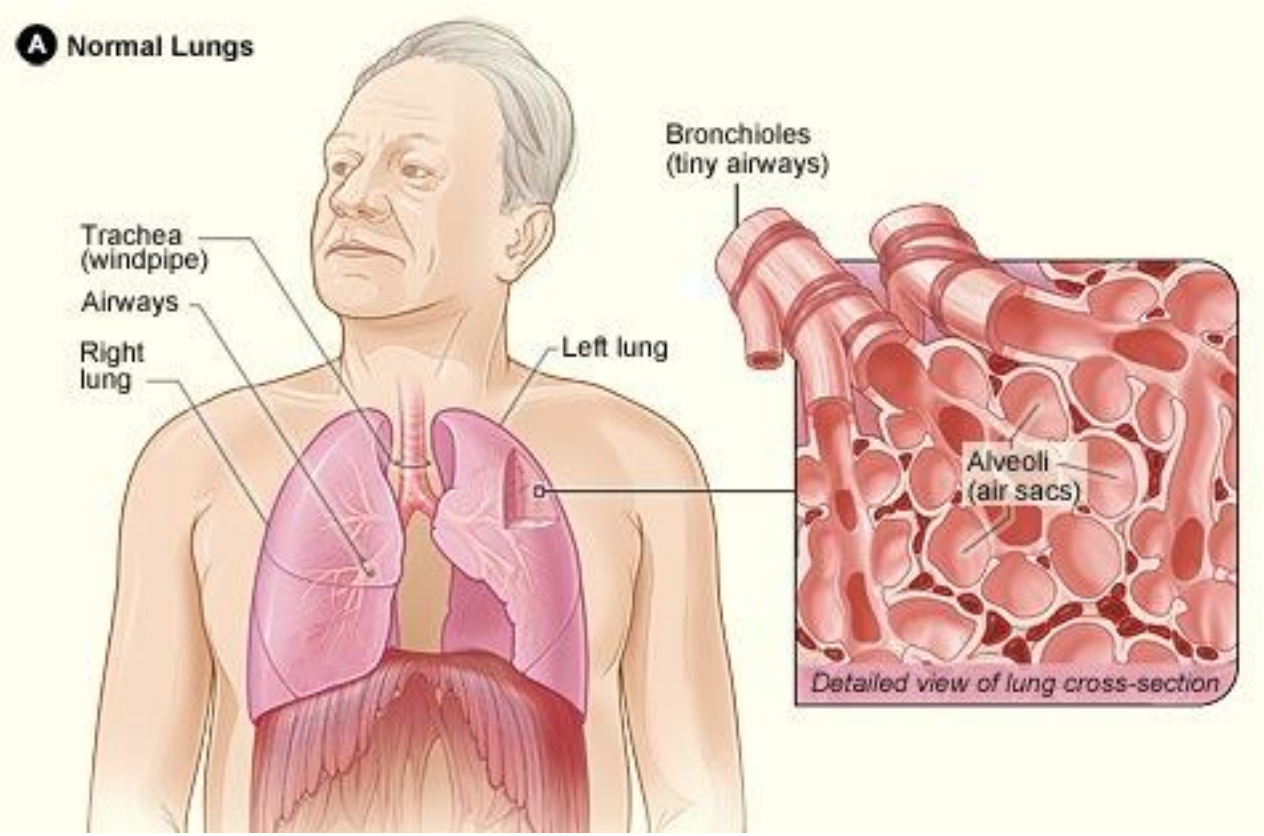
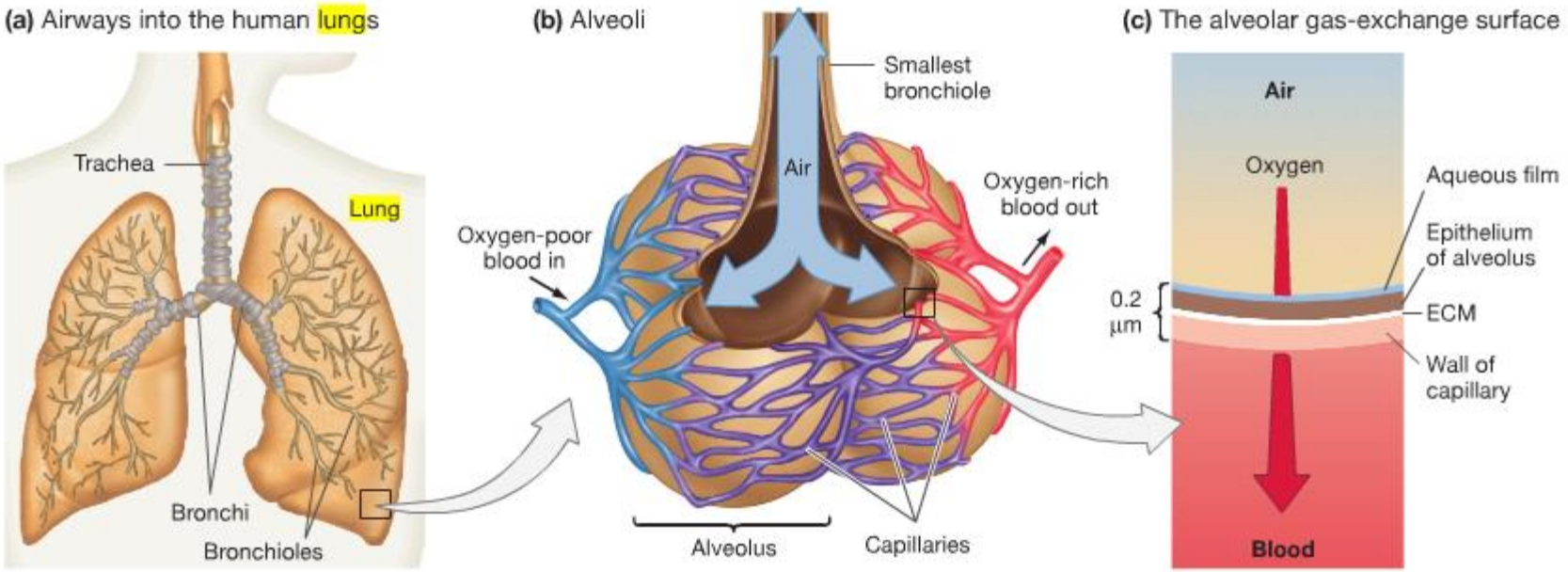


Image courtesy of The National Institute of health

Lung structure and function



"Biological Science Freeman", 2010

Lungs with copd

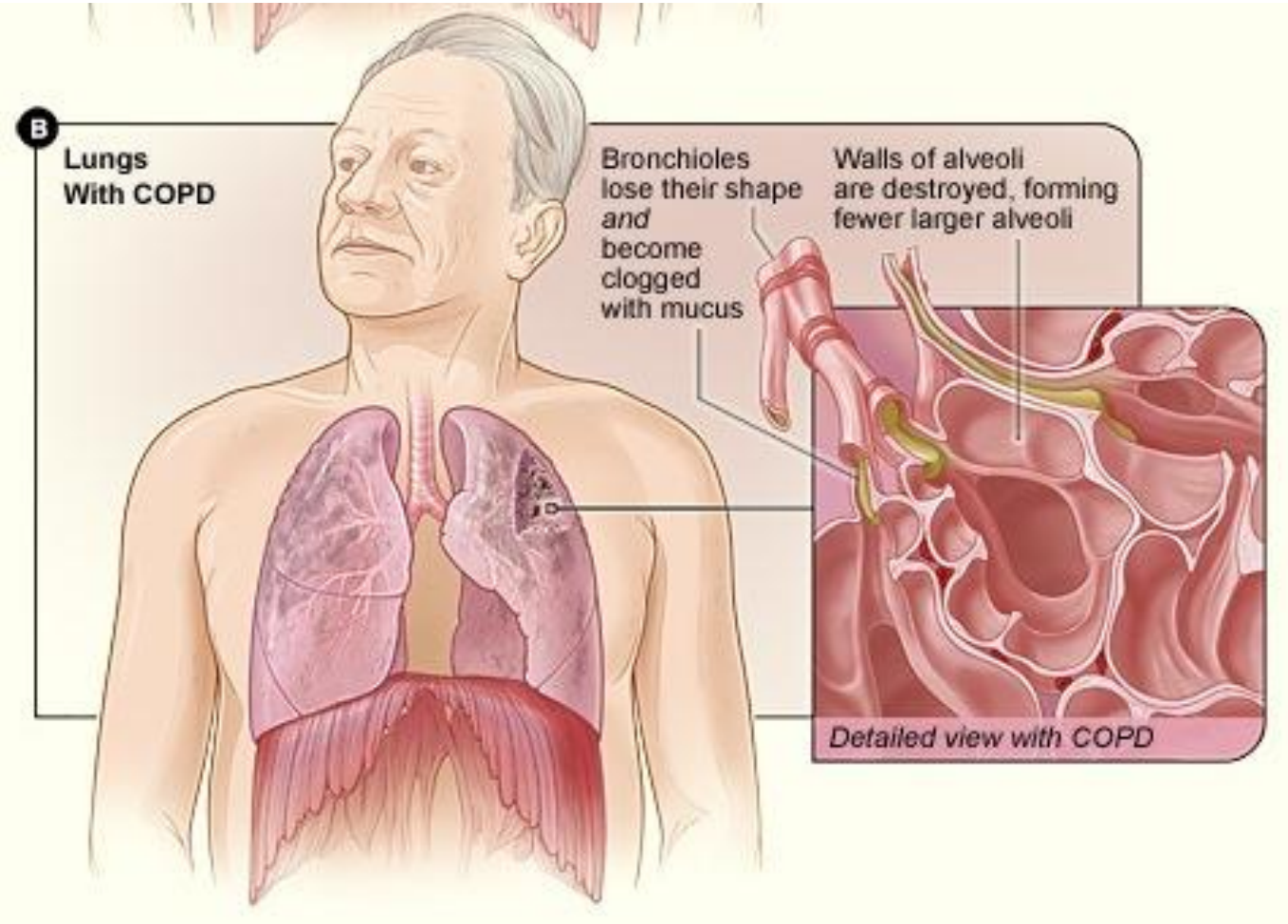
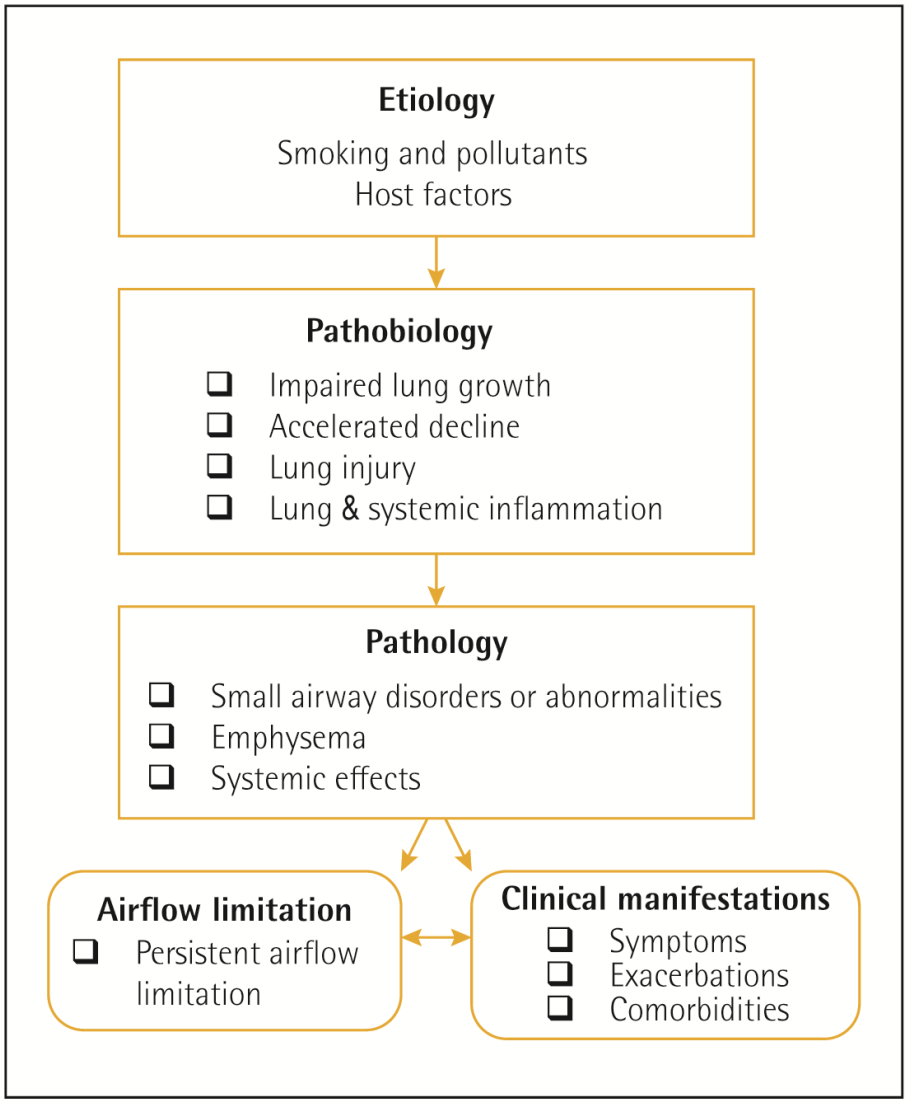


