

Chapter name: Respiratory data

Subheading: Imaging – plain radiography

Test: The chest X-ray

Indications

A 'routine' pre-operative chest x-ray (CXR) is not necessary unless there are specific indications. Management is changed as a result of less than 1% of pre-operative films. This figure increases as the ASA score rises. Acute change in cardiac/respiratory signs or symptoms is the only undisputed indication in elective non-thoracic surgery. See NIHCE guidelines (link at end of chapter). Similarly there seems to be no clear advantage to a 'routine' chest x-ray in ICU.

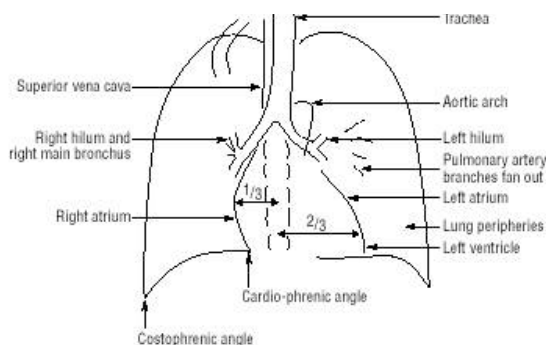
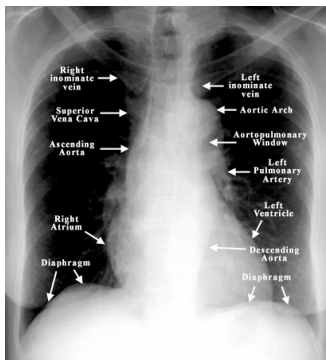
The chest x-ray is an essential part of the trauma series and is considered an important adjunct in the diagnosis of chest wall fractures, pneumothorax, haemothorax, and injuries to the heart and great vessels.

How it is done

The standard view is the posteroanterior (PA) film, with the patient facing the cassette and the x-ray tube two metres away. An anteriorposterior (AP) film is taken when the patient is unable to stand, with the cassette behind the patient. In the AP chest x-ray the heart will be magnified, and it is inappropriate to comment on cardiomegaly (unless it is gross) on this basis. A PA view shows the scapulae clear of the lungs whilst in AP they always overlap.

Interpretation

The chest x-ray is a two dimensional representation of a three dimensional structure. An understanding of chest anatomy is essential to interpretation of plain chest x-ray abnormalities.



The hilum is higher on the left but the hemidiaphragm is lower. The carina is at T4 on expiration and T6 in inspiration.

Due to the routine use of CT scanning, the lateral chest x-ray and decubitus film are now rarely seen. A lateral x-ray may differentiate structures that are unclear on PA chest x-ray.

A decubitus film is taken with the patient on his or her side to assess:

- (i) the volume and mobility of a pleural effusion on the dependent side
- (ii) a suspected pneumothorax in the non-dependent side in a patient who cannot be examined erect

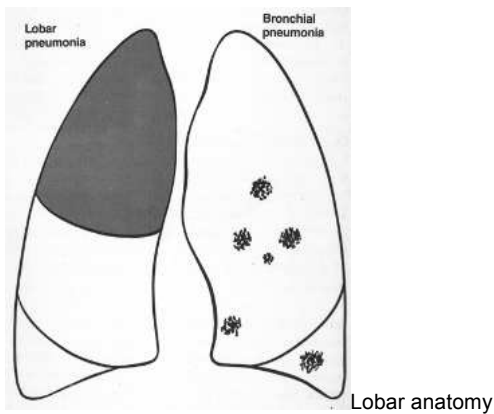
Abnormalities

Abnormalities of the lung

Consolidation is infiltration of the alveolar space by inflammatory tissue. The classic example is pneumonia: airspace disease and consolidation - not usually associated with volume loss. There may be an associated (parapneumonic) effusion.

The **silhouette sign** is an interface between isodense structures in contact with each other: the radiographic distinction between anatomical borders of lung and soft tissue can be 'lost' by abnormalities of the lung which increase its density.

Air bronchogram: When the air spaces fill with pus or fluid, the alveoli fill first with the bronchi being relatively spared, therefore the bronchi stand out. This is an air bronchogram - a sign of airspace disease such as consolidation, pulmonary oedema or atelectasis.



