Specific symptoms - history

Cough - productive (with sputum)
- non productive (dry cough)

hemoptysis
hemoptoe

Dyspnea

Wheezing

Chest pain

Voice changes – hoarse, rancous, croaking, rattling

Breathing

Eupnoea means normal breathing with a frequency of 16 - 18 breaths per minute.

A. Respiratory frequency
B. Respiratory depth – periodical breathing
C. Mode of breathing – intercostal muscle
abdominal muscle , diaphragm

Tachypnoea - manifests by increased breath frequency e.g. in anger, pain, fever, or in bronchopulmonary and cardiac disorders.

Bradypnoea - means decreased breath frequency, e.g. in alcohol poisoning or intracranial hypertension.

Apnoea - means halted breathing, may be temporary or permanent (death).

Hyperpnoea - means deepened breathing, e.g. fever, severe anaemia, or acidosis, also called Kussmaul respiration (decompensated diabetes mellitus, uraemia).

Periodical breathing (Cheyne-Stokes breathing) is characterised by an increasing speed and deepness of breathing, followed by speed and deepness decrease, apnoea appears and the cycle repeats. It occurs with severe cerebral apoplexy. During sleep it can be a marker of incipient left sided heart insufficiency.

Dyspnea - breathlessness

It is a subjective feel of air deficiency, which need not express any objective sign.

Physiologically it originates at excessive physical load, pathologically it is related to many diseases:

Obstructive breathlessness - is conditioned by an obstruction in respiratory tract (mucus), by spasm (chronic obstructive bronchopulmonary disease, bronchial asthma)

Restrictive breathlessness - accompanies infiltrative processes (bronchopneumonia) or depressing by aneurysm, eventually atelectasis

Other reasons of breathlessness - metabolic disorders (diabetic coma, uraemia), heart diseases.

According to clinical manifestation we recognise:

Inspiratory breathlessness - with more difficult inspiring (aspiration of foreign body, stenosis of larynx, compression of trachea and bronchi

Expiratory breathlessness - with remarkably prolonged expiring (bronchial asthma).

Dyspnea - breathlessness

We ask all patients complaining of dyspnea the following questions:

- for how long have you had shortness of breath ?
  did the shortness of breath occur suddenly ?
- is it constant ? Does it occur at rest? with exertion ?
  Lying flat ? Sitting up ?
- what makes the shortness of breath worse ?What relieves it ?
- how many level blocks can you walk without becoming short of breath ?
  How many could you walk 6 months ago ?(progression of disease?)
- is the shortness of breath accompanied by wheezing ? Fever? Cough?
  Coughing up blood?Hoarseness?Chest pain?
- do you smoke? If so,how much? For how long?
- exposure to an individual with tbc ? Any industrial exposure
  (dust,asbestos) ?

Table 6.5 Some causes of dyspnoea

	Acute dyspnoea at rest	Chronic exertional dyspnoea
Cardiovascular system	Acute pulmonary oedema Pulmonary embolus Major neonatal congenital heart disease	Chronic cardiac failure Chronic pulmonary thromboembolism Congenital heart disease
Respiratory system	Acute severe asthma Acute exacerbation of COPD Pneumothorax Pneumonia Adult Respiratory Distress Syndrome Inhaled foreign body (especially in the child) Lobar collapse Laryngeal oedema (e.g. anaphylaxis)	COPD 'Chronic asthma' Bronchial carcinoma Interstitial lung diseases: sarcoidosis, fibrosing alveolitis, extrinsic allergic alveolitis, pneumconiosis Lymphatic carcinomatosis (may cause intolerable dyspnoea) Large pleural effusion(s)
Others	Metabolic acidosis (e.g. diabetic ketoacidosis, lactic acidosis, uraemia, overdose of salicylates or ethylene glycol)	Severe anaemia

PHYSIOLOGICAL BASIS OF DYSPNOEA

Increased ventilatory rate

- ↑$PaCO_2$—e.g. COPD
- ↓PaO_2—e.g. cyanotic congenital heart disease, asthma, COPD
- Acidaemia—e.g. diabetic ketoacidosis, lactic acidosis
- Exercise
- Fever

Reduced ventilatory capacity

- ↓Lung volume, e.g. restrictive lung diseases—pneumonia, pulmonary oedema, interstitial lung diseases
- ↑Resistance to airflow, e.g. asthma, COPD, upper airway or laryngeal obstruction
- Pleural pain

Cough - history

COUGH is the most common symptom of lung disease. The cough reflex is a normal defense mechanism that serves to protect the lungs from foreign bodies and excessive secretions. Coughing is a coordinated forced expiration interrupted by repeated closure of the glottis The expiratory muscles contract against the partially closed glottis, creating high pressure within the lungs. When the glottis suddenly opens, there is an explosive rush of air that clears the air passages When a patient complains of coughing it is important to ask these **questions:**

- can you describe your cough ?
  how long have you had a cough ?
- was there a sudden onset of coughing ?
- do you smoke ? If so, how much and for how long ?
- does your cough produce sputum (phlegm) ? If so, can you estimate the amount of your expectorations ? (a teaspoon, spoon, or littler pot) What is the color of the sputum ? Does the sputum have a foul odor (bad smell) ?
- does the cough occur for prolonged periods ?
  does the cough occur after eating or drinking ?
- cough is most marked in the morning (in smokers)
  nocturnal (congestive heart failure)
- is the coughing worse in any position ?
- what relieves the cough ?
- are there any other symptoms associated with the cough ? Fever ? Headaches ? Night sweats ? Chest pain ? A runny nose ? Shortness of breath ? Weight loss ? Hoarseness (husky voice) ? Loss of cousciousness ?
- have you ever been exposed to anyone with tuberculosis ?

Coughing may be voluntary or involuntary, productive or nonproductive In a productive cough mucus (phlegm, sputum) or other materials are expelled
A dry cough does not produce any secretions.
Possible causes of non productive (dry, hacking) cough: viral infection, tumor, allergies, interstitial lung disease -may also be psychogenic in individuals with emotional stress-all other causes have to be eliminated before this dg is made !!
Causes of chronic productive cough: chronic bronchitis, bacterial pneumonia, tuberculosis, bronchiectasis

Cough - history

Table 5.1 Cough

Origin	Common causes	Nature/Characteristics
Pharynx	Post-nasal drip	Usually persistent
Larynx	Laryngitis, tumour	Harsh, barking, painful, persistent, associated with stridor (tumours)
	Whooping cough, croup	
Trachea	Tracheitis	Painful
Bronchi	Bronchitis (acute and chronic)	Dry or productive. Worse in mornings
	Asthma	Dry or productive. Worse at night
	Bronchial carcinoma	Persistent (often with haemoptysis)
	Pneumonia	Dry initially, productive later
	Bronchiectasis	Productive. Changes in posture induce sputum production
	Pulmonary oedema	Often at night (may be productive of pink frothy sputum)
	End-stage interstitial fibrosis	Dry, irritant and distressing

CAUSES OF HAEMOPTYSIS

Bronchial disease

- Carcinoma
- Bronchiectasis
- Acute bronchitis

Bronchial adenoma
Foreign body

Parenchymal disease

- Tuberculosis
- Suppurative pneumonia

Lung abscess
Parasites (e.g. hydatid disease, flukes)
Trauma
Actinomycosis
Aspergilloma

Lung vascular disease

- Pulmonary embolus and infarction

Polyarteritis nodosa
Goodpasture's syndrome
AV malformation
Idiopathic pulmonary haemosiderosis

Cardiovascular disease

- Acute left ventricular failure
- Mitral stenosis

Aortic aneurysm

Blood disorders

Leukaemia
Haemophilia
Anticoagulants

• = more common causes

1. **PHYSICAL EXAMINATION** INSPECTION

facial expression

patient´s posture

configuration of the chest: pectus excavatum (funnel chest)
pectus carinatum (pigeon chest)
barrel chest
kyphosis,kyphoscoliosis

assessment of respiratory rate and pattern:
bradypnea,tachypnea,apnea,hyperpnea
KUSSMAUL´S breathing
BIOT´s breathing
CHEYNE-STOKES b.

2. PALPATION OF THE CHEST -tactile fremitus (fremitus pectoralis)

3. PERCUSSION resonant
tympanic
dull
flat
hyperresonant

resonance
dullness
hyperresonance

4. AUSCULTATION Tracheal
bronchial (tubular)
bronchovesicular
vesicular (alveolar)

breathing (breath sounds)

Added breath sounds (abnormal):
crackles (coarse or large and minute),early,late
wheezes (rhonchi)
pleural rubs

/location,timing,intensity/

Increased breath sounds (crackles)
bronchophony
whispered pectoriloquy
egophony

PHYSICAL EXAMINATION

of the anterior and posterior aspects of the chest includes the following: inspection,palpation,percussion and auscultation.

The examination of the posterior chest is performed while the patient is still seated. The patient´s arms should be folded in his or her lap. After the completion of the examination of the posterior chest the patient is asked to lie down and we continue with the examination of the anterior chest.

Inspection- we inspect the patient´s **facial expression**,if cyanosis is present,audible signs of breathing such a stridor or wheezing, **patient´s posture**:patient with ortopnea and asthma cardiale remains seated or lies on several pillows,in airway obstruction supports his arms and fixes the muscles of the shoulder to aid respiration. Inspect the **neck**-if accessory muscles(trapezius and sternocleidomastoid) contract during inspiration= earliest signs of airway obstruction.

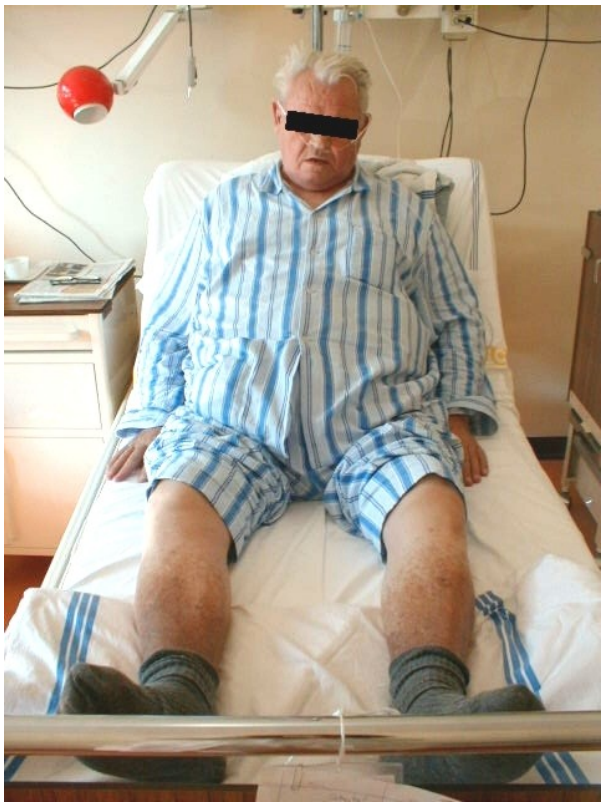


Table 4.19 Types of cyanosis

1.	Peripheral	Hands and feet cyanosed, cold, poor circulation, pulses weak or impalpable, arterial PaO_2 normal
	Causes	Low cardiac output Peripheral vasoconstriction (Raynaud's phenomenon, ergot poisoning)
2.	Central cyanosis	Central mucous membranes cyanosed, arterial PaO_2 reduced
	Causes	a. Impaired pulmonary gas exchange (respiratory disease, pulmonary oedema) Giving oxygen improves saturation b. Right-to-left shunting i. Intracardiac ii. Intrapulmonary Giving oxygen has little effect
3.	Methaemoglobinaemia/ sulphaemoglobinaemia	From nitrites, sulphites, etc. Greyish pigmentation, PaO_2 normal, oxygen saturation reduced