Image courtesy of The National Institute of health

COPD Etiology, Pathobiology & Pathology

Figure 1.1. Etiology, pathobiology and pathology of COPD leading to airflow limitation and clinical manifestations



Causes

- Most cases of COPD occur as a result of long-term exposure to lung irritants that damage the lungs and the airways

- The most common irritant that causes COPD cigarette smoke

- In rare cases, a genetic condition called alpha-1 antitrypsin deficiency may play a role in causing COPD

Who is at risk?

- People who smoke or are exposed to smoke
- People who have a family history of COPD are more likely to develop the disease if they smoke
- Long-term exposure to other lung irritants also is a risk factor for COPD
- Almost 90% of COPD deaths occur in low- and middle-income countries, where effective strategies for prevention and control are not always implemented or accessible.

Factors that influence disease progression

- Genetic factors
- Age and gender
- Lung growth and development
- Exposure to particles
- Socioeconomic status
- Asthma & airway hyper-reactivity
- Chronic bronchitis
- Infections

Symptoms

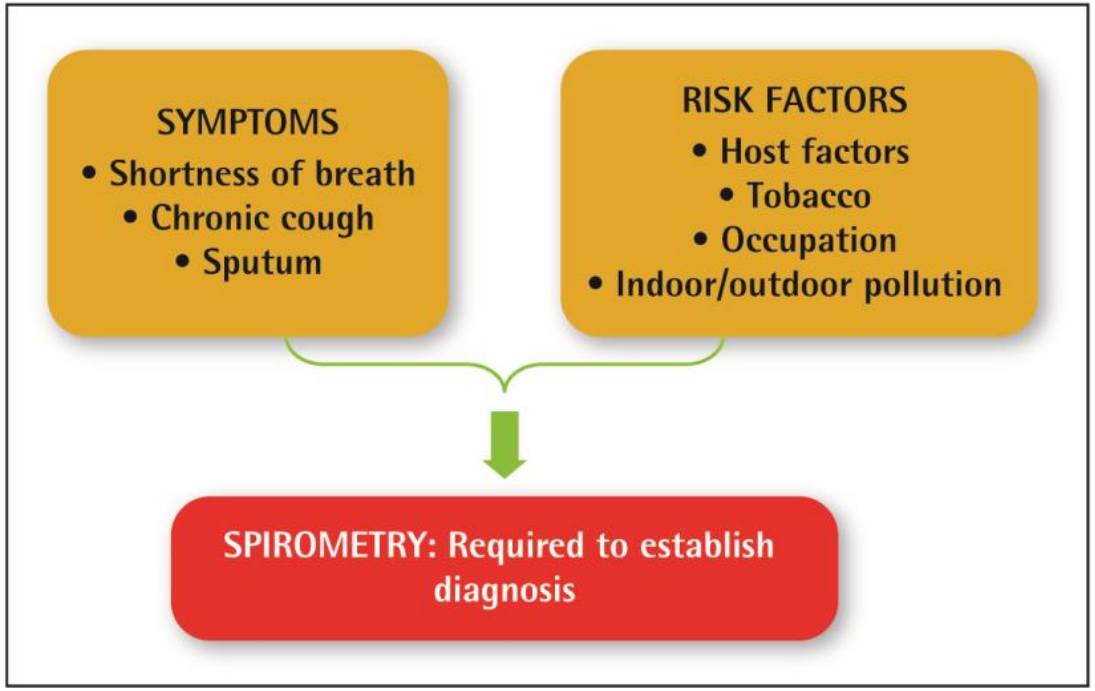
- The most common respiratory symptoms include dyspnea, cough and/or sputum production. These symptoms may be under-reported by patients.

Physical Exam

- ↑HR, ↓O_2 saturation
- Gen: Barrel-chest, accessory muscle use
- CV: Quiet heart sounds
- Resp: Decreased breath sounds, wheezing, rhonchi, crackles

Diagnosis and Initial Assessment

Figure 2.1. Pathways to the diagnosis of COPD



Spirometry is *required* to make the diagnosis;
the presence of a post-bronchodilator
$FEV_1/FVC < 0.70$ confirms the presence of persistent airflow limitation

Medical History

- Patient's exposure to risk factors
- Past medical history
- Family history of COPD or other chronic respiratory disease.
- Pattern of symptom development
- History of exacerbations or previous hospitalizations for respiratory disorder
- Presence of comorbidities
- Impact of disease on patient's life
- Social and family support available to the patient.
- Possibilities for reducing risk factors, especially smoking cessation.

Diagnosis and Initial Assessment

Table 2.3. Considerations in performing spirometry
Preparation
• Spirometers need calibration on a regular basis.
• Spirometers should produce hard copy or have a digital display of the expiratory curve to permit detection of technical errors or have an automatic prompt to identify an unsatisfactory test and the reason for it.
• The supervisor of the test needs training in optimal technique and quality performance.
• Maximal patient effort in performing the test is required to avoid underestimation of values and hence errors in diagnosis and management.
Bronchodilation
• Possible dosage protocols are 400 mcg short-acting beta$_2$-agonist, 160 mcg short-acting anticholinergic, or the two combined.[a] FEV_1 should be measured 10-15 minutes after a short-acting beta$_2$-agonist is given, or 30-45 minutes after a short-acting anticholinergic or a combination of both classes of drugs.
Performance
• Spirometry should be performed using techniques that meet published standards.[b]
• The expiratory volume/time traces should be smooth and free from irregularities. The pause between inspiration and expiration should be < 1 second.
• The recording should go on long enough for a volume plateau to be reached, which may take more than 15 seconds in severe disease.
• Both FVC and FEV_1 should be the largest value obtained from any of three technically satisfactory curves and the FVC and FEV_1 values in these three curves should vary by no more than 5% or 150 ml, whichever is greater.
• The FEV_1/FVC ratio should be taken from the technically acceptable curve with the largest sum of FVC and FEV_1.
Evaluation
• Spirometry measurements are evaluated by comparison of the results with appropriate reference values based on age, height, sex, and race.
• The presence of a postbronchodilator FEV_1/FVC < 0.70 confirms the presence of airflow limitation.

[a] Pellegrino et al. Eur Respir J 2005; 26(5): 948-68;

[b] Miller et al. Eur Respir J 2005; 26(2): 319-38.

Classification of severity of airflow limitation

Table 2.4. Classification of airflow limitation severity in COPD (Based on post-bronchodilator FEV_1)		
In patients with $FEV_1/FVC < 0.70$:		
GOLD 1:	Mild	$FEV_1 \geq 80\%$ predicted
GOLD 2:	Moderate	$50\% \leq FEV_1 < 80\%$ predicted
GOLD 3:	Severe	$30\% \leq FEV_1 < 50\%$ predicted
GOLD 4:	Very Severe	$FEV_1 < 30\%$ predicted

Choice of thresholds

- COPD Assessment Test (CAT ™)
- Chronic Respiratory Questionnaire (CCQ®)
- St George's Respiratory Questionnaire (SGRQ)
- Chronic Respiratory Questionnaire (CRQ)
- Modified Medical Research Council (mMRC) questionnaire

Figure 2.3. CAT Assessment

For each item below, place a mark (✗) in the box that best describes you currently. Be sure to only select one response for each question.

Example: I am very happy 0 ✗ 2 3 4 5 I am very sad

	SCORE
I never cough 0 1 2 3 4 5 I cough all the time	
I have no phlegm (mucus) in my chest at all 0 1 2 3 4 5 My chest is completely full of phlegm (mucus)	
My chest does not feel tight at all 0 1 2 3 4 5 My chest feels very tight	
When I walk up a hill or one flight of stairs I am not breathless 0 1 2 3 4 5 When I walk up a hill or one flight of stairs I am very breathless	
I am not limited doing any activities at home 0 1 2 3 4 5 I am very limited doing activities at home	
I am confident leaving my home despite my lung condition 0 1 2 3 4 5 I am not at all confident leaving my home because of my lung condition	
I sleep soundly 0 1 2 3 4 5 I don't sleep soundly because of my lung condition	
I have lots of energy 0 1 2 3 4 5 I have no energy at all	
TOTAL SCORE	

Reference: Jones et al. ERJ 2009; 34 (3); 648-54.

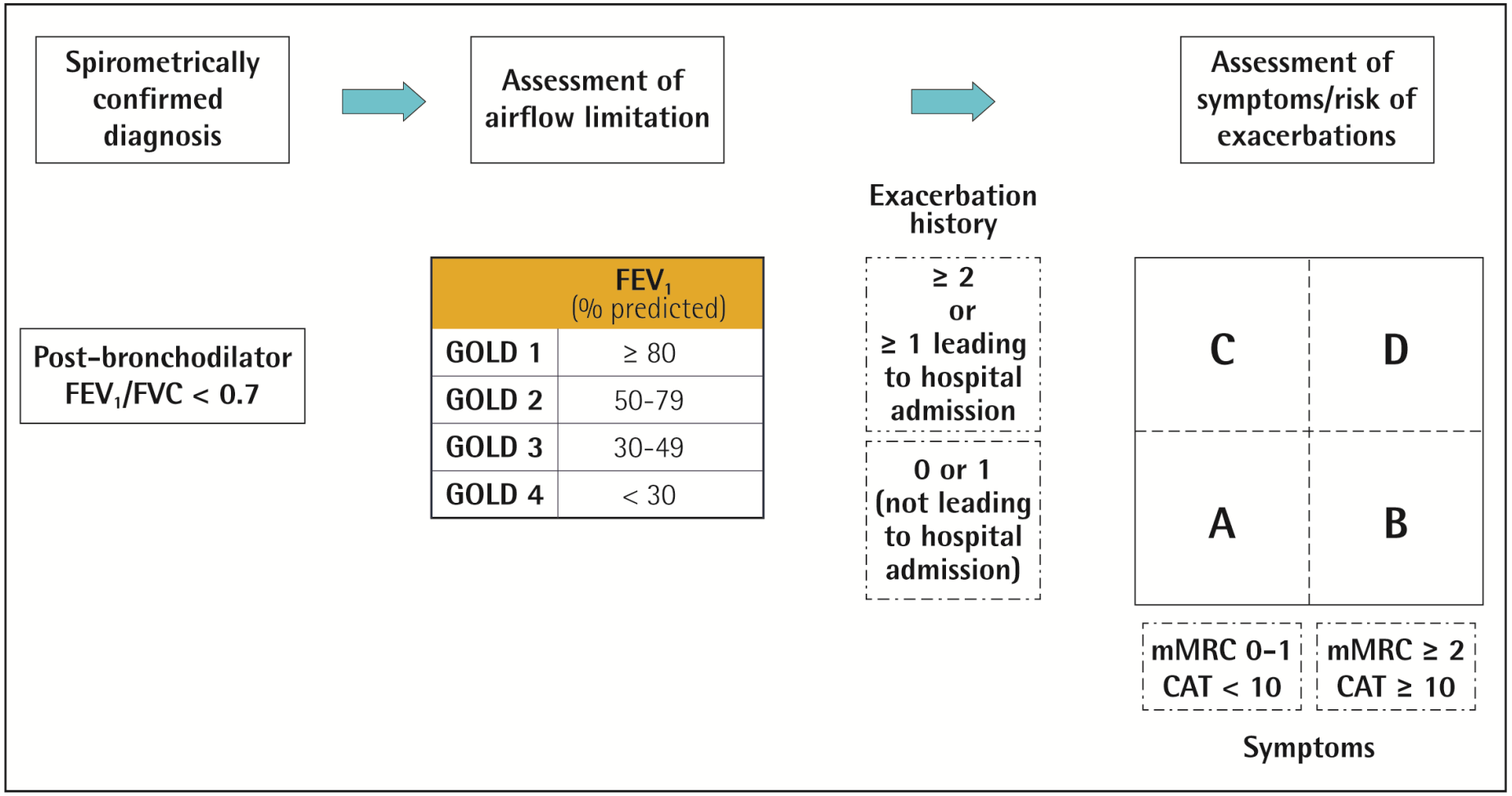
Table 2.5. Modified MRC dyspnea scale[a]