Atelectasis: a linear increased density and volume loss on chest x-ray. Some indirect signs of volume loss include vascular crowding or mediastinal shift, towards the collapse. There may be compensatory hyperinflation of adjacent lobes, or hilar elevation (upper lobe collapse) or depression (lower lobe collapse).

Interstitial disease

The interstitial space surrounds bronchi, vessels, and groups of alveoli. Disease in the interstitium manifests itself by *reticulonodular* shadowing (criss cross lines or tiny nodules or both). The main two processes affecting the interstitium are accumulation of fluid (pulmonary oedema) and inflammation leading to fibrosis.



The most common causes of interstitial fibrosis are:

- idiopathic (IPF, >50% of cases),
- collagen vascular disease,
- cytotoxic agents and nitrofurantoin,
- pneumoconioses,
- radiation
- sarcoidosis.

Figure: interstitial fibrosis

Pulmonary oedema may be cardiogenic or non-cardiogenic. In congestive heart failure, the pulmonary capillary wedge pressure (PCWP) rises and the upper zone veins dilate – this is called upper zone blood diversion. With increasing PCWP, interstitial oedema occurs with the appearance of Kerley B lines and prominence of the interlobar fissures. Increased PCWP above this level causes alveolar oedema, often in a classic perihilar bat wing pattern. Pleural effusions also occur. Unusual patterns may be found in patients with COPD who have predominant upper lobe emphysema.

A helpful mnemonic for noncardiogenic pulmonary oedema is NOT CARDIAC: near-drowning, oxygen therapy, transfusion or trauma, CNS disorder, ARDS, aspiration, or altitude sickness, renal disorder, drugs, inhaled toxins, allergic alveolitis, contrast or contusion.

Chronic obstructive pulmonary disease (COPD) is often seen on CXR as diffuse hyperinflation with flattening of diaphragms and enlargement of pulmonary arteries and right ventricle (cor pulmonale). In smokers the upper lung zones are commonly more involved.

Abnormalities of the mediastinum

On the PA film, the heart takes up to half of the total thoracic measurement in adults (more in children).

Mediastinal deviation: Tension pneumothorax and pleural effusion push the mediastinum away; lung collapse pulls it towards the affected side

Findings for *pneumomediastinum* include streaky lucencies over the mediastinum that extend into the neck, and elevation of the parietal pleura along the mediastinal borders. Causes of pneumomediastinum include

- Asthma
- surgery (post-op complication)
- traumatic tracheobronchial rupture
- abrupt changes in intrathoracic pressure (vomiting, coughing, exercise, parturition), ruptured esophagus

- barotrauma
- Crack cocaine

Pericardial Effusion: causes a globular enlarged heart shadow. More than 400ml of fluid must be in the pericardium to lead to a detectable change on plain x-ray. If it is chronic then there may be little functional impairment. An echocardiogram is indicated to detect ventricular impairment and to guide drainage.

Abnormalities of the hila and pulmonary vessels

Enlarged hila could be due to an abnormality in any of the three structures which lie there:

- *The pulmonary artery* (eg pulmonary hypertension or pulmonary embolus)
- *The main bronchus* (eg carcinoma)
- *Enlarged lymph nodes* (eg sarcoidosis)

Pulmonary Embolism (PE): Most chest x-rays in patients with a PE are normal. The primary purpose of a chest film in suspected PE is therefore to rule out other diagnoses as a cause of dyspnea or hypoxia. Further imaging is indicated, such as V/Q scan, pulmonary arteriogram, and CT pulmonary angiogram (CTPA).

Chest x-ray signs of pulmonary embolus may include:

- *Westermark's sign (oligemia in affected area)*
- *Enlarged hilum (caused by thrombus impaction)*
- *atelectasis with elevation of hemidiaphragm*
- *pleural effusion*
- *consolidation*

Pleural abnormalities

Pleural effusion: On an upright film, an effusion will cause blunting of the costophrenic angle. Sometimes a depression of the involved diaphragm will occur. A large effusion can lead to a mediastinal shift. Approximately 200 ml of fluid is needed to detect an effusion in the frontal film – a lateral is more sensitive.

In the supine film, an effusion will appear as a graded haze that is denser at the base. To differentiate it from lung disease, vascular shadows can usually be seen through the effusion.