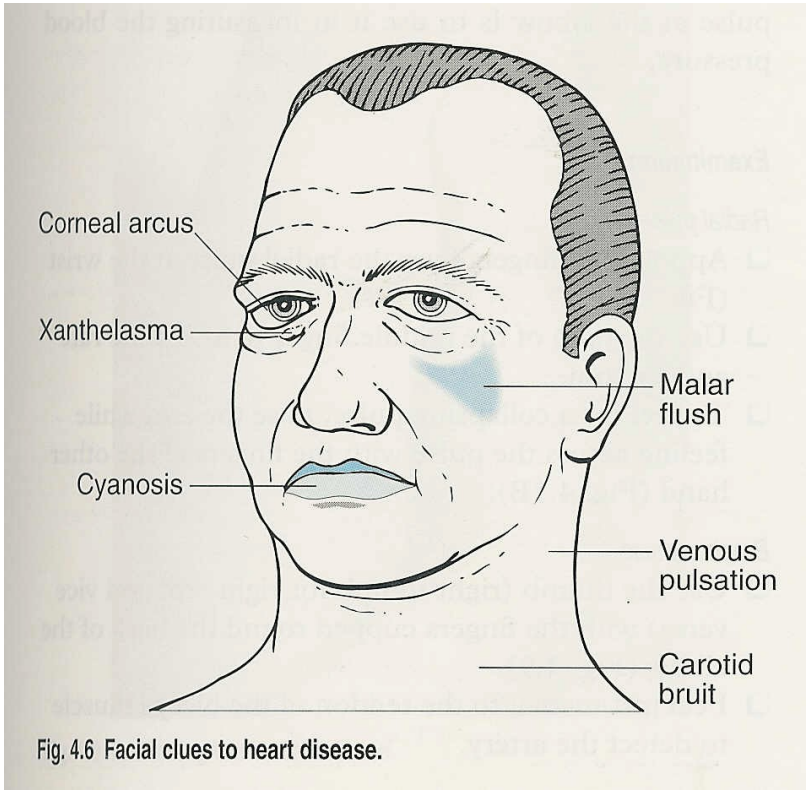
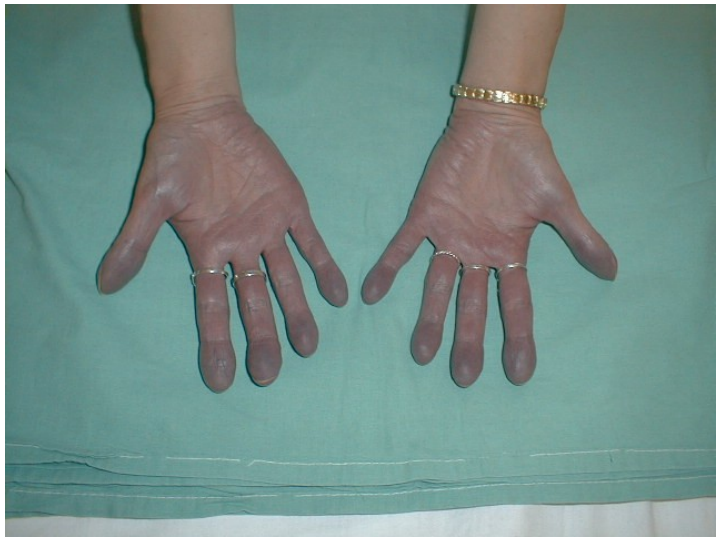
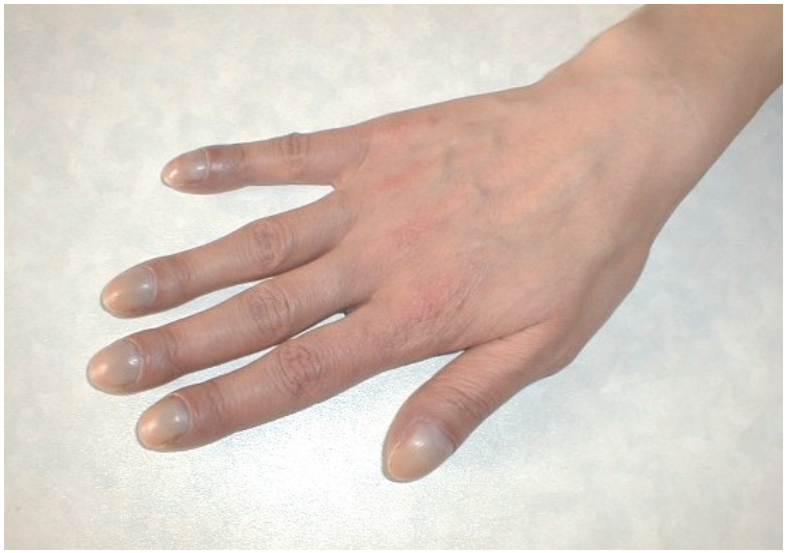


Fig. 4.6 Facial clues to heart disease.





Observation

A normal thorax is symmetrical, widening evenly with breathing.

It is necessary to notice possible breathlessness, cyanosis, or painful breathing before starting the physical examination.

Shape variations of the thorax:

Pyknic - the front-back diameter is longer, ribs stand horizontally.
Asthenic - is long and flat.
Funnel shaped - the inferior part of the sternum is intracted.
Barrel-shaped - is short, fixed in the inspirational state, with a longer front-to-back diameter, occurs in emphysema or chronic bronchopulmonary obstructive disease.
Kyphoscoliotic - is asymmetrical, with gibbus, dextro- or sinistroscoliosis, occurs in rickets during childhood.
Avian (bird-like) - is characterised by swollen cartilaginous ends of the ribs after rickets.
Retraction of hemithorax - may occur in atelectasis, because of adhesions, or after thoracoplasty.
Thoracal arching - occurs with a massive pneumothorax or pleural exudate.

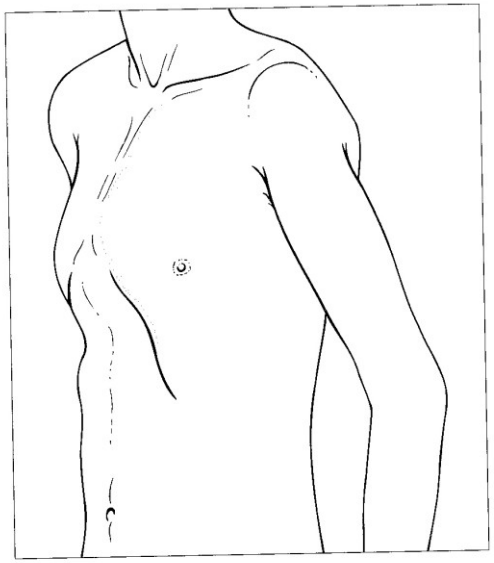


Fig. 5.8 **Pigeon chest deformity** (Pectus carinatum).

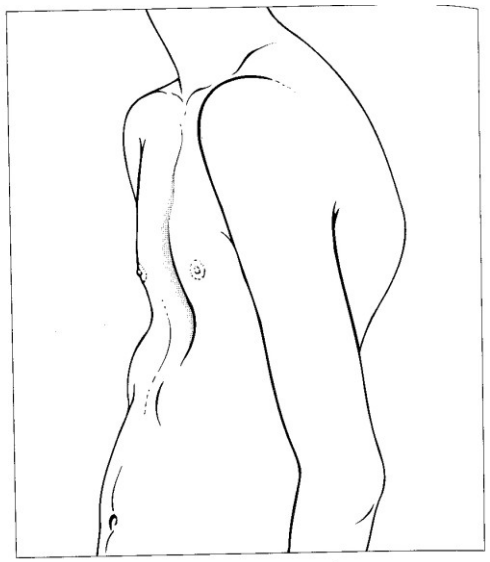


Fig. 5.9 **Funnel chest deformity** (Pectus excavatum).

During examination, it is possible to notice kyphosis or kyphoscoliosis.

Post-operative scars after thoracotomy
(lung and heart surgery) should also be concerned.

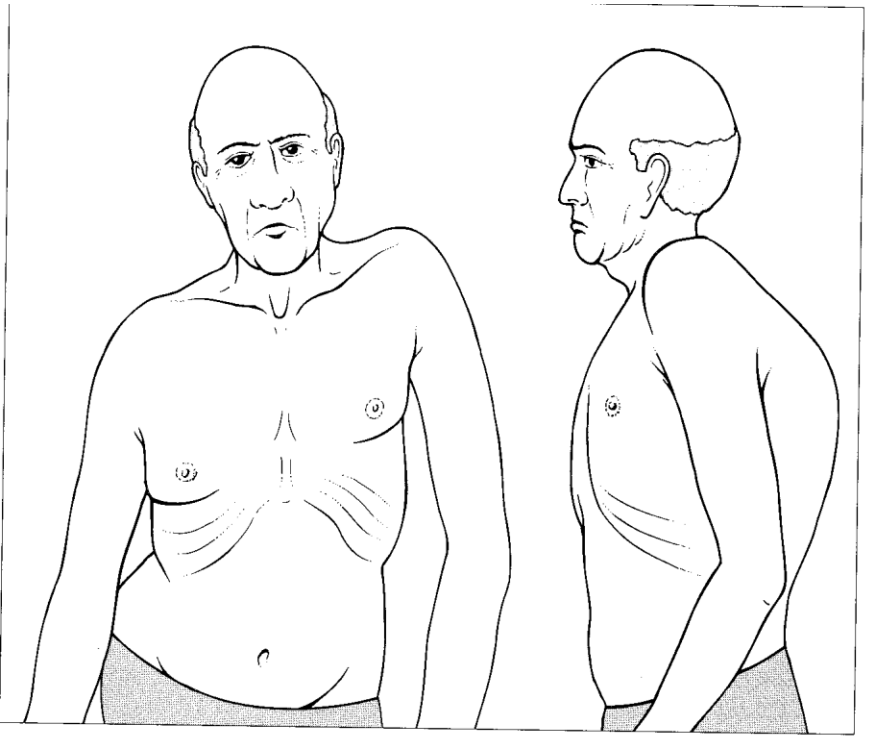


Fig. 5.7 Kyphoscoliosis.