PLEASE TICK IN THE BOX THAT APPLIES TO YOU (ONE BOX ONLY) (Grades 0-4)	
mMRC Grade 0. I only get breathless with strenuous exercise.	❑
mMRC Grade 1. I get short of breath when hurrying on the level or walking up a slight hill.	❑
mMRC Grade 2. I walk slower than people of the same age on the level because of breathlessness, or I have to stop for breath when walking on my own pace on the level.	❑
mMRC Grade 3. I stop for breath after walking about 100 meters or after a few minutes on the level.	❑
mMRC Grade 4. I am too breathless to leave the house or I am breathless when dressing or undressing.	❑

[a] Fletcher CM. BMJ 1960; 2: 1662.

ABCD Assessment Tool

Figure 2.4. The refined ABCD assessment tool



Assessment of Exacerbation Risk

- COPD exacerbations are defined as an acute worsening of respiratory symptoms that result in additional therapy.

- Classified as:
  - Mild (treated with SABDs only)
  - Moderate (treated with SABDs plus antibiotics and/or oral corticosteroids) or
  - Severe (patient requires hospitalization or visits the emergency room). Severe exacerbations may also be associated with acute respiratory failure.

- Blood eosinophil count may also predict exacerbation rates (in patients treated with LABA without ICS).

Smoking Cessation

- Smoking cessation has the greatest capacity to influence the natural history of COPD.
- If effective resources and time are dedicated to smoking cessation, long-term quit success rates of up to 25% can be achieved.

Table 3.1. Brief strategies to help the patient willing to quit

• **ASK:**	Systematically identify all tobacco users at every visit. *Implement an office-wide system that ensures that, for EVERY patient at EVERY clinic visit, tobacco-use status is queried and documented.*
• **ADVISE:**	Strongly urge all tobacco users to quit. *In a clear, strong, and personalized manner, urge every tobacco user to quit.*
• **ASSESS:**	Determine willingness and rationale of patient's desire to make a quit attempt. *Ask every tobacco user if he or she is willing to make a quit attempt at this time (e.g., within the next 30 days).*
• **ASSIST:**	Aid the patient in quitting. *Help the patient with a quit plan; provide practical counseling; provide intra-treatment social support; help the patient obtain extra-treatment social support; recommend use of approved pharmacotherapy except in special circumstances; provide supplementary materials.*
• **ARRANGE:**	Schedule follow-up contact. *Schedule follow-up contact, either in person or via telephone.*

Vaccination

- Influenza vaccination can reduce serious illness (such as lower respiratory tract infections requiring hospitalization)24 and death in COPD patients.
- Pneumococcal vaccinations, PCV13 and PPSV23, are recommended for all patients ≥ 65 years of age

Table 3.2. Vaccination for stable COPD

- Influenza vaccination reduces serious illness and death in COPD patients **(Evidence B)**.
- The 23-valent pneumococcal polysaccharide vaccine (PPSV23) has been shown to reduce the incidence of community-acquired pneumonia in COPD patients aged < 65 years with an FEV_1 < 40% predicted and in those with comorbidities **(Evidence B)**.
- In the general population of adults ≥ 65 years the 13-valent conjugated pneumococcal vaccine (PCV13) has demonstrated significant efficacy in reducing bacteremia and serious invasive pneumococcal disease **(Evidence B)**.

Pharmacologic Therapy

Table 3.3. Commonly used maintenance medications in COPD*					
Drug	**Inhaler (mcg)**	**Solution for nebulizer (mg/ml)**	**Oral**	**Vials for injection (mg)**	**Duration of action (hours)**
Beta$_2$-agonists					
Short-acting					
Fenoterol	100-200 (MDI)	1	2.5 mg (pill), 0.05% (syrup)		4-6
Levalbuterol	45-90 (MDI)	0.1, 0.21, 0.25, 0.42			6-8
Salbutamol (albuterol)	90, 100, 200 (MDI & DPI)†	1, 2, 2.5, 5 mg/ml	2, 4, 5 mg (pill), 8 mg (extended release tablet) 0.024%/0.4 mg (syrup)	0.1, 0.5 mg	4-6, 12 (extended release)
Terbutaline	500 (DPI)		2.5, 5 mg (pill)	0.2, 0.25, 1 mg	4-6
Long-acting					
Arformoterol		0.0075†			12
Formoterol	4.5-9 (DPI)	0.01^			12
Indacaterol	75-300 (DPI)				24
Olodaterol	2.5, 5 (SMI)				24
Salmeterol	25-50 (MDI & DPI)				12
Anticholinergics					
Short-acting					
Ipratropium bromide	20, 40 (MDI)	0.2			6-8
Oxitropium bromide	100 (MDI)				7-9
Long-acting					
Aclidinium bromide	400 (DPI), 400 (MDI)				12
Glycopyrronium bromide	15.6 & 50 (DPI)†		1 mg (solution)	0.2 mg	12-24
Tiotropium	18 (DPI), 2.5 & 5 (SMI)				24
Umeclidinium	62.5 (DPI)				24
Combination of short-acting beta$_2$-agonist plus anticholinergic in one device					
Fenoterol/ipratropium	50/20 (SMI)	1.25, 0.5 mg in 4ml			6-8
Salbutamol/ipratropium	100/20 (SMI), 75/15 (MDI)	0.5, 2.5 mg in 3ml			6-8

Pharmacologic Therapy

Combination of long-acting beta$_2$-agonist plus anticholinergic in one device				
Formoterol/aclidinium	12/400 (DPI)			12
Formoterol/glycopyrronium	9.6/18 (MDI)			12
Indacaterol/glycopyrronium	27.5/15.6 & 110/50 (DPI)[ǂ]			12-24
Vilanterol/umeclidinium	25/62.5 (DPI)			24
Olodaterol/tiotropium	5/5 (SMI)			24
Methylxanthines				
Aminophylline		105 mg/ml (solution)	250, 500 mg	Variable, up to 24
Theophylline (SR)		100-600 mg (pill)	250, 400, 500 mg	Variable, up to 24
Combination of long-acting beta$_2$-agonist plus corticosteroids in one device				
Formoterol/beclomethasone	6/100 (MDI)			
Formoterol/budesonide	4.5/160 (MDI), 4.5/80 (MDI), 9/320 (DPI), 9/160 (DPI)			
Formoterol/mometasone	10/200, 10/400 (MDI)			
Salmeterol/fluticasone	5/100, 50/250, 5/500 (DPI), 21/45, 21/115, 21/230 (MDI)			
Vilanterol/fluticasone furoate	25/100 (DPI)			
Phosphodiesterase-4 inhibitors				
Roflumilast		500 mcg (pill)		

MDI = metered dose inhaler; DPI = dry powder inhaler; SMI = soft mist inhaler

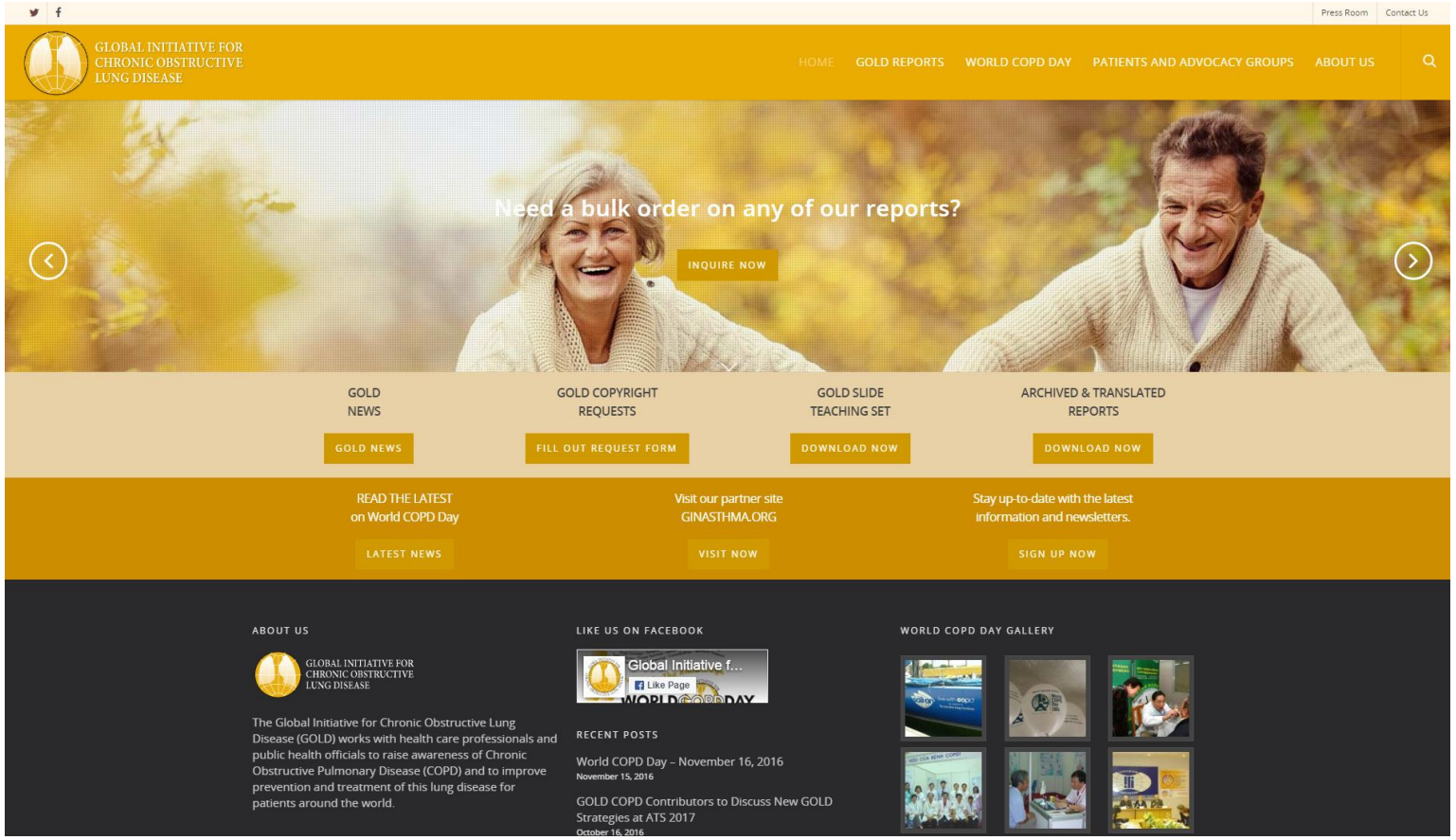
* Not all formulations are available in all countries; in some countries other formulations and dosages may be available

ǂ Dose availability varies by country

^ Formoterol nebulized solution is based on the unit dose vial containing 20 mcg in a volume of 2.0 ml

† Dose varies by country

GOLD Website
www.goldcopd.org



Pneumonia

- **Pneumonia** is an infection of lung parenchyma, which leads to inflammation and exudates filling air spaces with fluid (consolidation). This leads to reduced lung compliance and ventilation-perfusion mismatch or shunt, which results in decreased oxygenation.