Common causes for a pleural effusion include:

- Congestive heart failure
- infection (parapneumonic)
- trauma
- PE
- tumour
- autoimmune disease

Pneumothorax: A pneumothorax is air inside the thoracic cavity but outside the lung. It appears as air without lung markings in the least dependant part of the chest. It is best demonstrated by an expiration film. It can be difficult to see when the patient is in a supine position - air rises to the medial aspect of the lung becoming a lucency along the mediastinum. It may also collect in the inferior sulci causing a deep sulcus sign.

Causes of spontaneous pneumothorax include:

- idiopathic
- asthma
- COPD
- pulmonary infection
- neoplasm
- Marfans syndrome

A *hydropneumothorax* is both air and fluid in the pleural space. It is characterized by an air-fluid level on an upright or decubitus film in a patient with a pneumothorax.

Further investigations

Computed Tomography (CT) scan, Ventilation/perfusion (VQ) scan etc. See below

Limitations and complications

To be adequate, a chest x-ray must be:

- Taken on inspiration (unless suspecting a pneumothorax). On good inspiration the diaphragm should be at the 8-9th posterior rib or the 5-6th anterior rib.
- Adequately penetrated - thoracic disc spaces should be visible through the heart but bony details should not.

- Not rotated –medial heads of the clavicles equidistant from vertebral bodies
- Ionising radiation is contraindicated for women who might be pregnant. See below for radiation doses

Subheading: Other imaging

Test: Computed Tomography (CT) scan

Indications

- Identification of structural thoracic pathologies, including abnormalities of the pulmonary vasculature, lung parenchyma and mediastinum.

How it is done

- A beam of x-rays is passed through the chest and picked up on iodide crystals within a detector. These iodide crystals emit photons when struck by x-rays, which are detected by a photomultiplier.
- The x-ray tube passes around the patient, allowing multiple data to be collected for each section of the chest being viewed. This data is converted by a computer into two- or three-dimensional images
- Computed tomography can be given with intravenous contrast to highlight the pulmonary arterial tree. This is a CT pulmonary angiogram (CTPA). It can only show central emboli and gives a large dose of radiation to the patient.

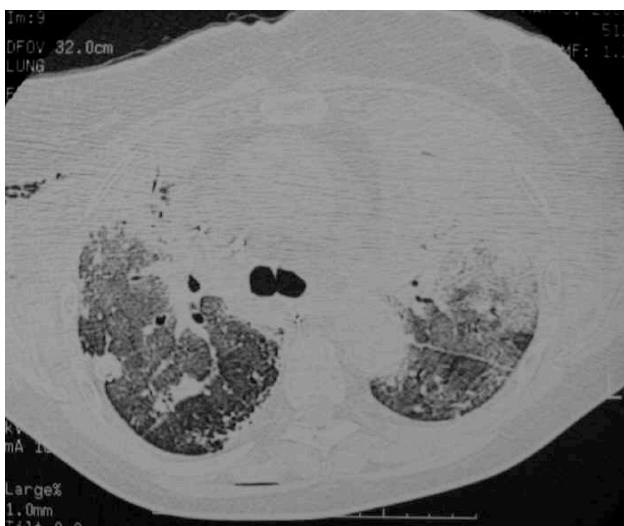


Figure:ARDS. Patient ventilated prone

Data presented as

- CT scans are viewed 'from the feet up' with the patient on their back
- As with a conventional x-ray, denser tissues are paler.

Limitations and complications

- CT scans give large doses of radiation to the patient.
- The CT scanner is often in a distant part of the hospital and considerable logistic difficulties arise when transporting a critically ill patient there.
- Medical staff may receive a radiation dose if a patient cannot be left alone in the CT scanner.
- Visualisation of the pulmonary vasculature requires intravenous contrast, which causes is contraindicated in patients with iodine allergy and may worsen renal impairment, especially in patients taking metformin
- Lesions less than 1 cm in size may be missed
- CT scans without contrast cannot differentiate between structures of very similar density

Radiation dose (millisieverts):

Annual background 2.5-3

Chest x-ray	0.1
Abdominal x-ray	2
CT head	2
CT Chest	8
CT abdomen	10

Test: Ventilation-Perfusion scan (VQ scan)

Indications

- Aims to detect mismatches in lung ventilation and perfusion, such as pulmonary embolus.
- There is a lower radiation dose for V/Q scans than for CTPA, making it the investigation of choice for the pregnant patient with suspected pulmonary embolus.