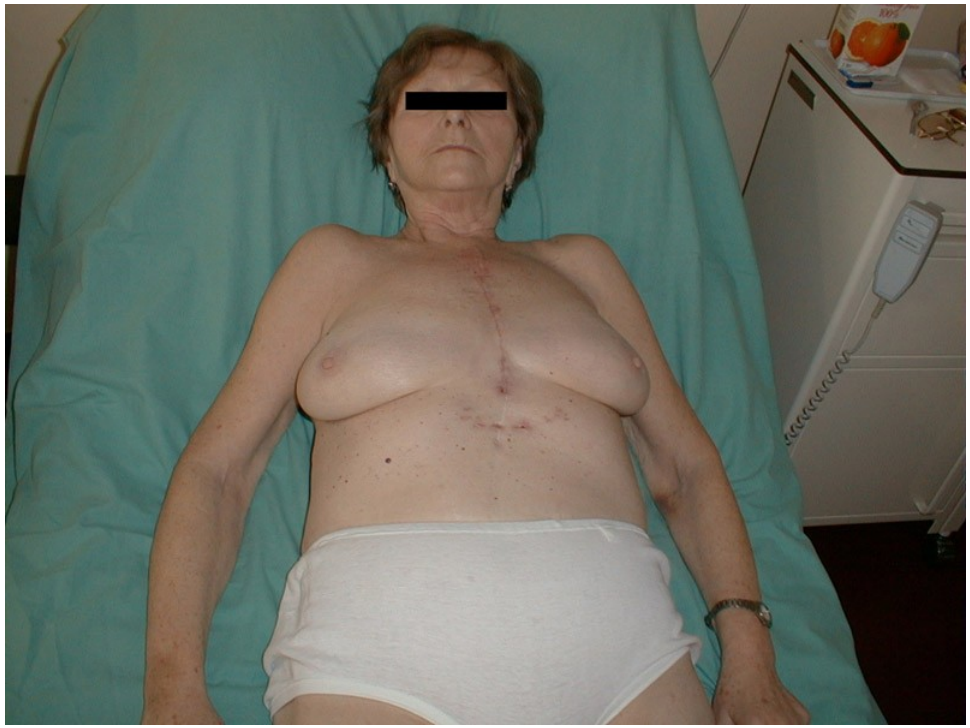


Table 5.9 Lesions of the chest wall

Cutaneous lesions	Skin eruptions, sarcoid nodules, neurofibromas, purpuric spots (Fig. 3.10), bruises, scars, discharging sinuses
Subcutaneous lesions	Inflammatory swellings, metastatic tumour nodules, sebaceous cysts, sarcoid nodules, neurofibromas (Fig. 3.34), lipomas
Subcutaneous emphysema	
Vascular anomalies	Spider naevi, enlarged vascular channels (arterial in coarctation of the aorta; venous in superior vena caval obstruction)
Localised prominences and deformities	Clavicles, scapulae, sternum, ribs, costochondral junctions, spinous processes
Localised tenderness	Fractured rib, tumour involving chest wall, spinal nerve root disorders
Lesions of breast (p. 80)	
Enlargement of axillary lymph nodes (p. 74)	

Palpation

Of the thoracic wall is used mainly for detection:

of thoracic vibrations,
a pleural frictional murmur,
the quality of thoracic wall,
tenderness,
and resistance.

Thoracic vibrations (fremitus pectoralis) are normally symmetrical on both halves of the thorax.

Strengthened vibrations - are found over an infiltrated lung tissue (pneumonia, bronchopneumonia - better conductivity of the tissue).

Weakened or missing vibrations - occur with fluidothorax or pneumothorax (isolating layer decreases conduction of the vibrations) and emphysema.

The technique of percussion: we use the middle finger of the left hand placed firmly against the chest wall parallel to the ribs in an interspace, other fingers and the palm are held off the chest. The tip of the right middle finger strikes a quick sharp blow to the terminal phalanx of the left finger on the chest wall. The motion of the striking finger should come from the wrist and not from the elbow.

The sites on the posterior chest for percussion are above, between and below the scapulae in the intercostal spaces; we start at the top and work downward, proceeding from side to side, comparing one side with the other.

We perform percussion at many lines: midclavicular, anterior-, midaxillary, posterior axillary, scapular and parasternal lines.

We also can evaluate the **diaphragmatic movement:** the patient is asked to take a deep breath and hold it. Percussion at the lung base determines the lowest area of resonance which represents the lowest level of the diaphragm. Below this level is dullness.

Then the patient is instructed to exhale as much as possible and the percussion is repeated. The level of dullness moves upward and the difference between the inspiration and expiration represents diaphragmatic motion, which is normally, for instance, over liver 4-5 cm. In patients with emphysema the motion is reduced.

Try the percussion on yourself: percuss over your right lung (resonant note), over the liver dull, over stomach tympanic, over your thigh flat note.

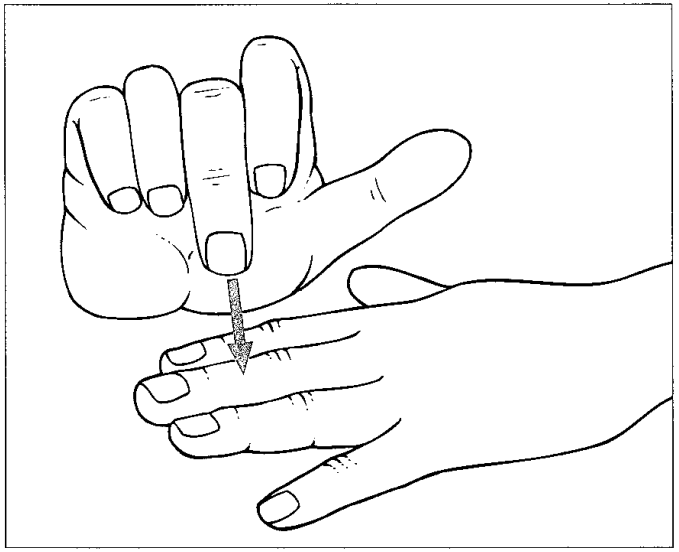


Fig. 5.14 Technique of percussion.

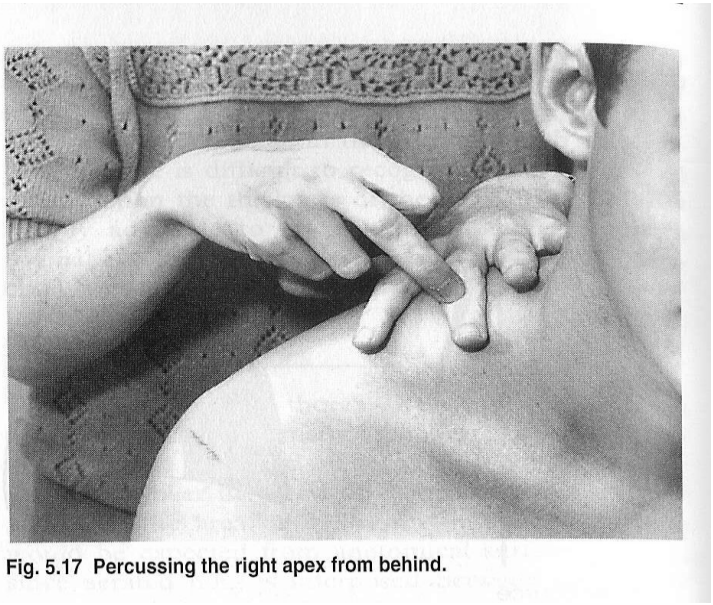


Fig. 5.17 Percussing the right apex from behind.

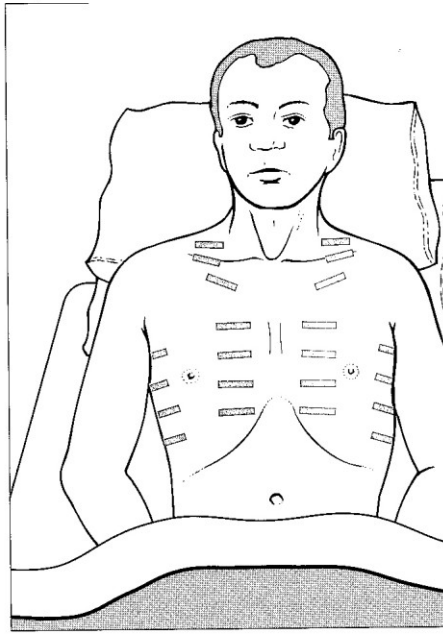


Fig. 5.15 Sites for percussion – anterior and lateral chest w

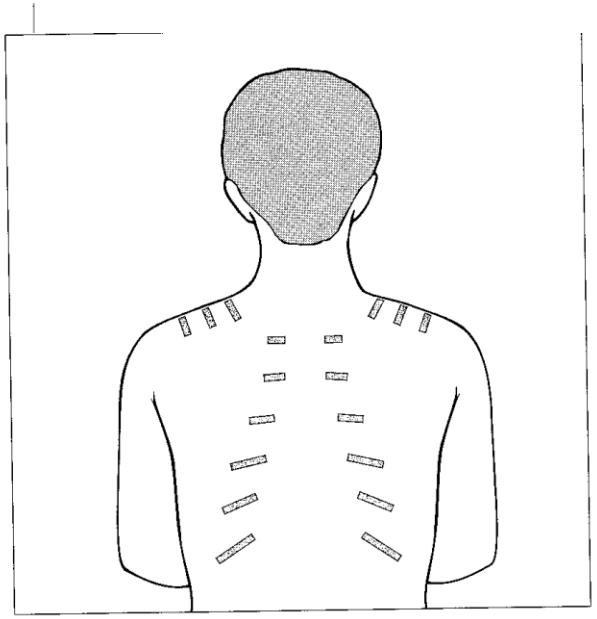


Fig. 5.16 Sites for percussion – posterior chest wall.

Percussion

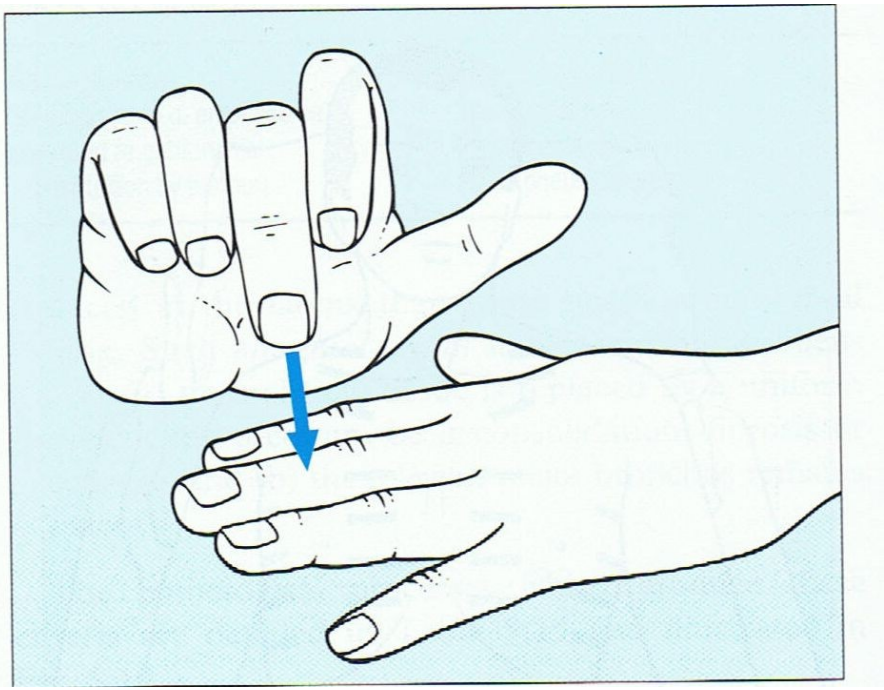
In healthy person it is full, bright, even on both halves of the thorax.