Classification:

- **Community-acquired pneumonia** (CAP) acquired outside the hospital; common in nonimmuno-compromised individuals, may be primary or secondary to underlying disease.
- **Hospital-acquired pneumonia** develops after 48-72 hours after hospital admission and is not apparent at admission.
- **Aspiration pneumonia** follows the aspiration of exogenous material or endogenous secretions into the lower respiratory tract. Can be in patients with stroke, myasthenia, bulbar palsies, diminished consciousness (post-ictal, drunk), oesophageal disease (achalasia, reflux), or with poor dental hygiene, risk aspirating oropharyngeal anaerobes.
- **Pneumonia at immunocompromised patients** (AIDS, prolonged systemic corticosteroids and other immunosuppressive therapy)

CAP - Why do we care about it?

- 5.6 million cases annually
- 1.1 million require hospitalization
- Mortality rate =12% in-hospital; near 40% in ICU patients

- **Causes of CAP:**
- S. pneumoniae, M. pneumoniae, C. pneumoniae, H. influenzae, S. aureus, Legionella spp., M. catarrhalis, Gram-negative bacilli and Viruses.

CAP – Modifying Factors

Table 6. Epidemiologic conditions related to specific pathogens in patients with community-acquired pneumonia

Condition	Community Encountered Pathogens
Alcoholism	Streptococcus pneumoniae (including DRSP), anaerobes, gram-negative bacilli, tuberculosis
COPD/smoker	S. pneumoniae, Hemophilus influenzae, Moraxella catarrhalis, Legionella
Nursing home residency	S. pneumoniae, gram-negative bacilli, H. influenzae, Staphylococcus aureus, anaerobes, Chlamydia pneumoniae, tuberculosis
Poor dental hygiene	Anaerobes
Epidemic Legionnaire's disease	Legionella species
Exposure to bats	Histoplasma capsulatum
Exposure to birds	Chlamydia psittaci, Cryptococcus neoformans, H. capsulatum
Exposure to rabbits	Francisella tularensis
Travel to southwest United States	Coccidiodomycosis
Exposure to farm animals or parturient cats	Coxiella burnetii (Q fever)
Influenza active in community	Influenza, S. pneumoniae, S. aureus, H. influenzae
Suspected large-volume aspiration	Anaerobes, chemical pneumonitis, or obstruction
Structural disease of the lung (bronchiectasis, cystic fibrosis, etc.)	P. aeruginosa, Pseudomonas cepacia, or S. aureus
Injection drug use	S. aureus, anaerobes, tuberculosis, Pneumocystis carinii
Endobronchial obstruction	Anaerobes
Recent antibiotic therapy	Drug-resistant pneumococci, P. aeruginosa

Am J Respir Crit Care Med 163:1730-54, 2001

CAP – Modifying Factors

MODIFYING FACTORS THAT INCREASE THE RISK OF INFECTION WITH SPECIFIC PATHOGENS

Penicillin-resistant and drug-resistant pneumococci
- Age > 65 yr
- B-Lactam therapy within the past 3 mo
- Alcoholism
- Immune-suppressive illness (including therapy w/ corticosteroids)
- Multiple medical comorbidities
- Exposure to a child in a day care center

Enteric gram-negatives
- Residence in a nursing home
- Underlying cardiopulmonary disease
- Multiple medical comorbidities
- Recent antibiotic therapy

Pseudomonas aeruginosa
- Structural lung disease (bronchiectasis)
- Corticosteroid therapy (10 mg of prednisone per day)
- Broad-spectrum antibiotic therapy for > 7 d in the past month
- Malnutrition

Am J Respir Crit Care Med 163:1730-54, 2001

- **Clinical features of lobar CAP**
- Breathlessness occurs due to large injury of lung that results to diminution of it function and changing of gas content of blood.
- Chest pain happens due to involving of pleura in inflammatory process.
- Cough can be dry or with purulent bloody sputum. It is depended from stage or form of disease.
- Fever, rigors, malaise, anorexia

- **Signs of lobar CAP**
- *Visual examination*: It is severe condition of the patient; confusion (may be the only sign in the elderly). He has redness in cheek at the affected side, herpes on his lips, sometimes prefers to lie on the affected side. There is tachypnea, cyanosis, tachycardia, hypotension.
- *Visual examination of the chest:* There is limitation of the chest moving at the affected side. Auxiliary muscles take part in breathing.
- *Palpation of the chest*
- There is a tenderness of the pleural points, positive Potendzher symptom, because large part of lung with pleura is involved to inflammatory process and surrounded tissues react to this. It is obtained amplifying of voice resonance according to the affected lobe or segments, because consolidated lung tissue conducts acoustic waves better than normal one.

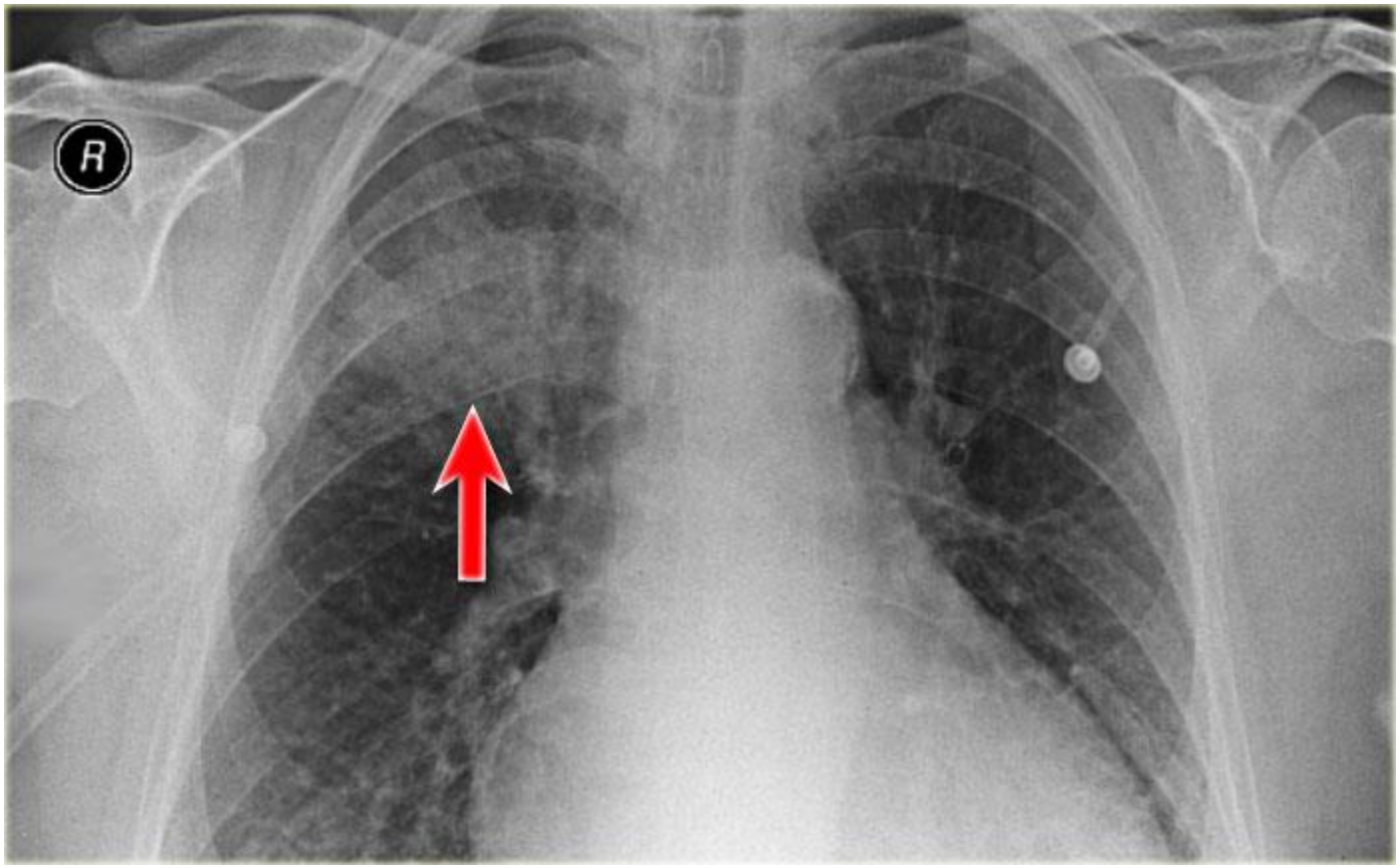
- **X-ray signs of lobar CAP:** There is intensive and homogeneous infiltration of lobe or segments.

- *Percussion of the chest*
- *Comparative percussion*: Over the consolidated lobe the percussion sound is dull because only solid components of infiltrated lung tissue get to percussion sphere.
- *Topographic percussion:* The lower border of the affected lung lifts up if pathological process localizes in lower lobe. The height of lung apex pulls down if infiltration of the upper lobe is presented. But size of the lung doesn't change. It is obtaining due to increasing of solidity of lung tissue. There is dimension of lower lung border excursion.
- *Auscultation of the lung*
- There is pathological bronchial breathing because all alveoli are filled up with inflammatory exudates, whispering pectoriloquy. There is pleural rub due to inflamed pleura rubbing against each other. You can hear depressed vesicular or bronchial breathing and crepitations in the beginning or end of pathological process.

- **Lobar CAP has three stages**: rising tide, high point and resolution.
- During **rising tide** inflammation process is begun and alveolus walls are impregnated with exudation fluid became adhesive. When air get to them alveoli fill out with sound due to sticking off. This added sound is named crepitations (crackles). The main sound is diminished vesicular during this stage. By percussion you can obtain dullness with tympanic inflection.
- During **high point** alveoli full up with exudates and air cannot gets to them. Vesicular breathing isn't formed, crackles disappear, but consolidation lung conducts bronchial breathing and whispering speech from vocal cords. By percussion you can obtain dullness.
- During **resolution** exudates gradually disappears from alveoli and changes of their walls same as the first stage. By percussion and auscultation you can receive similar data.

- **Signs of the focal CAP:**
- *Visual examination*
- It is satisfactory or moderate serious. Sometimes there is limitation of the chest moving at the affected side.
- *Palpation of the chest*
- May be, there is a tenderness of the pleural points, positive Potendzher symptom, amplifying of voice resonance if the site of consolidation is near surface of chest.
- *Percussion of the chest*
- *Comparative percussion*: If site of consolidation is near of the chest surface it can be dullness of percussion sound. Because account of solid components of lung tissue increase due to infiltration but air presents in alveoli which haven't been involved to pathological process and they get to percussion sphere. If site of consolidation is deeply in lung percussion sound is clear without change.

- *Topographic percussion:* Some change can be if site of consolidation is near of chest surface. There is dimension of lower lung border excursion.
- *Auscultation of the lung*
- There is diminished vesicular breathing because less alveoli involve to act of breathing. There are sonorous bubbling (moist) rales. They are formed in bronchus which around by consolidated lung tissue. Such tissue conduct sounds from bronchus better then healthy and we can hear them as sonorous. Sometimes if site of consolidation is near chest surface crepitations can be heard.
- **X-ray signs of the focal CAP:** There is peribronchial and perivascular and/or focal infiltration of lung tissue.