How it is done

- The pulmonary arterial circulation is viewed using intravenous technetium-99, which fixes in the distal pulmonary capillaries showing where blood is flowing. At the same time, the patient breathes a radioactive gas, usually krypton-81, which shows ventilation in the lungs

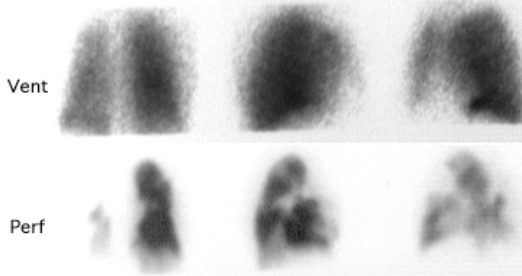


Figure: Abnormal perfusion suggesting multiple pulmonary emboli

Interpretation

Normal range

- A normal scan shows the same appearance in both the ventilation and perfusion images, showing no mismatch

Abnormalities

- A pulmonary embolus obstructs distal blood flow and so there are defects in perfusion. This is seen on a ventilation-perfusion scan as a perfusion mismatch, there being no obstruction to ventilation.

Further investigations

- A CT pulmonary angiogram may help with diagnosis if the VQ scan is equivocal

Limitations and complications

- A VQ scan may suffer from poor specificity: any abnormality in lung tissue will affect ventilation and perfusion to some extent. A VQ scan will therefore only detect pulmonary embolus in otherwise fairly normal lungs
- This investigation exposes the patient to a dose of ionising radiation, albeit small.
- This investigation cannot usually be done out of hours

Test: Positron Emission tomography (PET) scan

Indications

- Detection of tumours and bony deposits.

How it is done

- PET uses deoxyglucose, a glucose analogue labelled with fluorine-18, which is taken up avidly by malignant cells. Fluorine-18 emits positrons, which combine with electrons to produce photons.

Further investigations

- Staging investigations and tissue diagnosis directs subsequent management

Limitations and complications

- The (albeit small) dose of radiation given makes this examination contraindicated in pregnant women
- Because of its reliance on normal glucose metabolism, a PET scan can be unreliable in diabetic patients or those with insulin abnormalities.



Figure: pulmonary angiogram showing filling defect in right upper zone – pulmonary embolus

Rational selection of thoracic imaging modality

Thoracic imaging and the diagnosis of Pulmonary embolism

Pulmonary Angiography is the **gold standard**. In the presence of a non-diagnostic pulmonary V/Q scan, **pulmonary angiography commonly reveals pulmonary embolism** when the level of clinical suspicion is high. **Drawback: invasive and has morbidity and mortality.** Selective catheterisation of the right and left pulmonary arteries is done. Images are obtained in two views usually on a DSA machine. Acute pulmonary emboli are demonstrated as intraluminal filling defects, peripheral occlusion of pulmonary vessels and/or wedge shaped perfusion defects.

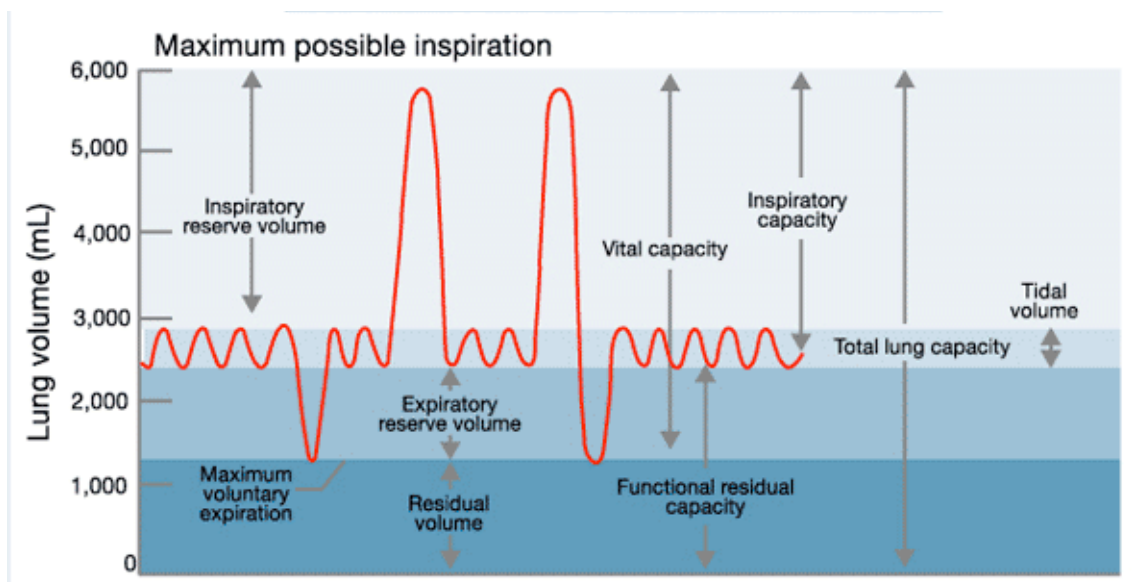
Imaging in Blunt Trauma to the Heart and Great Vessels