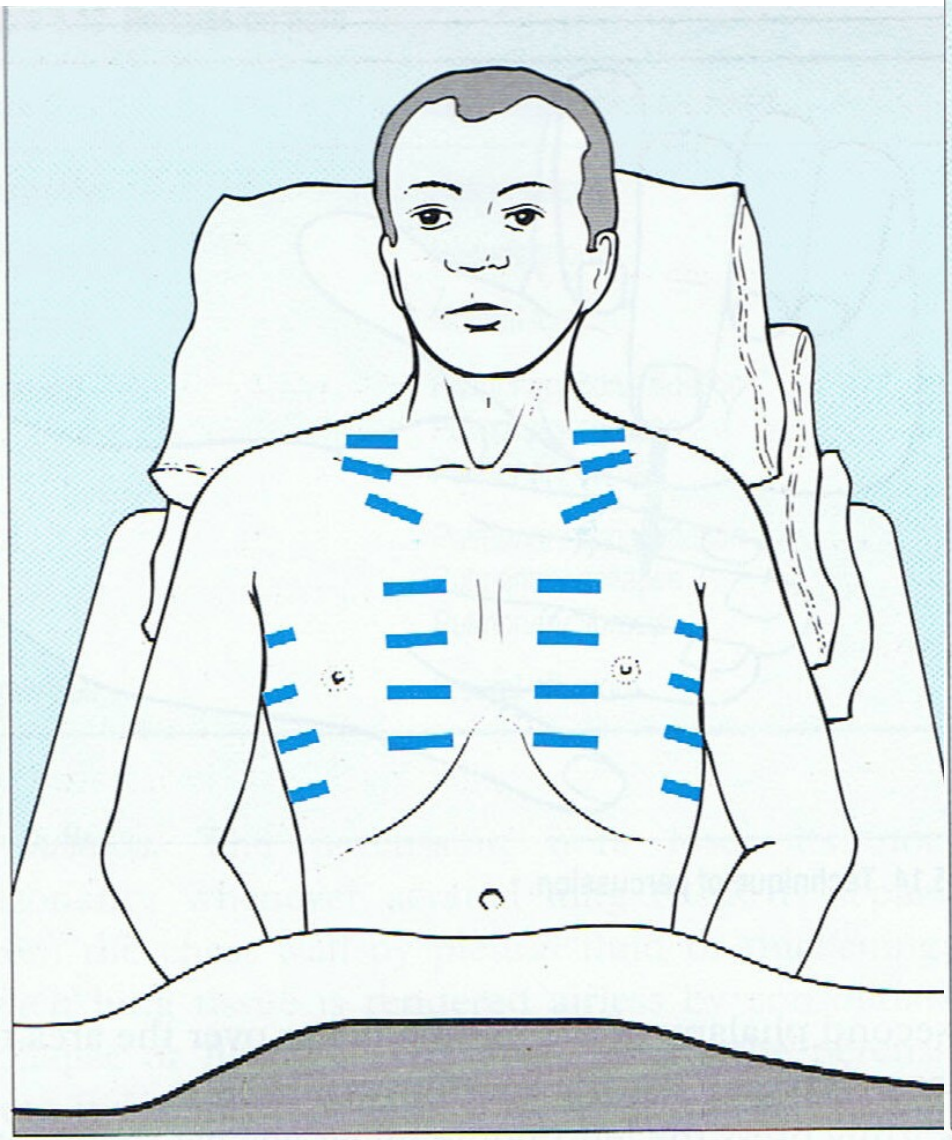
Shortened - appears with a loss of air supply (pneumonia, atelectasis), with fluidothorax (the shape is parabolic with the top located in axilla).

Hypersonic - is connected with excessive air supply (emphysema) or with pneumothorax, when it can be also tympanic

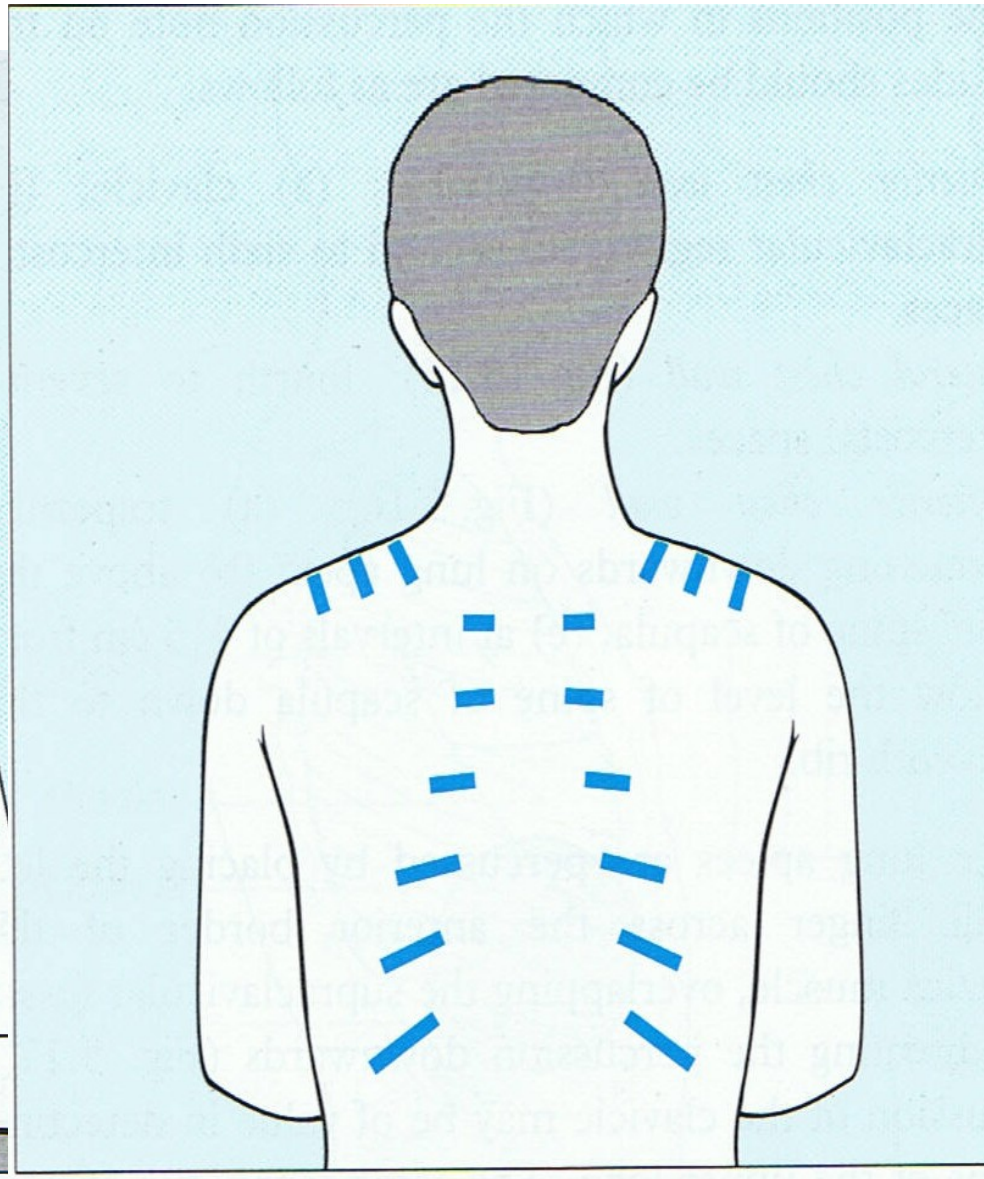
Table 5.10 Percussion note

Type	Lesions by which produced
Tympanitic	Hollow viscus
Hyperresonant	Pneumothorax
Resonant	Normal lung
Impaired	Pulmonary consolidation Pulmonary collapse Pulmonary fibrosis
Dull	Pulmonary consolidation Pulmonary collapse Pulmonary fibrosis
Stony dull	Pleural effusion





g. 5.15 Sites for percussion – anterior and lateral chest wall.



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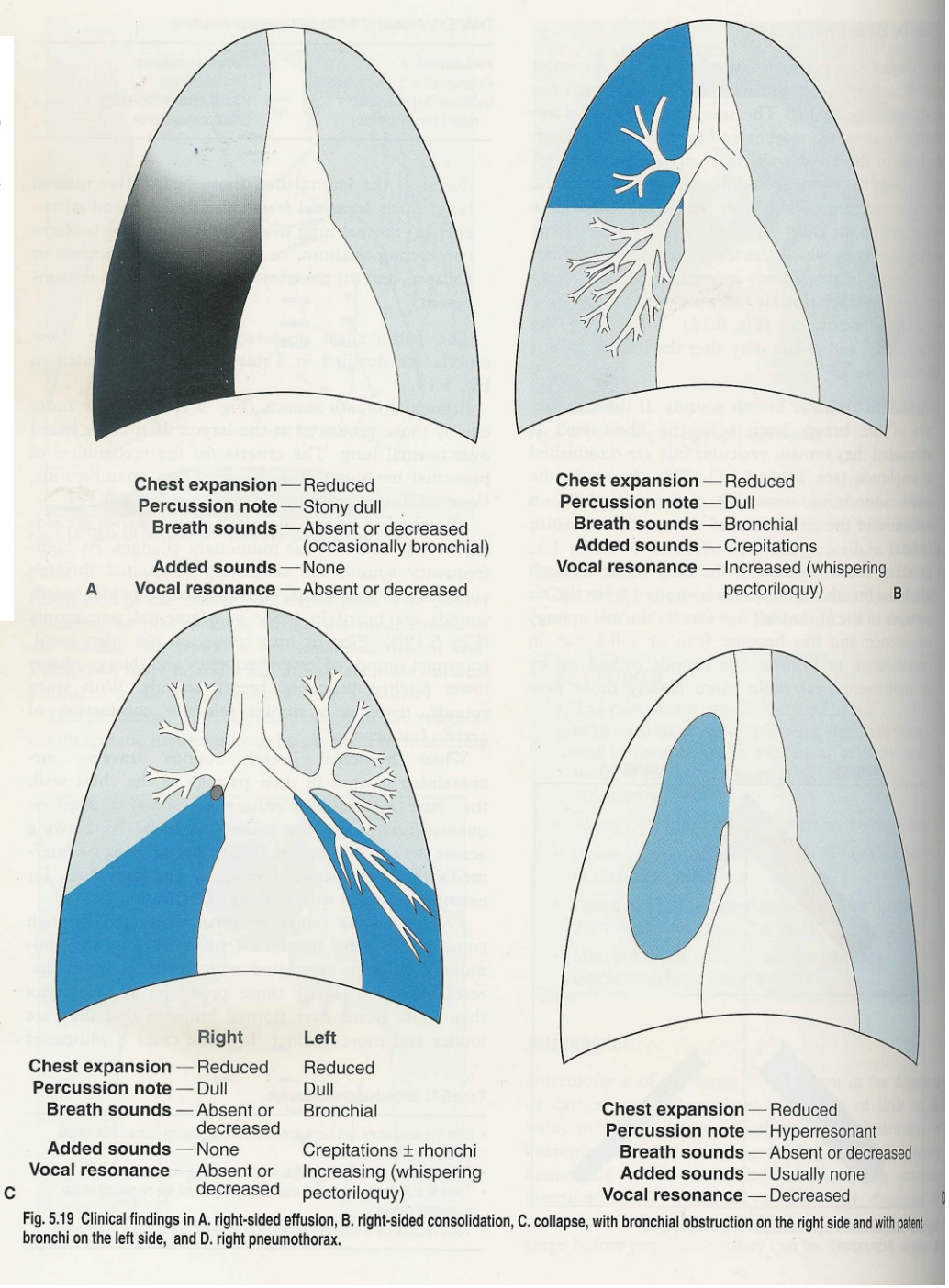


Fig. 5.19 Clinical findings in A. right-sided effusion, B. right-sided consolidation, C. collapse, with bronchial obstruction on the right side and with patent bronchi on the left side, and D. right pneumothorax.

Auscultation

Under physiological circumstances, clear alveolar breathing is present over the lungs, without any side phenomena. Tubal or tubular breathing can only be heard over the upper sternum and between the scapulae.

Alveolar breathing :

Sharpened **<u>diffusely</u> - during stronger breathing, e.g. acidotic breathing (decompensated diabetes mellitus, uraemia).**

<u>unilaterally</u> - compensatory manifestation (broad infiltration, compressio by fluidothorax).

Weakened - present with emphysema, atelectasis, pleural exudate, and pneumothorax.

With prolonged expiration - occurs with obstruction of the airways

over your thigh flat note.