Lobar pneumonia

On the chest x-ray there is an ill-defined area of increased density in the right upper lobe without volume loss.

The right hilus is in a normal position.

In the proper clinical setting this is most likely a lobar or segmental pneumonia.

However if this patient had weight loss or long standing symptoms, we would include the list of causes of chronic consolidation.

This was an acute lobar pneumonia caused by Streptcoccus pneumoniae.

Severity of PNA

- Severity-of-illness scores can help guide whether a pt needs hospital admission and should always be supplemented with clinical judgement

- **CURB-65 criteria**
  - **C**onfusion
  - **U**rea >19 mg/dL
  - **R**espiratory rate ≥30
  - **B**lood pressure (SBP <90 or DBP ≤60)
  - **≥65** year old
  - ≥2 criteria then needs hospital admission and ≥3 criteria may need ICU level of care

- Can also use Pneumonia Severity Index (PSI) instead of CURB-65

- **Data of additional methods of examination of CAP**:
- *Assess oxygenation*: oxygen saturation (if SaO_2 <92% analysis of blood gas)
- *Blood test:*
- Full blood count: leukocytosis, left shift of blood formula – increasing young forms of neutrophiles.
- Urine and enzymes (ALT, AST, LDH), C-reactive protein are increased if CAP severe.
- Blood culture and/or sputum culture are positive if patient has bacteriemia and purulent sputum.

TOPIC: RESPIRATORY SYSTEM

Title of the seminar: Instrumental methods of examination (x-ray, spirometry, peak flow meter, pulse oximetry)

Name of the tutor: Kristian Debretseni

Duration: 90 min

Audience: 3rd course students

Goals:

General: To raise the level of knowledge, skills of instrumental research methods of basic respiratory diseases.

Special goals:

- Knowledge: Interpretation of the results of instrumental methods of examination.
- Practical skills: To learn the basics of instrumental methods of examination, practice to perform spirometry in respiratory patients.
- Changing attitude: Instrumental methods of functional diagnosis are widely used in pulmonology. They help us to determine pathological changes more accurately and conduct early diagnostics of respiratory diseases.

Methodology of the seminar:

5 minutes Short introduction

5 minutes Ice breaker

20 minutes Theory presentation (Power Point presentation, video demonstrations)

40 minutes Interactive
Practical training - spirometry procedure presentation, work in pairs

10 minutes Control – verbal interview

10 minutes Feedback from each participant: take home notes: “What was new for me today? What did I learn/practice? What will I introduce into my future practice?”

Results: we expect each participant:

- Master the basic knowledge about Instrumental methods of examination.
- Practice to perform spirometry.

Spirometry

What is Spirometry?

Spirometry is a method of assessing lung function by measuring the total volume of air the patient can expel from the lungs after a maximal inhalation.

Introduction

- A non-invasive method for evaluation of pulmonary function
- Simple, cost-effective, accessible
- Not for definite diagnosis of disease but help diagnosis along with history, physical examination and other paraclinical diagnostic method

TABLE 1 Indications for spirometry

Diagnostic

- To evaluate symptoms, signs or abnormal laboratory tests
- To measure the effect of disease on pulmonary function
- To screen individuals at risk of having pulmonary disease
- To assess pre-operative risk
- To assess prognosis
- To assess health status before beginning strenuous physical activity programmes

Monitoring

- To assess therapeutic intervention
- To describe the course of diseases that affect lung function
- To monitor people exposed to injurious agents
- To monitor for adverse reactions to drugs with known pulmonary toxicity

Disability/impairment evaluations

- To assess patients as part of a rehabilitation programme
- To assess risks as part of an insurance evaluation
- To assess individuals for legal reasons

Public health

- Epidemiological surveys
- Derivation of reference equations
- Clinical research

Contraindications of Spirometry

- Uncontrolled hypertension
- Suspected presence of active TB other communicable respiratory disease
- Thoracic or abdominal surgery within recent 3 wks
- MI or unstable angina within recent 6 wks
- Respiratory distress
- Active hemoptysis
- Recent eye/ear surgery or ear drum perforation
- Abdominal or thoracic aortic aneurysm

Confounding factors

- Common cold (3 days ego)
- Severe respiratory infection (3w)
- Smoking(1hr)
- Heavy food (1hr)
- Bronchodilator use

Complications

- Chest pain
- Syncope, dizziness
- Increased ICP
- Paroxysmal coughing
- Nosocomial infection
- Bronchospasm

Spirometry Parameters

- Forced Vital Capacity
  - FVC
- Forced Expiratory Volume in One Second
  - FEV1
- Forced Expiratory Volume in One Second Expressed as a Percentage of the Forced Vital Capacity
  - FEV1/FVC %
- Mean Forced Expiratory Flow during the Middle Half of the Forced Vital Capacity
  - FEF 25-75%

FVC

- Definition:
  - Defined as the maximal amount of air that can be exhaled **forcefully** after a maximal inspiration or the most air a person can blow out after taking the deepest possible breath.

FVC - forced vital capacity

- defines maximum volume of exchangeable air in lung (vital capacity)
  - forced expiratory breathing maneuver
  - requires muscular effort and some patient training
- initial (healthy) FVC values approx 4 liters
  - slowly diminishes with normal aging
- significantly reduced FVC suggests damage to lung parenchyma
  - restrictive lung disease (fibrosis)
  - loss of functional alveolar tissue (atelectasis)
- intra-subject variability factors
  - age
  - sex
  - height
  - ethnicity

FEV1

- Definition:
  - The volume of air exhaled during the first second of a **forced** expiratory maneuver.
  - normal FEV1 about 3 liters

FEV1/FVC%

- Definition:
  - The value expresses the volume of air the worker exhales in one second as a percent of the total volume of air that is exhaled.
  - Calculated by using **largest** valid FEV1 and **largest** FVC even if they are not from the same tracing.
    - Find largest valid FEV1
    - Find largest valid FVC
    - Divide FEV1 by FVC
    - Multiply by 100 to obtain percentage.

FEF25-75%

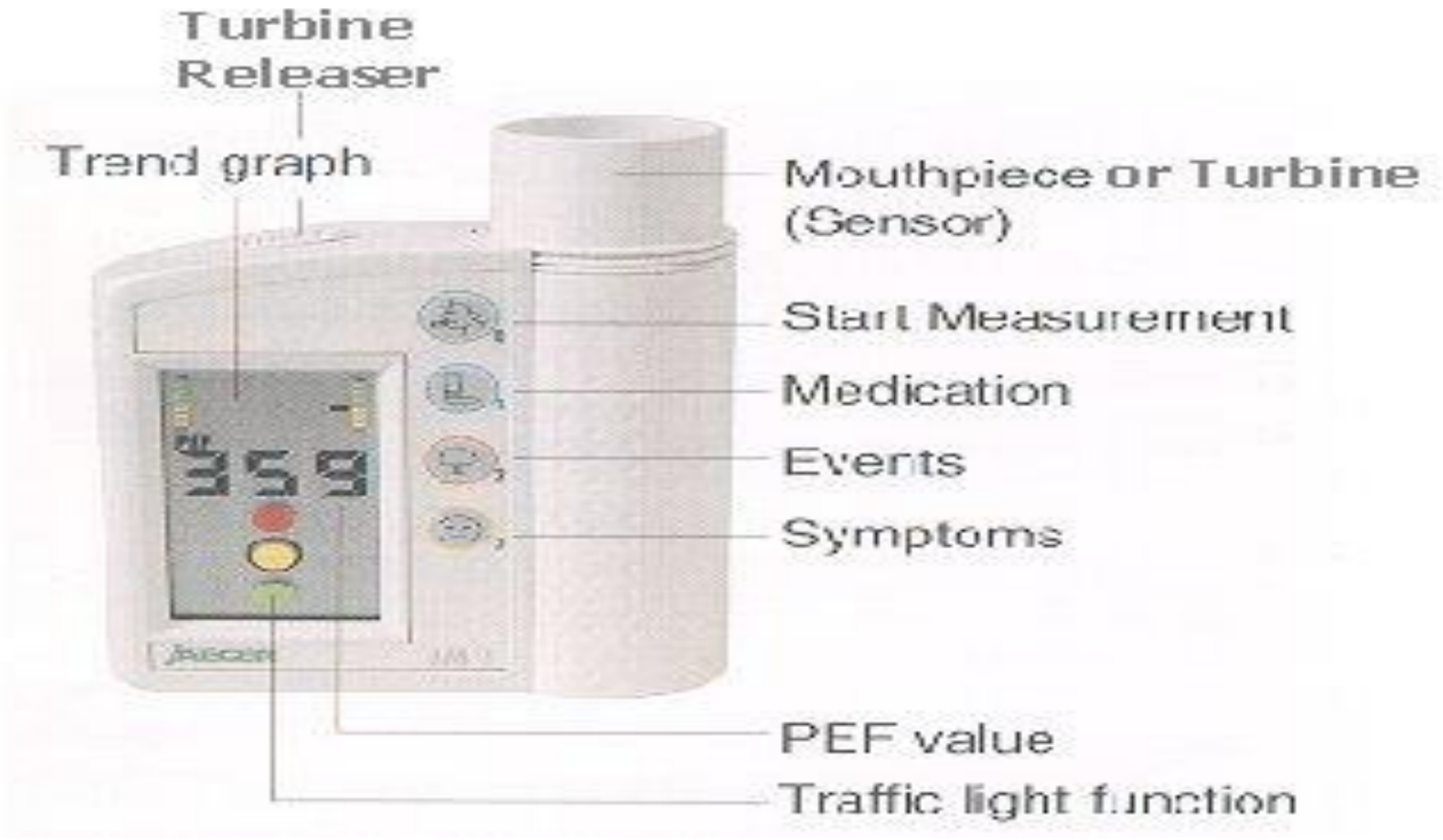
- Definition:
  - The mean expiratory flow during the middle half of the FVC
    - More sensitive than FEV1.
    - Considerably more variability than FVC and FEV1.
    - ATS recommends only be considered after determining presence and clinical severity of impairment and should not be used to diagnosis disease in individual patients

PEF - Peak Expiratory Flow rate

- measures airflow limitations in large (central) airways
- PEF measurements recommended for asthma management
  - spirometry is recommended to help make the diagnosis of asthma
- PEF not recommend to evaluate patients for COPD
  - cannot measure small airway airflow limitations
- advantages of PEF tests
  - measurements within a minute (three short breaths)
  - uses simple, safe, hand-held devices that typical, costs $20
- disadvantages of PEF tests (compared to spirometry)
  - insensitive to obstruction of small airways (mild or early obstruction)
  - PEF is very dependent on patient effort (large intra-subject variability)
  - mechanical PEF meters are much less accurate than spirometers

mechanical

electronic


Turbine
Releaser
Trend graph
Mouthpiece or Turbine
(Sensor)
Start Measurement
Medication
Events
Symptoms
PEF value
Traffic light function

The Original Wright Peak Flow Meter - Standard and Low Range versions

The Mini Wright Peak Flow Meter - Standard and Low Range versions (from left to right: Wright scale, EU scale, ATS scale)