AUSCULTATION is a technique of listening for sounds produced in the lung during inspiration and expiration. The stethoscope usually has two heads: the bell and the diaphragm. The bell is used to detect low pitched sounds whereas the diaphragm is better at detecting higher pitched sounds. There are 4 types of normal breath sounds: tracheal, bronchial, bronchovesicular and vesicular(=alveolar).

Tracheal breath sounds are very loud, harsh, high-pitched, located over trachea. **Bronchial(tubular)** sounds are loud, high-pitched and sound like air rushing through a tube. Expiratory component is louder and longer. Normally heard are over the manubrium. When they are heard in periphery, we must suppose a consolidated lung parenchyma as in pneumonia or atelectasis. **Bronchovesicular** breath sound are a mixture of bronchial and vesicular sounds. They are normally heard only in the first two interspaces anteriorly and betweeen the scapulae posteriorly, i.e. over the mainstem bronchi.

Vesicular (alveolar) sounds are soft, low-pitched sounds heard over most of the lung fields. It is a gentle rustling where the inspiratory component is much longer than the expiratory component.

In addition to the normal breath sounds other lung sounds may be produced in abnormal clinical states: crackles, wheezes, pleural rubs (added sounds).

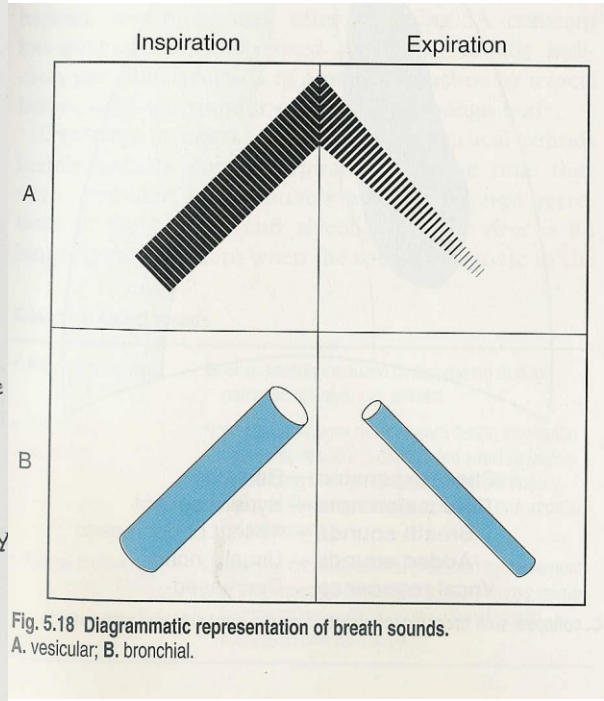
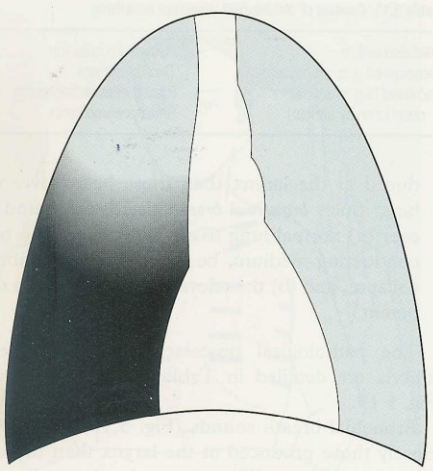


Fig. 5.18 Diagrammatic representation of breath sounds.
A. vesicular; B. bronchial.

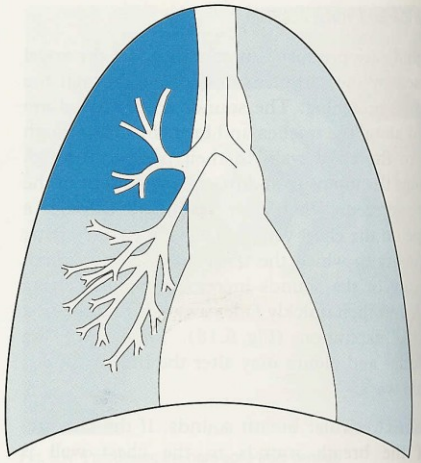
Table 5.15 Bronchial, diminished or absent breath sounds

Auscultatory findings	Disease process
High-pitched bronchial breath sounds	Pneumonic consolidation (Fig. 5.19B) Large superficial pulmonary cavity Collapsed lung or lobe when large bronchi are patent (Fig. 5.19C) Lung compressed by pleural effusion (sometimes) Tension pneumothorax (sometimes)
Low-pitched bronchial breath sounds	Localised areas of pulmonary fibrosis e.g. chronic pulmonary tuberculosis, chronic suppurative pneumonia
Diminished or absent breath sounds	Pleural effusion (Fig. 5.19A) Marked pleural thickening Collapsed lung or lobe when large bronchi occluded (Fig. 5.19C) Pneumothorax (Fig. 5.19D) Emphysema (symmetrical diminution over both lungs)



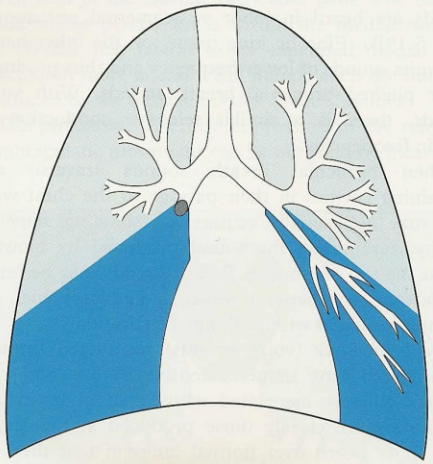
Chest expansion — Reduced
Percussion note — Stony dull
Breath sounds — Absent or decreased (occasionally bronchial)
Added sounds — None
Vocal resonance — Absent or decreased

A



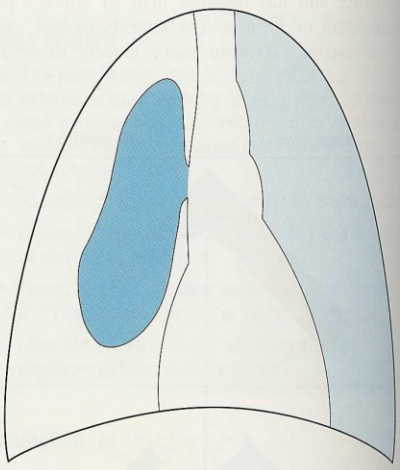
Chest expansion — Reduced
Percussion note — Dull
Breath sounds — Bronchial
Added sounds — Crepitations
Vocal resonance — Increased (whispering pectoriloquy)

B



	Right	Left
Chest expansion	Reduced	Reduced
Percussion note	Dull	Dull
Breath sounds	Absent or decreased	Bronchial
Added sounds	None	Crepitations ± rhonchi
Vocal resonance	Absent or decreased	Increasing (whispering pectoriloquy)

C



Chest expansion — Reduced
Percussion note — Hyperresonant
Breath sounds — Absent or decreased
Added sounds — Usually none
Vocal resonance — Decreased

D

Fig. 5.19 Clinical findings in A. right-sided effusion, B. right-sided consolidation, C. collapse, with bronchial obstruction on the right side and with patent bronchi on the left side, and D. right pneumothorax.

Added sounds:

Crackles (rales) are short,discontinuous,nonmusical sounds heard mostly during inspiration. They have also been called rales,however since 1980 the correct term is crackles.
They are produced when there is fluid (excess airway secretion) inside a bronchus and there is a collapse of the distal airways and alveoli. A sudden equalization of pressure seems to result in a crackle, coarse or minute.
Minute crackles are likened to the sound made by rubbing hair next to the ear. **Coarser** (larger) crackles are related to larger airways and sound like cracking-bursting-breaking of larger bubbles.

Inspiratory crackles are described as **early** ,**or late**,depending on when they are heard during inspiration.
Early fine crackles are common in cardiac failure and obstructive lung disease, late crackles in interstitial fibrosis and pneumonia.
Otherwise etiology is very very varied: bronchitis,respiratory infections,fibrosis,congestive heart failure,pulmonary edema.

Wheezes,also known as **rhonchi** are continuous musical sounds heard mostly during expiration. They are produced by airflow through narrowed bronchi. This narrowing may be due to spasm,swelling, sekretions,tumor or foreign body. Wheezes are commonly associated with bronchospasm of asthma ,however also with bronchitis, congestive heart failure,pulmonary edema.
They may have whistling or creaky character.

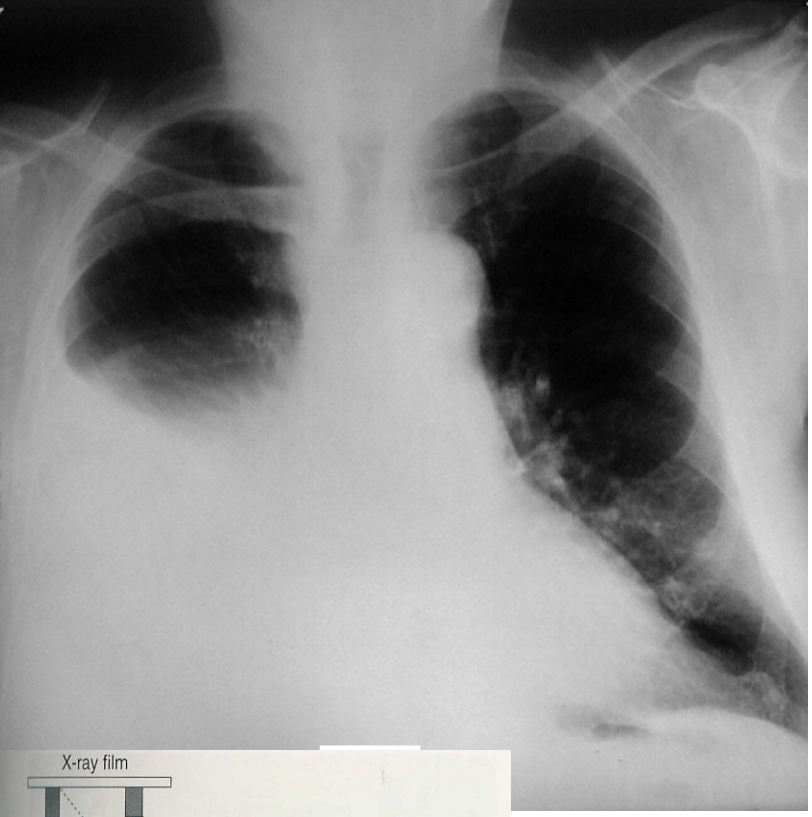
Pleural rub is a grating sound produced by motion of the pleura, which is impeded by frictional resistance; when the sticky fibrinous mass is present,pleural surfaces are roughened or thickened by inflammatory or neoplastic cells,or by fibrinous deposits,they are rubbing against each other. It sounds as you walk on the fresh snow.
Pleural rub is best heard at the end of inspiration and at the beginning of expiration.
All of these added sounds should be described as to their location,timing and intensity.

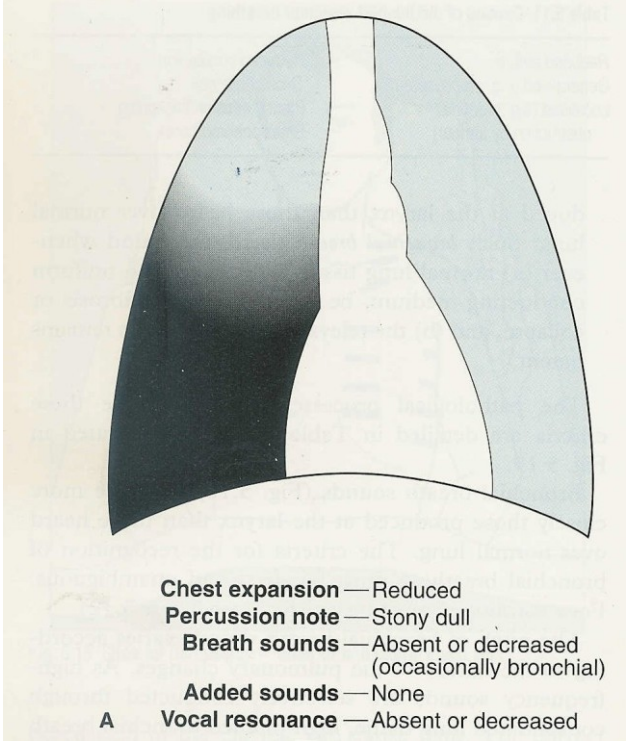
Occasionally breath sounds are transmitted abnormally and this may result in auscultatory changes:
f.i. pleural effusion is characterized by decreased or disappeared breath sounds,percutatory dullness and decreased fremitus and **bronchophony.**
Consolidation(inflammatory etc.) of the lung leads to the increased transmission of spoken words,tactile fremitus and bronchophony and also increased breath sounds(bronchial) and crackles.

We have 3 terms to explain: **bronchophony,whispered pectoriloquy** and **egophony**.

The patient is asked to say ninety-nine while the examiner listens to the chest. If **bronchophony** is present,the words will be transmitted louder in the area of lung consolidation(pneumonia).

Or the patients is instructed to whisper "one-two-three" Nothing is heard in normal chest,if consolidation is present,the transmission of words will be clearly heard. We say that **whispered pectoriloquy** is present.
Or the patient is asked to say eeee If **egophony is** present the eeee will be heard as aaaa. This change of "e" to "a" is again seen in consolidation of lung tissue,f.i. in the area of compressed lung above a pleural effusion.





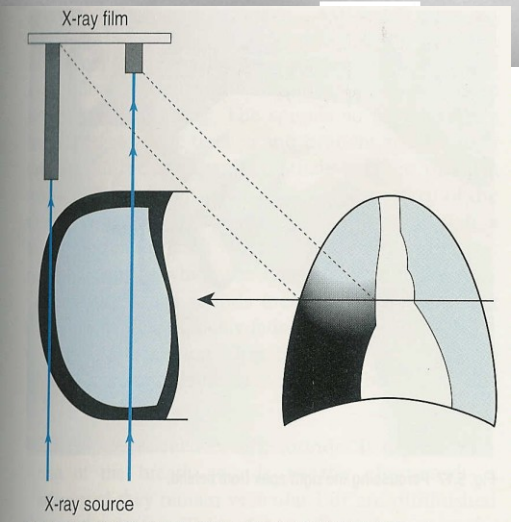


Fig. 5.13 Radiographic appearances of a right pleural effusion. A horizontal section of hemithorax close to the upper margin of the effusion (represented by the horizontal arrow) shows that there is at this site a similar amount of liquid anteriorly, posteriorly and laterally. However, because of the shape of the hemithorax, the x-ray beams traverse more fluid laterally than they do centrally. This produces the characteristic radiographic shape of a pleural effusion shadow with a curved upper margin ascending towards the axilla.

Pleural exudate

Presence of fluid between the two pleural layers.
It can be detected when the volume exceeds 500 ml.

Usually without breathlessness, but it depends on the size of the exudate.
Fremitus pectoralis weakened in the area of exudate.
Percussion shortened or even obscured,
edge in parabolic shape with the top in axilla.
Near the upper edge the percussion is hypersonic or
even tympanal (skodaic resonance).
Bronchophony weakened.

Kinds of exudates according to aetiology:

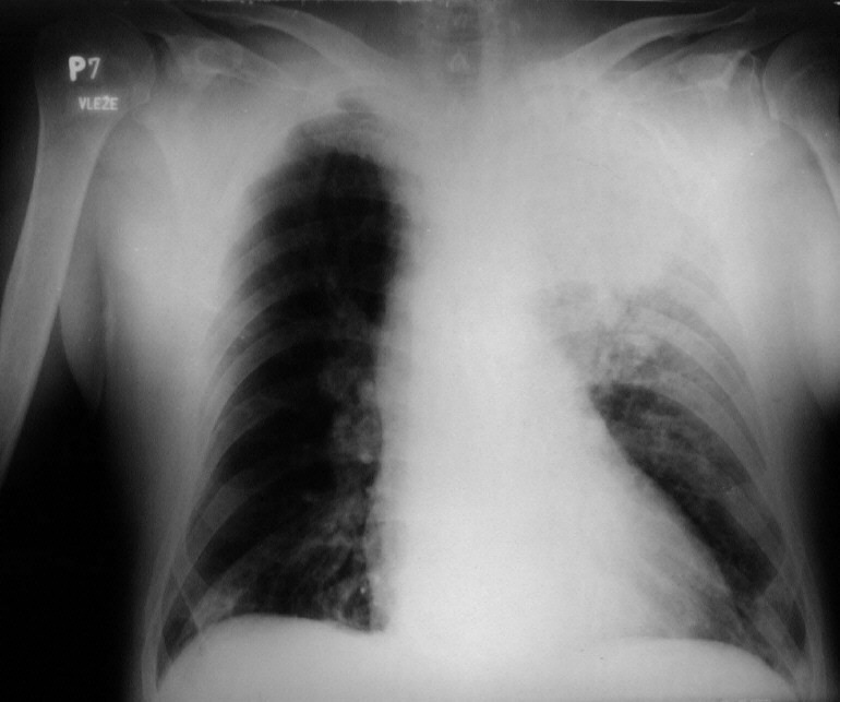
Transudate - low amount of proteins, specific weight up to 1013 g/l, amount of proteins less then 30 g/l, appears most frequently in case of cardiac insufficiency.

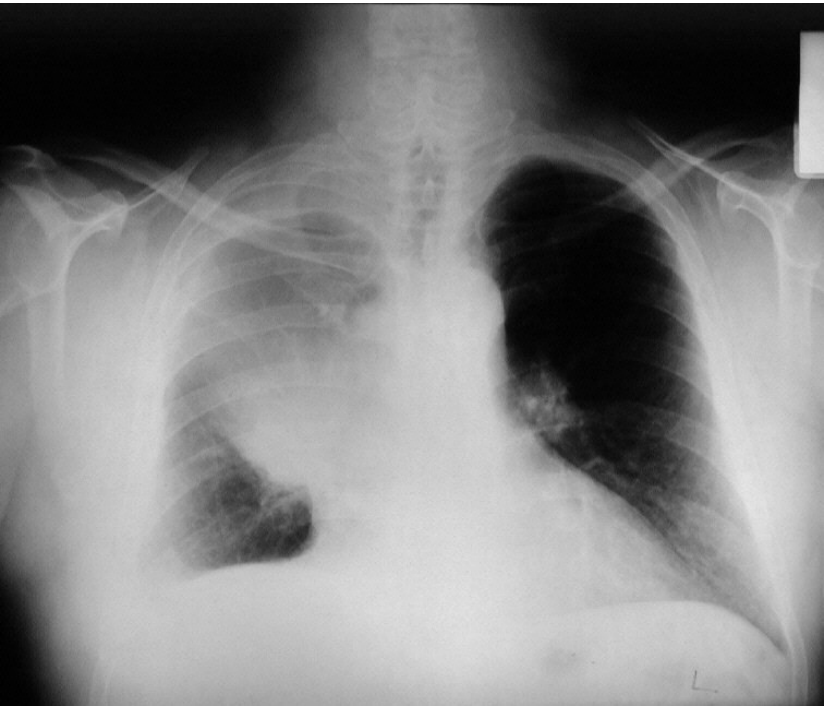
Exudate - high amount of proteins, specific weight higher then 1013 g/l, amount of proteins higher than 30 g/l, occurs in case of TBC, tumours, pleuropneumonia, or collagenoses.

Empyema - means presence of pus in the pleural cavity.

Haemothorax - presence of blood in the pleural cavity (trauma).

Chylothorax - presence of lymph in the pleural cavity in case of a damaged thoracic duct.

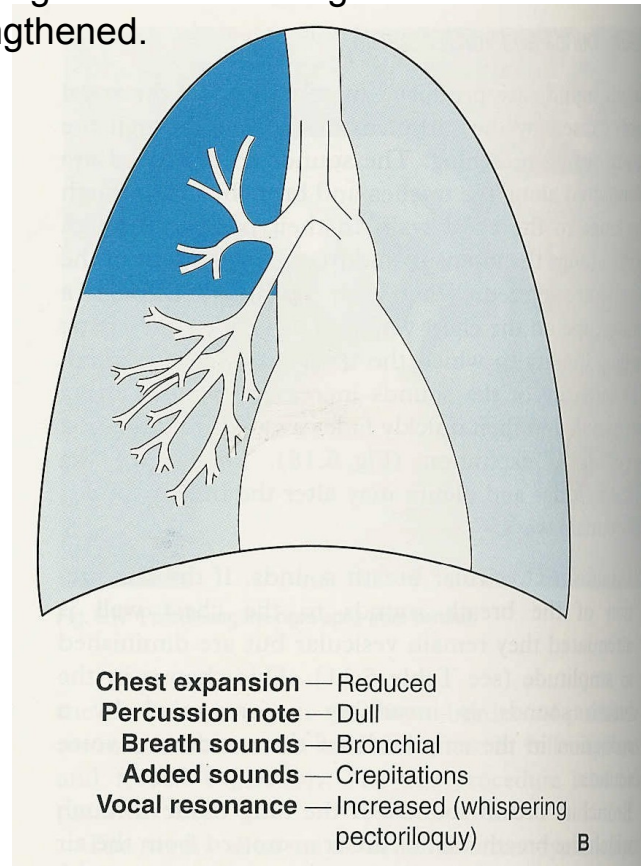




Pneumonia (croupous)

The finding described below is seen only rarely in practice, because of current antibiotic treatment.

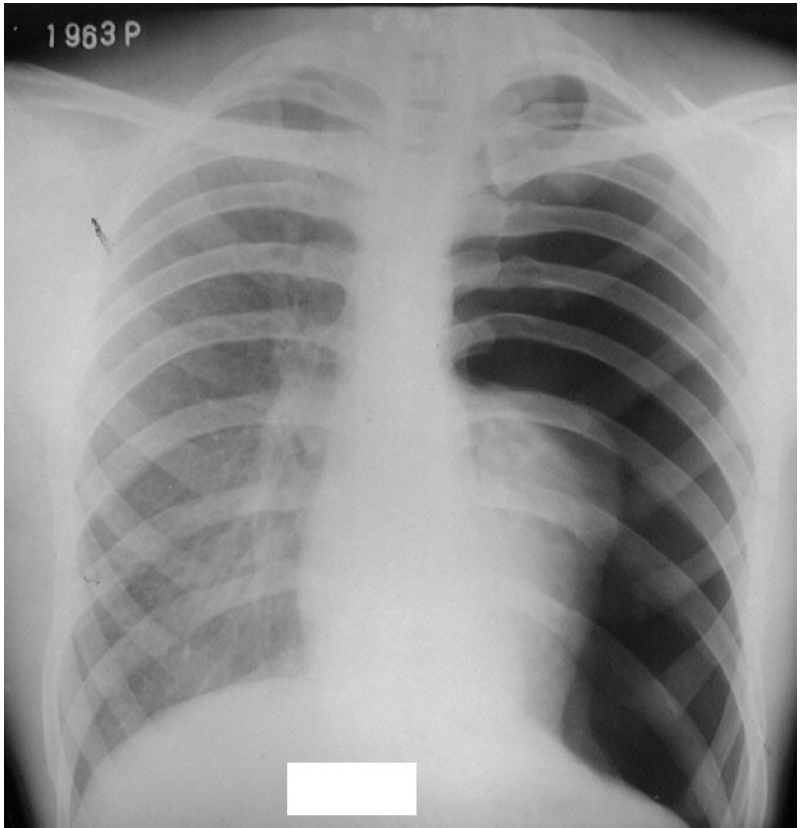
Breathlessness (may be manifested).
Fremitus pectoralis strengthened.
Percussion shortened.
Initially, crepitations are audible (crepitus indux), later tubal breathing (phase of hepatisation), crepitations again (crepitus redux during resorbence), progressive weakening of tubal breathing.
Bronchophony strengthened.