Other designs of peak flow meters are available

Most important parameters

- FEV1
- FVC

$$\frac{FEV_1}{FVC}\,\text{ratio}$$

- PEF
- FEF25-75%
- V-T Curve
- F-V loop

FIGURE 2-3. FVC AND FEV_1 ON A NORMAL VOLUME TIME CURVE

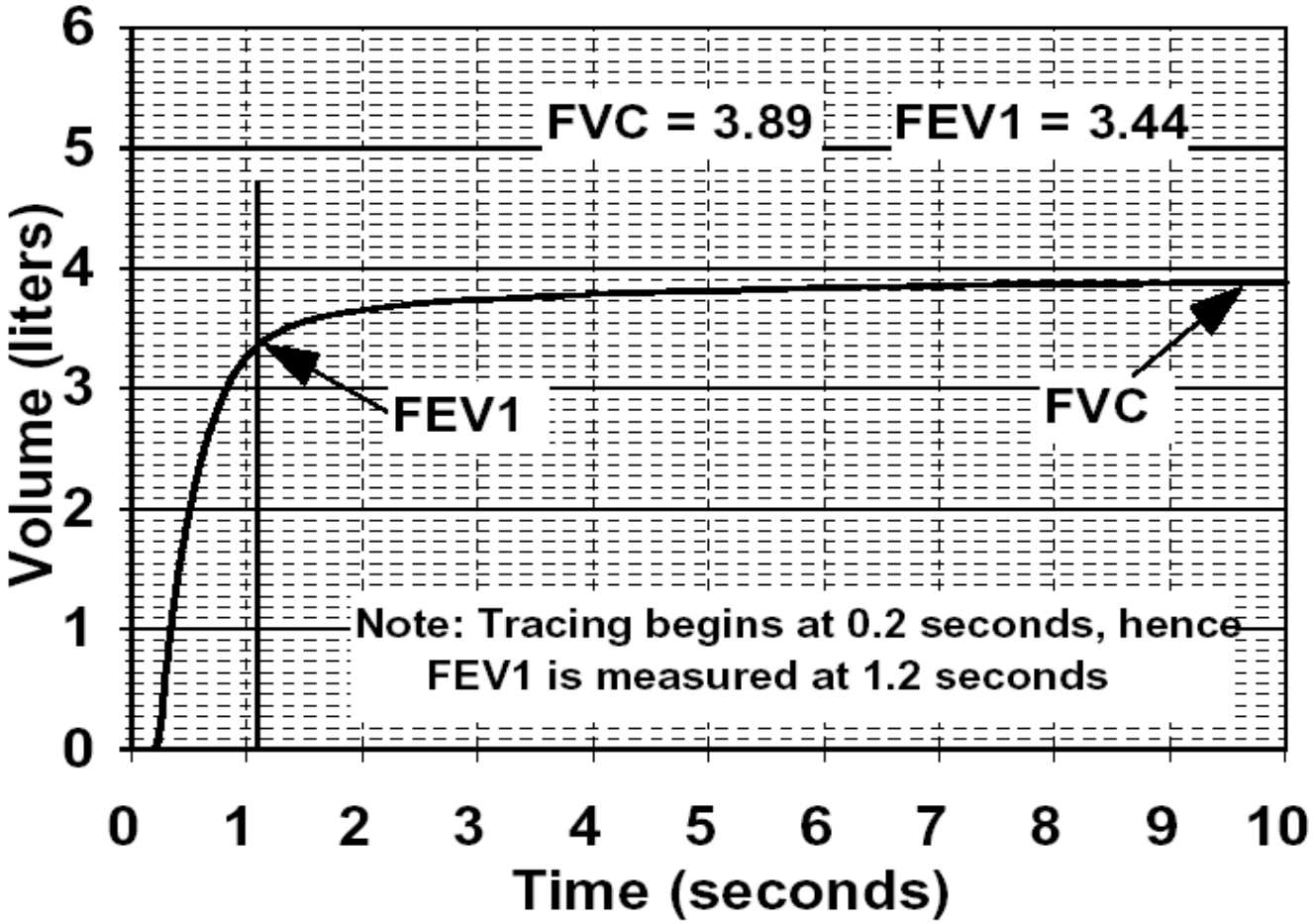
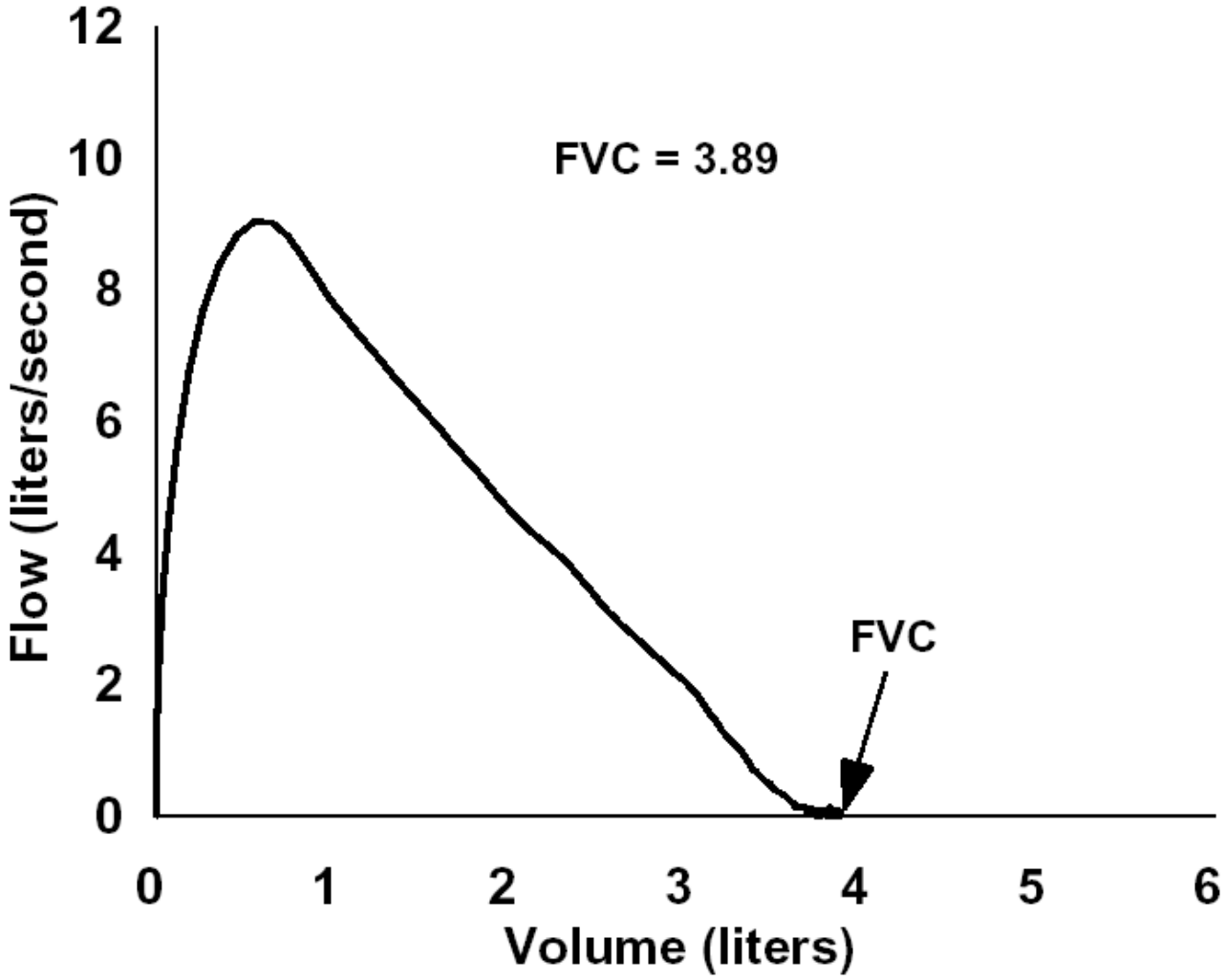
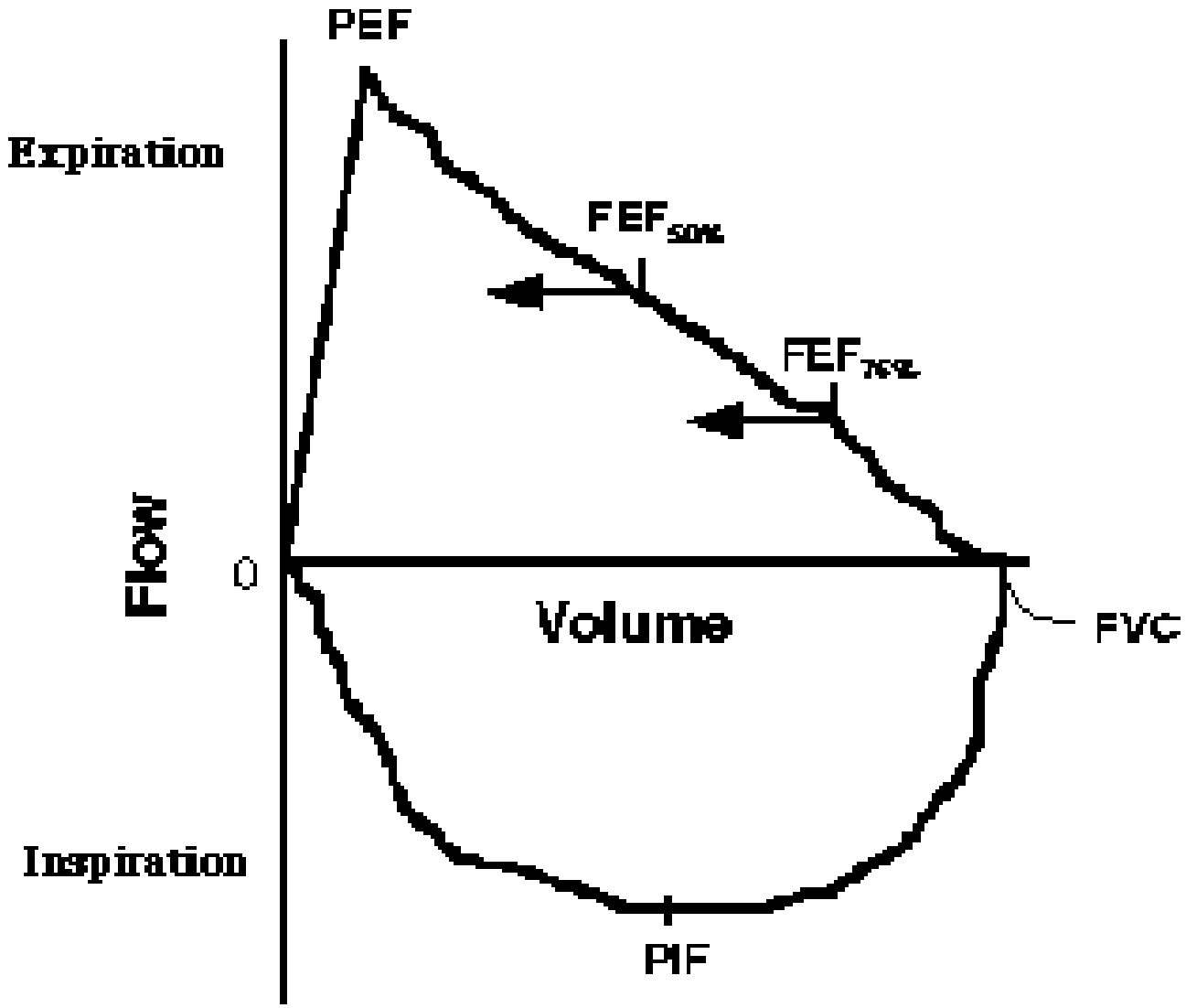


FIGURE 2-4. FVC AND FEV_1 ON A NORMAL FLOW VOLUME CURVE


12
10
8
6
4
2
0
Flow (liters/second)
FVC = 3.89
FVC
0
1
2
3
4
5
6
Volume (liters)


PEF
Expiration
FEF50%
FEF75%
Flow
0
Volume
FVC
Inspiration
PIF

Spirometric Flow Diagram

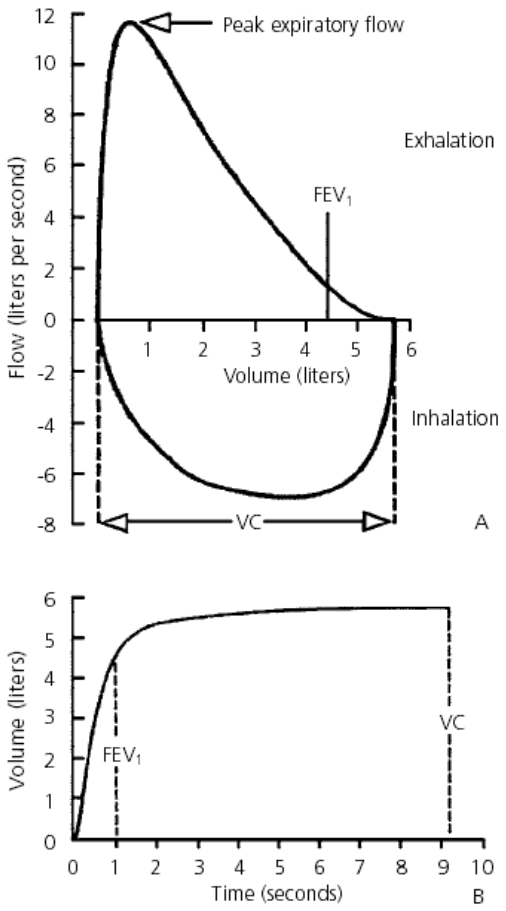


FIGURE 4. Normal spirometric flow diagram. *(A)* Flow-volume curve. *(B)* Volume-time curve. The smooth lines, expiratory time of greater than six seconds, and quick peak of the peak expiratory flow rate indicate a good spirometric effort.

Spirometry Performance Steps

- Equipment performance criteria
- Equipment quality control (calibration & leak)
- Contraindications & interfering condition
- Age, height, race, gender
- Selection of appropriate reference value
- Patient maneuver
- Acceptability criteria
- Reproducibility criteria
- Selection of best curve and best result
- interpretation

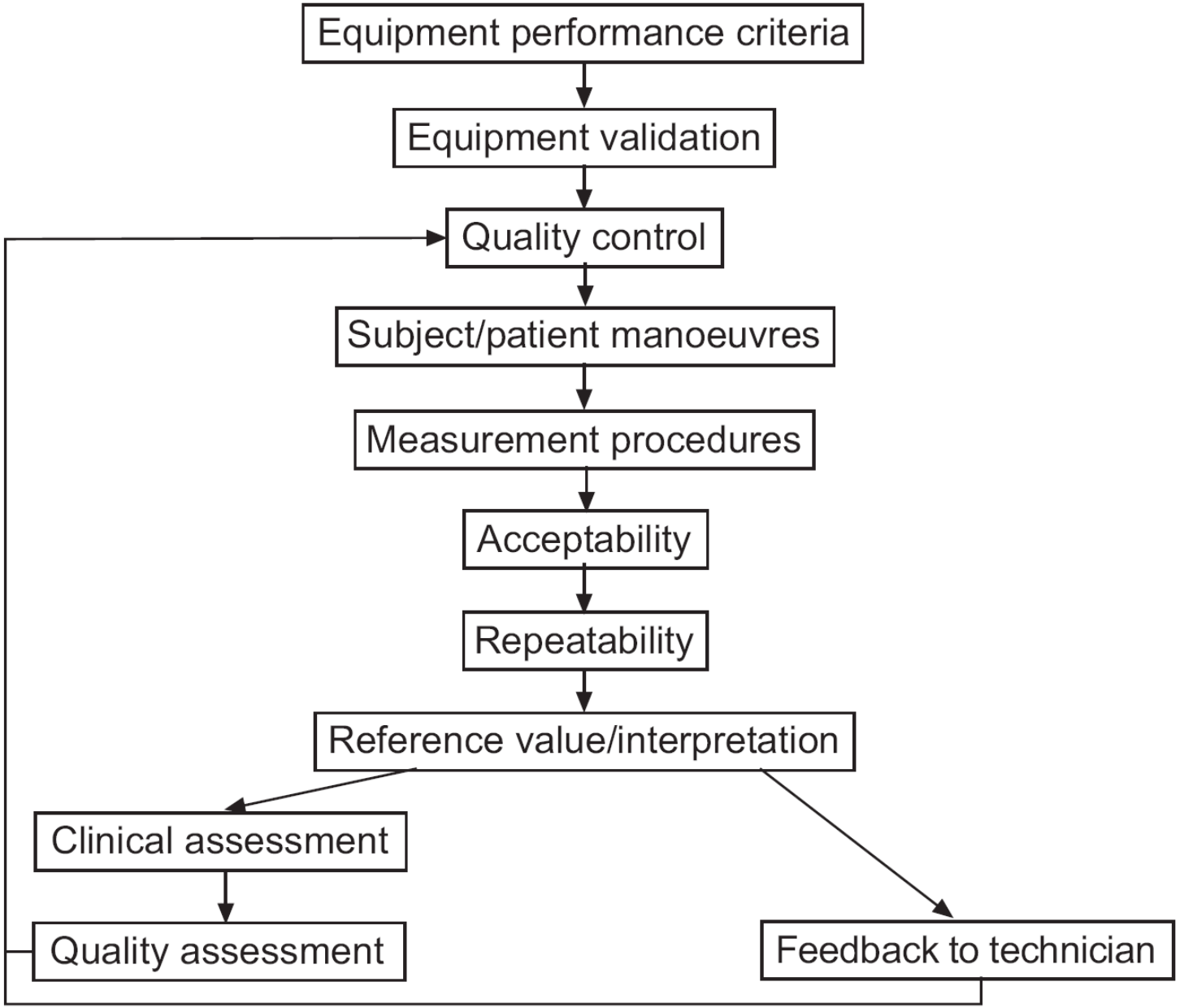


FIGURE 1. Spirometry standardisation steps.

Subject's position:

1. Sitting or standing?
2. Chair with arms & without wheels
3. Clothing
4. Chin & neck position
5. Nose clip
6. Denture

TABLE 5 Summary of within- and between-manoeuvre acceptability criteria

Within-manoeuvre criteria

- Individual spirograms are ''acceptable'' if
  - They are free from artefacts [3]
    - Cough during the first second of exhalation
    - Glottis closure that influences the measurement
    - Early termination or cut-off
    - Effort that is not maximal throughout
    - Leak
    - Obstructed mouthpiece
  - They have good starts
    - Extrapolated volume $<5\%$ of FVC or 0.15 L, whichever is greater
  - They show satisfactory exhalation
    - Duration of $\geqslant 6$ s (3 s for children) or a plateau in the volume–time curve or
      - If the subject cannot or should not continue to exhale

Between-manoeuvre criteria

- After three acceptable spirograms have been obtained, apply the following tests
  - The two largest values of FVC must be within 0.150 L of each other
  - The two largest values of FEV_1 must be within 0.150 L of each other
- If both of these criteria are met, the test session may be concluded
- If both of these criteria are not met, continue testing until
  - Both of the criteria are met with analysis of additional acceptable spirograms
    - or
  - A total of eight tests have been performed (optional) or
  - The patient/subject cannot or should not continue
- Save, as a minimum, the three satisfactory manoeuvres

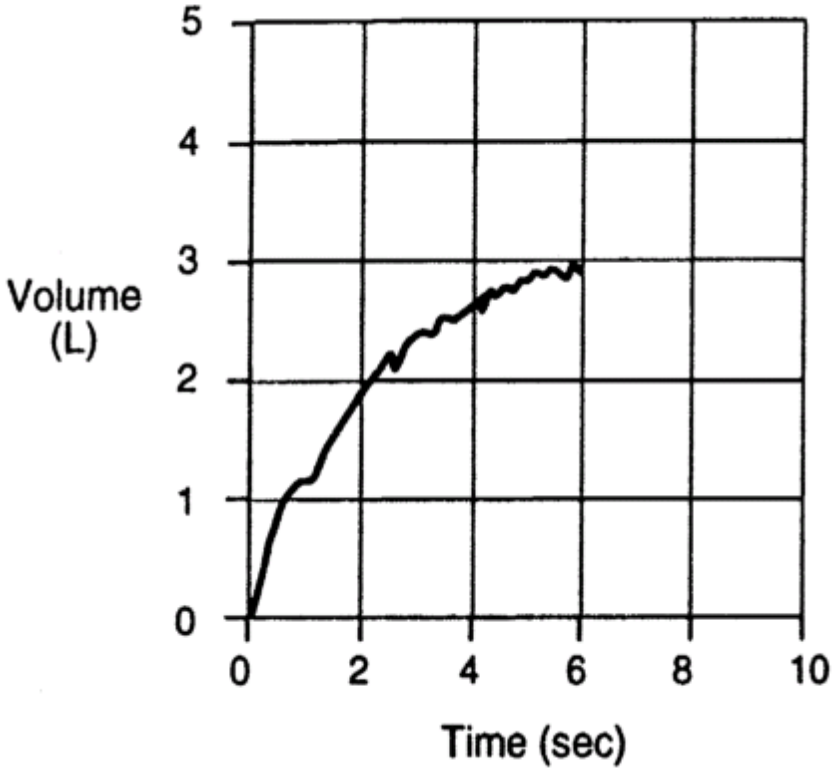
Good Start of Test

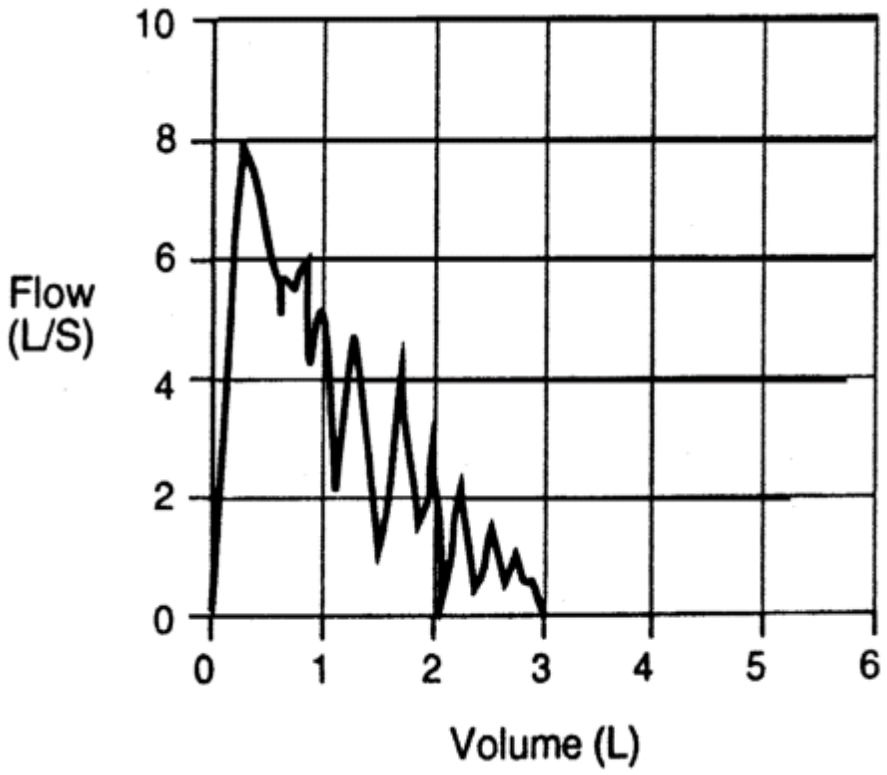
- Start of test must be quick and forceful
- No excessive hesitation
- Best evaluated using the Flow-Volume tracing
- No excessive back extrapolated volume

No Coughing

- Especially during the first 1 second of the maneuver
- Best if no coughing present during maneuver, however:
  - Some patients cough near the end of each test, if present then document

No Coughing


Volume
(L)
5
4
3
2
1
0
0
2
4
6
8
10
Time (sec)


Flow
(L/S)
10
8
6
4
2
0
0
1
2
3
4
5
6
Volume (L)

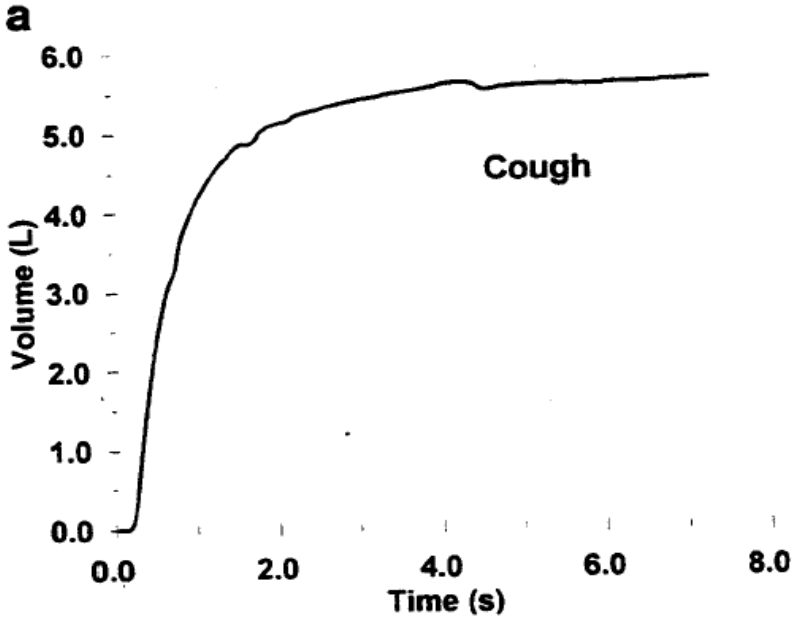


Figure A2a. Volume–time spirogram with a cough during the first second of exhalation.