Pneumothorax

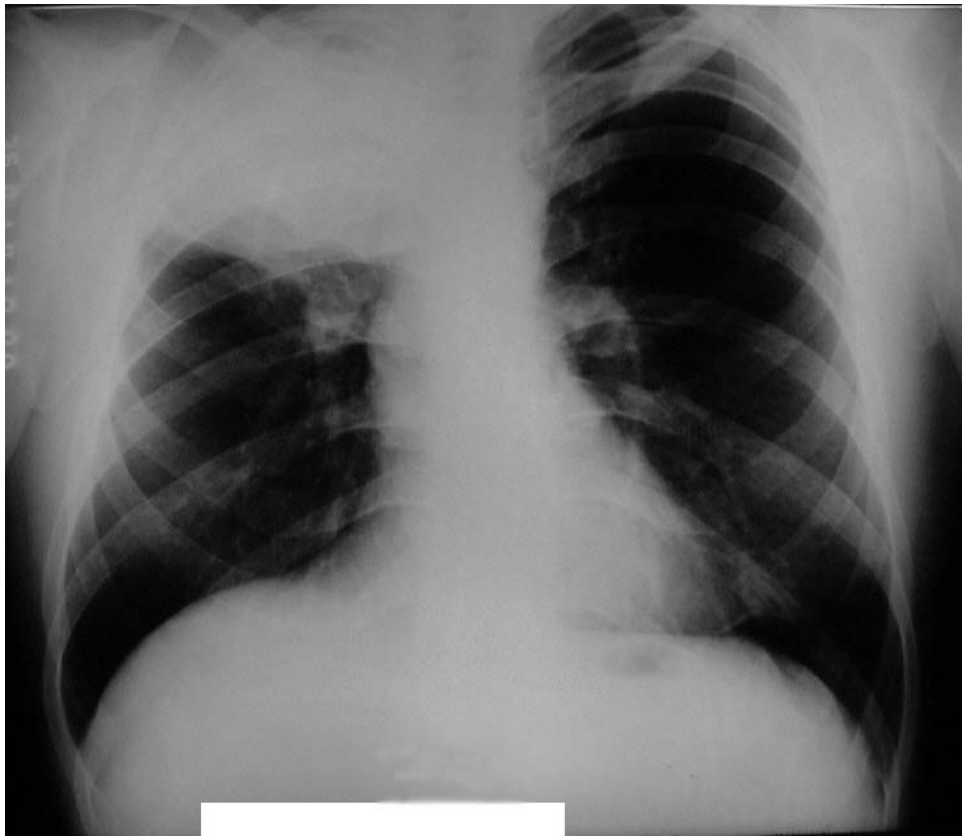
presence of air in the pleural cavity
(trauma, rupture of emphysematous bulla, iatrogenic origin).

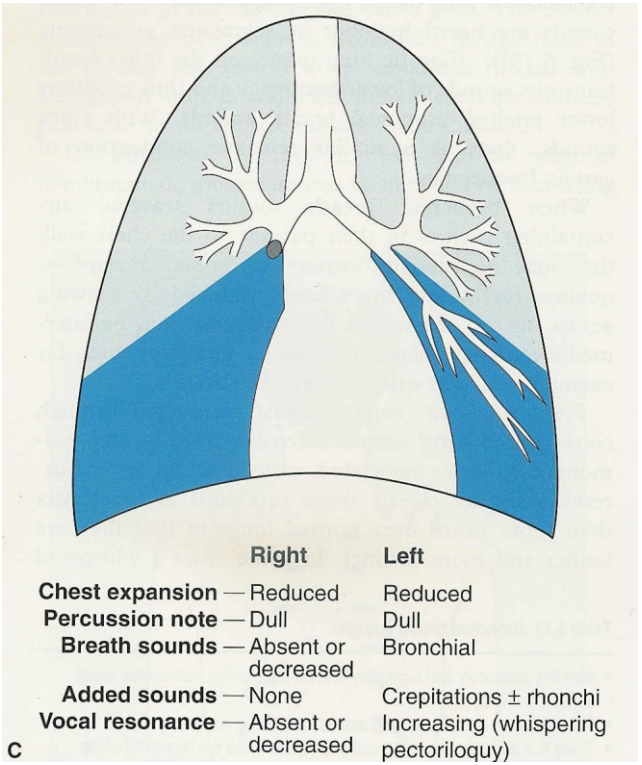
Breathlessness depends on the size and cause of the pneumothorax.
Limited breathing movements of the affected half of thorax.
Fremitus pectoralis weakened or missing.
Percussion hypersonic.
Breathing weakened or missing if the lung is completely collapsed.
Bronchophony weakened.

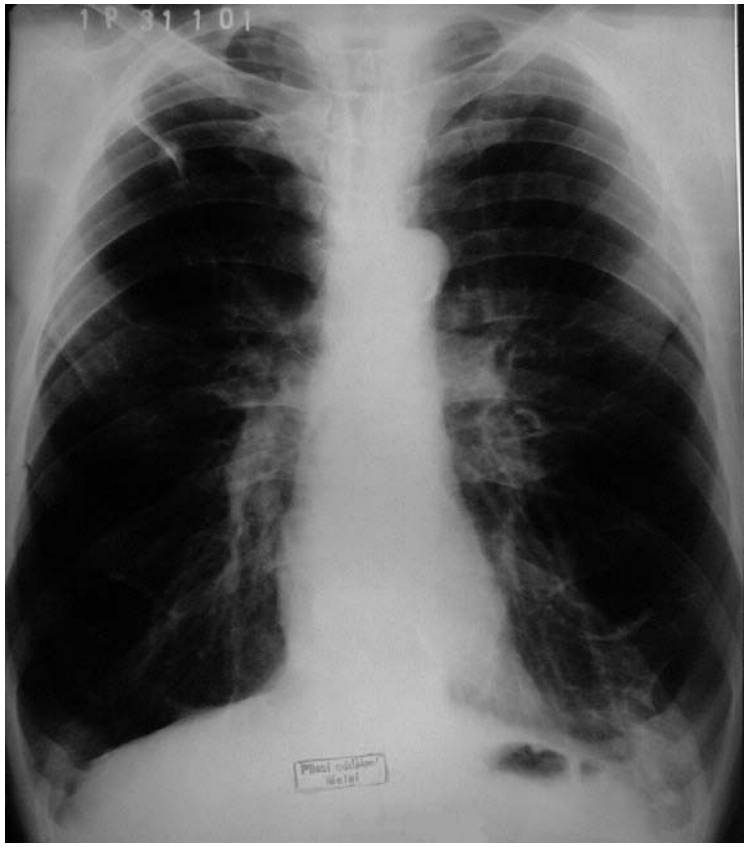


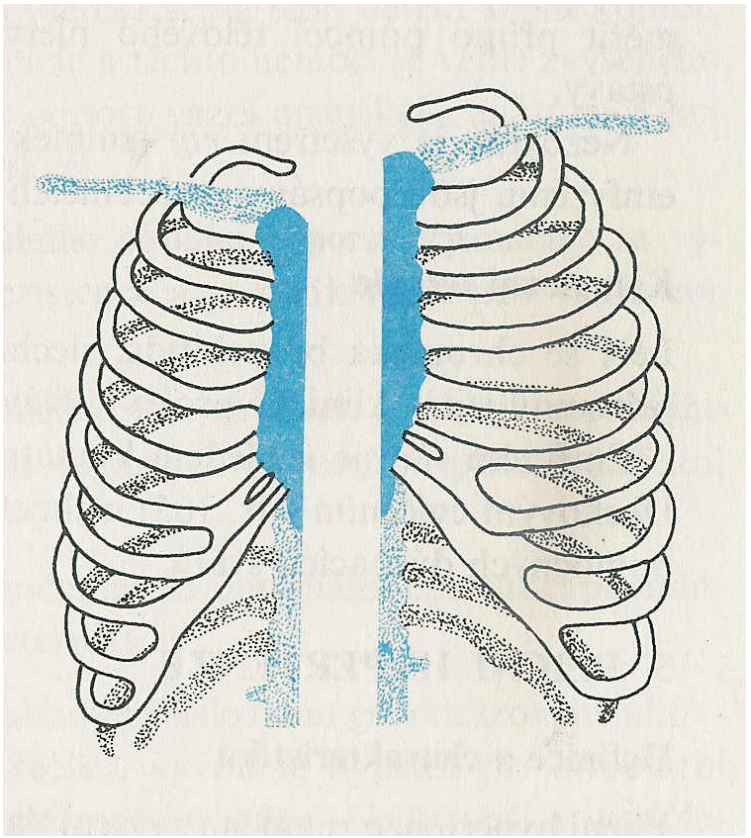
Atelectasis
means loss of air supply to alveoli, bronchi, bronchioles, or the whole lung. The size of affection depends on its cause.

In case of larger atelectasis there is breathlessness and cyanosis present.
Fremitus pectoralis weakened.
Percussion shortened.
Breathing shortened.
Bronchophony weakened.



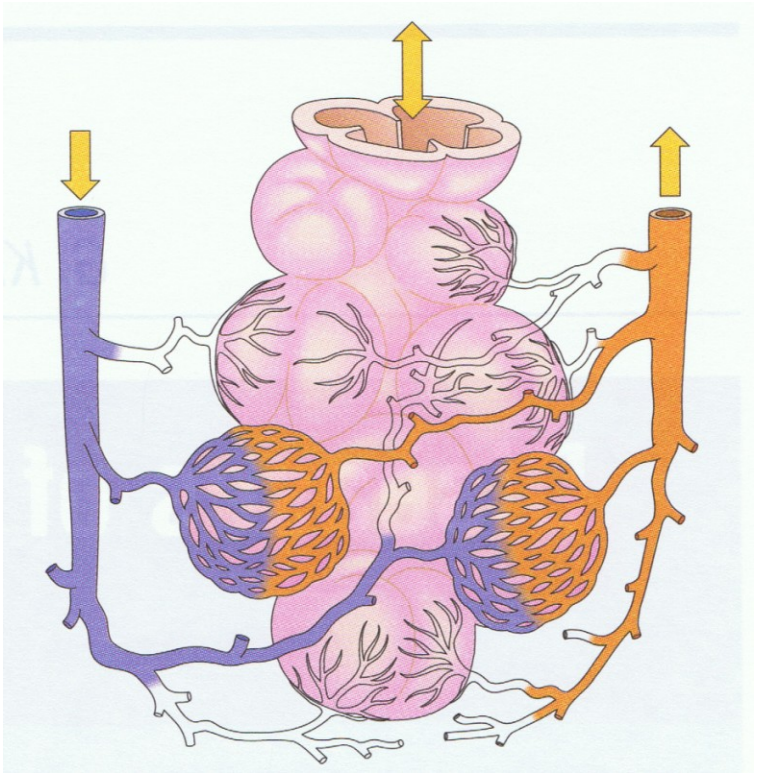


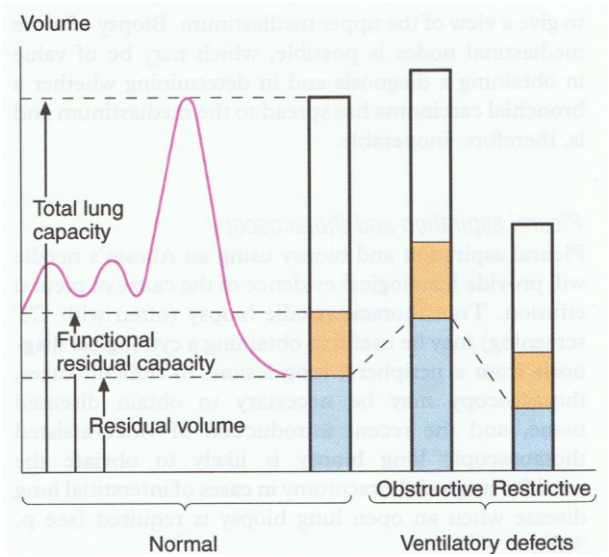




Emphysema
Barrel-shaped thorax.
Fremitus pectoralis weakened.
Percussion hypersonic.
Breathing alveolar, weakened.
If there is a chronic bronchitis present, the dry or wet phenomena are often heard.
Bronchophony weakened.

volume





Gas exchange

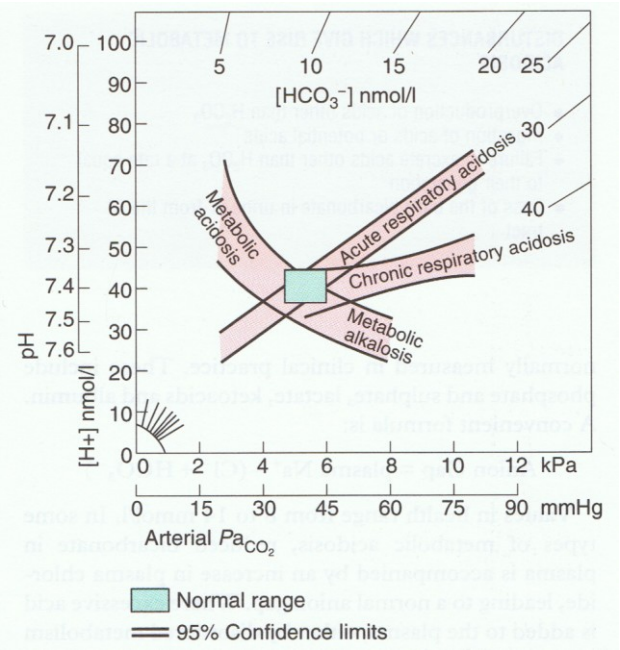


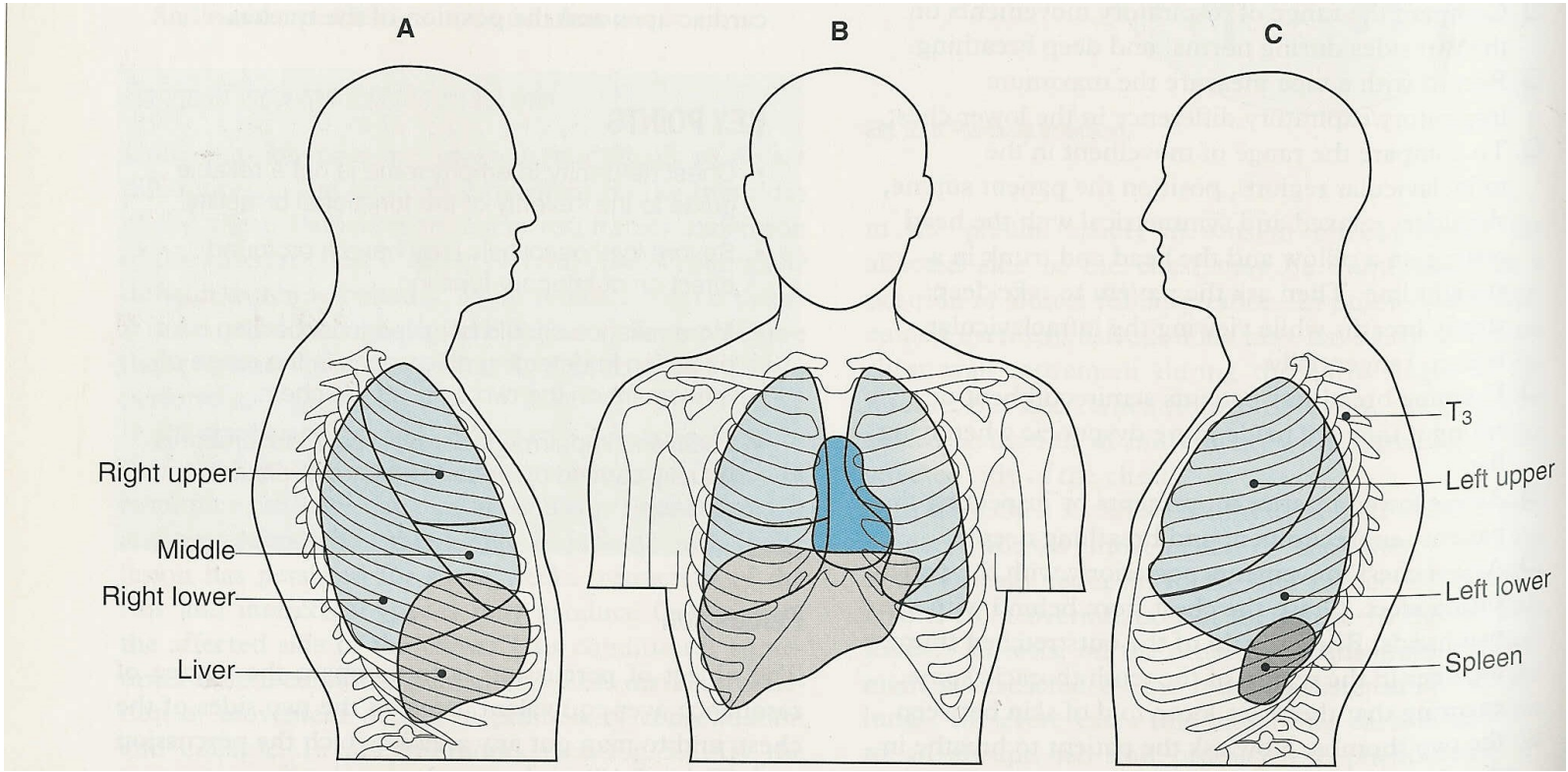
Table 6.7 Respiratory failure

	Type I ($PaO_2 < 8.0$ kPa) ($PaCO_2 < 6.6$ kPa)		Type II ($PaO_2 < 8.0$ kPa) ($PaCO_2 > 6.6$ kPa)	
	Acute	**Chronic**	**Acute**	**Chronic**
Typical blood gases	$PaO_2 \downarrow\downarrow$ $PaCO_2 \leftrightarrow$ or $\downarrow$ pH $\leftrightarrow$ or $\Downarrow$ $HCO_3 \leftrightarrow$	$PaO_2 \downarrow$ $PaCO_2 \leftrightarrow$ pH $\leftrightarrow$ $HCO_3 \leftrightarrow$	$PaO_2 \downarrow$ $PaCO_2 \uparrow$ pH $\downarrow$ $HCO_3 \leftrightarrow$	$PaO_2 \downarrow$ $PaCO_2 \uparrow$ pH $\downarrow$ or $\leftrightarrow$ $HCO_3 \uparrow$
Causes	Asthma	Emphysema	Severe acute asthma	Chronic bronchitis
	Pulmonary embolus	Lung fibrosis	Acute epiglottis	Primary alveolar hypoventilation
	Pulmonary oedema	Lymphangitis carcinomatosa	Inhaled foreign body	Kyphoscoliosis
	Adult respiratory distress syndrome	L→R shunts	Respiratory muscle paralysis	Ankylosing spondylitis
	Pneumothorax	Anaemia	Flail chest injury	
	Pneumonia		Sleep apnoea	
			Brain stem lesion	
			Narcotic drugs	
Therapy	Treat underlying cause	Treat underlying disorder	Treat underlying disorder	Treat underlying disorder
	High concentration O_2	Long-term O_2	Controlled low concentration O_2	Controlled long-term O_2 delivery
	Mechanical ventilation if necessary		Mechanical ventilation or tracheostomy if necessary	Mechanical ventilatory support if necessary

Table 6.6 Differential diagnosis of acute severe dyspnoea

Condition	History	Signs	Chest radiography	Arterial blood gases	ECG	Other tests
Left ventricular failure	Chest pain, orthopnoea, palpitations, *a previous cardiac history	Central cyanosis, JVP (→ or ↑), *sweating, cool extremities, *dullness and crepitations at bases	Cardiomegaly, *Upper zone vessel enlargement, *overt oedema/pleural effusions	↓PaO_2 ↓$PaCO_2$	Sinus tachycardia, signs of myocardial infarction, arrhythmia	Echo-cardiography (↓left ventricular function)
Massive pulmonary embolus	Recent surgery or other risk factors. Chest pain, previous pleurisy, *syncope, *dizziness	Severe central cyanosis, *elevated JVP, *absence of signs in the lung (unless previous pulmonary infarction), shock (tachycardia, reduced blood pressure)	May be subtle changes only, prominent hilar vessels, *oligaemic lung fields	⇓PaO_2 ↓$PaCO_2$	Sinus tachycardia $S_1Q_3T_3$ pattern ↓ T (V1–V4) Right bundle branch block	*Echo-cardiography V/Q scan *Pulmonary angiography
Acute severe asthma	*History of previous episodes, asthma medications, wheeze	Tachycardia and pulsus paradoxus. Cyanosis (late) *JVP→ *⇓ peak flow rhonchi	*Hyperinflation only (unless complicated by pneumothorax)	↓PaO_2 ↓$PaCO_2$ (until late)	Sinus tachycardia, (bradycardia with severe hypoxaemia—late)	
Acute exacerbation of COPD	*Previous episodes (admissions). If in Type II respiratory failure, may not be distressed	Cyanosis *Signs of COPD (barrel chest, intercostal indrawing, pursed lips, tracheal tug) *Signs of CO_2 retention (warm periphery, flapping tremor, bounding pulses)	*Hyperinflation, minor signs of emphysema, signs of events precipitating exacerbation (see p. 326)	↓ or ⇓PaO_2. In type II failure $PaCO_2$ may be ↑, with ↑ $[H^+]$ and ↑ bicarbonate	Nil, or signs of right ventricular failure (in cor pulmonale)	
Pneumonia	*Prodromal illness *Fever *Rigors *Pleurisy	Fever *Pleural rub *Consolidation Cyanosis (only if widespread)	*Pneumonic consolidation	↓$PaCO_2$ ↓PaO_2	Tachycardia	
Metabolic acidosis	*Evidence of diabetes/renal disease *Overdose of aspirin or ethylene glycol	Fetor (ketones) *Hyperventilation without physical signs in heart or lungs *Dehydration Air hunger (Kussmaul's respiration)	Normal	*PaO_2 normal ⇓$PaCO_2$ ⇓pH (↑ H^+)		
Psychogenic (a diagnosis of *exclusion*)	(Previous episodes)	***Not** cyanosed ***No** heart signs ***No** lung signs Carpo-pedal spasm	Normal	*PaO_2 normal ⇓$PaCO_2$ *pH (H^+) normal or ↑		End-tidal $PaCO_2$

* Denotes a valuable discriminatory feature


A
B
C
Right upper
Middle
Right lower
Liver
T_3
Left upper
Left lower
Spleen