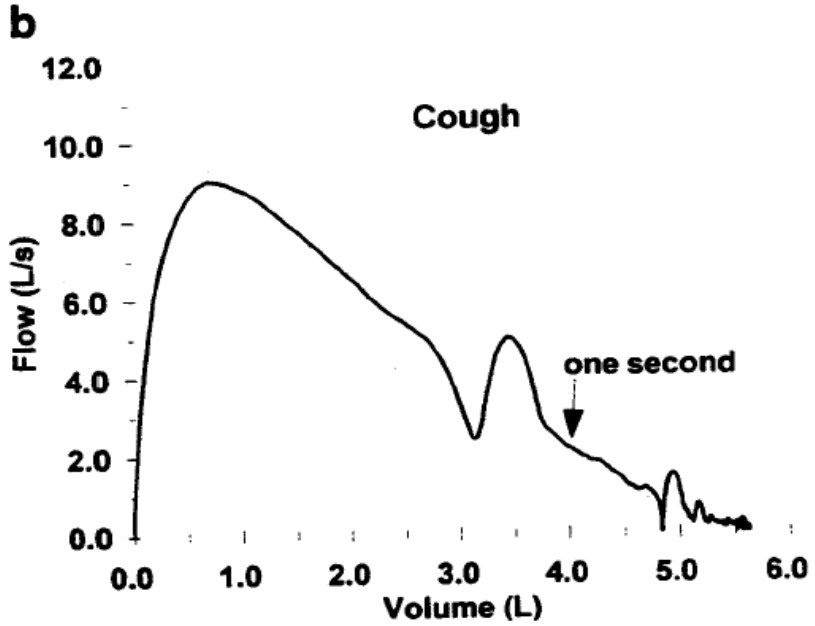


Figure A2b. Flow–volume spirogram with a cough during the first second of exhalation.

No Early Termination of Effort

- Best if maneuver lasts at least six (6) seconds
- Less than six seconds acceptable if a plateau of al least one (1) second is present
- If patient is unable to meet the above criteria, document in comment section

No Early Termination of Effort

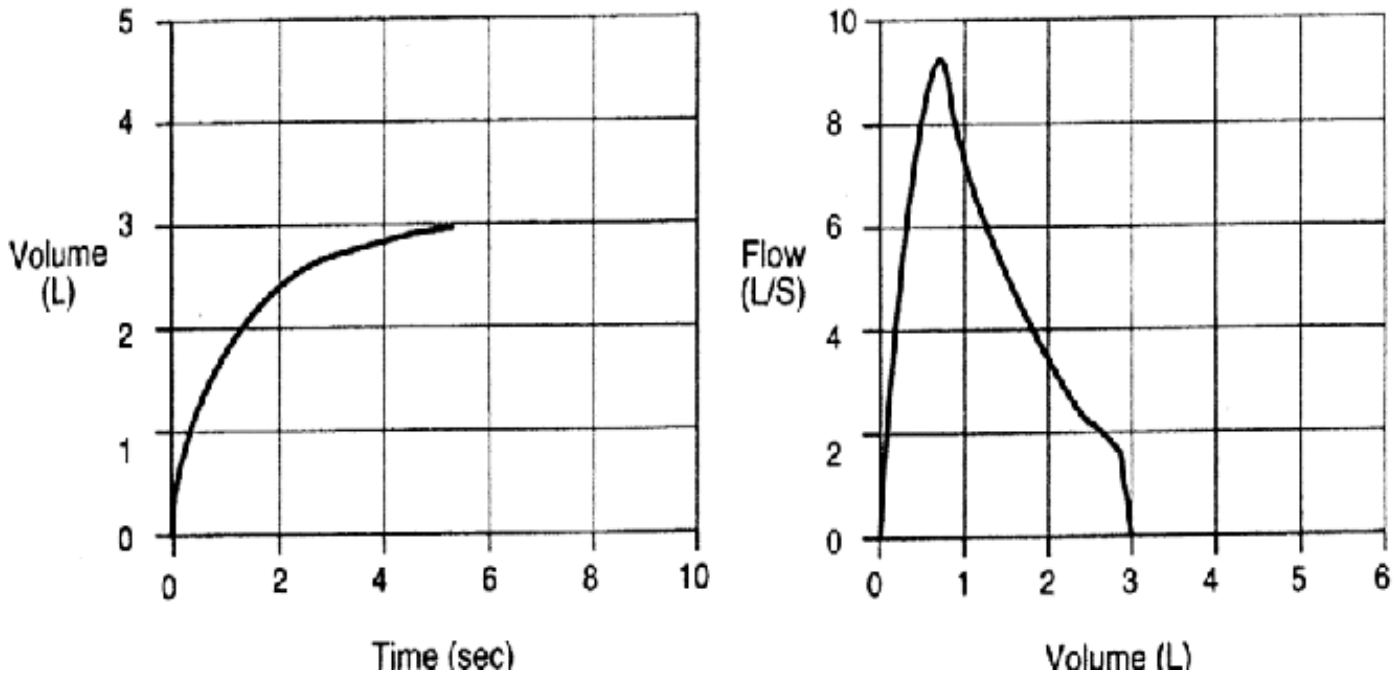


Table 2 – Messages Related to Patient's Effort

Message	Criteria	Explanation	Abbreviation
DON'T HESITATE	BEV>150ml	Patient hesitated after beginning the expiration.	
BLAST OUT FASTER	PEFT>120ms	The effort to produce a maximal peak flow was insufficient.	Time
BLOW OUT LONGER	FET<6.0 sec and EOTV>100ml	The patient stopped blowing before 6 seconds even though at least 0.1L of exhalable air was in their lungs.	
BLAST OUT HARDER	PEF values do not match within 1.0 L/sec.	If several tests were done and the peak flow variability is too high, it indicates that the effort was insufficient.	
DEEPER BREATH	FEV6 values do not match within 0.150 L.	If several tests were done and the FEV6 variability is too high, it indicates that the patient is taking inconsistent deep breaths before each effort.	
BEV = Back Extrapolated Volume in ml **EOTV** = End of test volume – change in volume during the last 0.5 seconds of the maneuver. **PEFT** = Time to peak flow in msec **FET** = Forced Expiratory Time in seconds Only one QC message is displayed, in order of top to bottom priority shown in the table above. After 2 acceptable maneuvers that match, the QC message is GOOD TEST SESSION.			

Selection of measures:

1. Report the largest FVC & FEV1, even if they are not from the same tracing.
2. Calculate FEV1/FVC by dividing the above parameters.
3. All flow rates from one acceptable tracing with largest sum of FEV1 + FVC.

PREDICTED NORMAL VALUES

These values apply to Caucasians. Reduce values by 7% for Asians and by 13% for Afro-Caribbeans.

Male			Height						
			5'3" 160cm	5'5" 165cm	5'7" 170cm	5'9" 175cm	5'11" 180cm	6'1" 185cm	6'3" 190cm
Age	38 - 41 years	FVC	3.81	4.10	4.39	4.67	4.96	5.25	5.54
		FEV_1	3.20	3.42	3.63	3.85	4.06	4.28	4.49
	42 - 45 years	FVC	3.71	3.99	4.28	4.57	4.86	5.15	5.43
		FEV_1	3.09	3.30	3.52	3.73	3.95	4.16	4.38
	46 - 49 years	FVC	3.60	3.89	4.18	4.47	4.75	5.04	5.33
		FEV_1	2.97	3.18	3.40	3.61	3.83	4.04	4.26
	50 - 53 years	FVC	3.50	3.79	4.07	4.36	4.65	4.94	5.23
		FEV_1	2.85	3.07	3.28	3.50	3.71	3.93	4.14
	54 - 57 years	FVC	3.39	3.68	3.97	4.26	4.55	4.83	5.12
		FEV_1	2.74	2.95	3.17	3.38	3.60	3.81	4.03
	58 - 61 years	FVC	3.29	3.58	3.87	4.15	4.44	4.73	5.02
		FEV_1	2.62	2.84	3.05	3.27	3.48	3.70	3.91
	62 - 65 years	FVC	3.19	3.47	3.76	4.05	4.34	4.63	4.91
		FEV_1	2.51	2.72	2.94	3.15	3.37	3.58	3.80
	66 - 69 years	FVC	3.08	3.37	3.66	3.95	4.23	4.52	4.81
		FEV_1	2.39	2.60	2.82	3.03	3.25	3.46	3.68

For men over 70 years predicted values are less well established but can be calculated from the equations below (height in cms; age in years):

FVC = (0.0576 x height) - (0.026 x age) - 4.34 (SD: ± 0.61 litres)

FEV_1 = (0.043 x height) - (0.029 x age) - 2.49 (SD: ± 0.51 litres)

Female			Height						
			4'11" 150cm	5'1" 155cm	5'3" 160cm	5'5" 165cm	5'7" 170cm	5'9" 175cm	5'11" 180cm
Age	38 - 41 years	FVC	2.69	2.91	3.13	3.35	3.58	3.80	4.02
		FEV_1	2.30	2.50	2.70	2.89	3.09	3.29	3.49
	42 - 45 years	FVC	2.59	2.81	3.03	3.25	3.47	3.69	3.91
		FEV_1	2.20	2.40	2.60	2.79	2.99	3.19	3.39
	46 - 49 years	FVC	2.48	2.70	2.92	3.15	3.37	3.59	3.81
		FEV_1	2.10	2.30	2.50	2.69	2.89	3.09	3.29
	50 - 53 years	FVC	2.38	2.60	2.82	3.04	3.26	3.48	3.71
		FEV_1	2.00	2.20	2.40	2.59	2.79	2.99	3.19
	54 - 57 years	FVC	2.27	2.49	2.72	2.94	3.16	3.38	3.60
		FEV_1	1.90	2.10	2.30	2.49	2.69	2.89	3.09
	58 - 61 years	FVC	2.17	2.39	2.61	2.83	3.06	3.28	3.50
		FEV_1	1.80	2.00	2.20	2.39	2.59	2.79	2.99
	62 - 65 years	FVC	2.07	2.29	2.51	2.73	2.95	3.17	3.39
		FEV_1	1.70	1.90	2.10	2.29	2.49	2.69	2.89
	66 - 69 years	FVC	1.96	2.18	2.40	2.63	2.85	3.07	3.29
		FEV_1	1.60	1.80	2.00	2.19	2.39	2.59	2.79

For women over 70 years predicted values are less well established but can be calculated from the equations below (height in cms; age in years):

FVC = (0.0443 x height) - (0.026 x age) - 2.89 (SD: ± 0.43 litres)

FEV_1 = (0.0395 x height) - (0.025 x age) - 2.60 (SD: ± 0.38 litres)