Chest CT scans are more sensitive than CXRs for the detection of injuries such as pneumothoraces and pulmonary contusions. Spiral CT has excellent accuracy in this setting but may be suboptimal in assessing arch vessels, where angiography may be more appropriate. Aortic angiography provides images of the entire thoracic aorta and arch vessels that are easy to interpret, however it is a time consuming technique and availability of service may limit its clinical utility.

Ultrasound: More recently the greater availability of handheld cardiac ultrasound devices and trained physicians skilled in their use, allow early ultrasonic assessment of the trauma patient in the emergency department. Pericardial effusions or tamponade can be reliably recognised, as can hemothoraces associated with trauma.

The decision to proceed to definitive radiological investigation is always a balance of risk versus benefit in the trauma setting. Full ATLS patient assessment and physiological stability is mandated prior to transfer.

Subheading: Pulmonary Function tests



Indication

- Seldom used in clinical practice although important physiological concept.
- For anaesthetists, the most important lung volume is *Functional Residual Capacity (FRC)*

How FRC is measured

- steady state or single breath helium dilution, nitrogen washout or whole body plethysmography
- steady-state helium dilution involves connecting the patient to a helium-containing spirometer at the end of a normal exhalation: the change in helium concentration allows a calculation of FRC

Interpretation

Normal Value: 20 mls per kg - 1.5L in a 70kg adult male

Physiological principals:

FRC is

- the combination of residual volume and expiratory reserve volume
- the volume left in the lungs after a normal exhalation
- the lung volume at elastic equilibrium.
- tested in combination with other lung volumes
- important as during apnoea it is an oxygen reservoir: if it falls below closing capacity, airway closure (and potential hypoxia) will occur during tidal breathing.

Abnormalities

Reduced:

- Restrictive lung disease (eg pulmonary fibrosis)
- Extrinsic lung compression (eg obesity, plural effusion, scoliosis)
- Reduced lung volume (eg post pneumonectomy)
- Pregnancy, lying supine, neonates
- Neuromuscular disease (eg Guillain-Barré Syndrome)

Increased:

- Airflow obstruction: emphysema, asthma

Further investigations

- flow/volume curves and arterial blood gas analysis

Limitations and complications

- dependent on age, sex and height

Test: Volume/time curve

Indications

With flow/volume curves, occasionally used preoperatively to

- investigate cause of shortness of breath (to differentiate obstructive from restrictive lung disease)
- assess degree of disease
- assess response to treatment (eg pre/post β_2 agonist) and decide if treatment is 'optimal'

How it is done

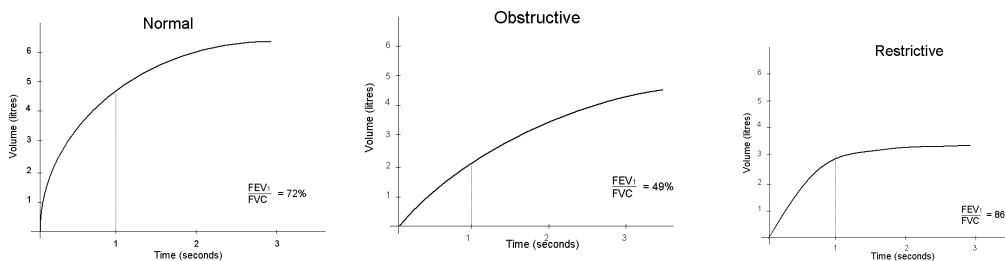
- measured by spirometer, which alone cannot measure volumes that do not take part in normal ventilation (ie RV and FRC).
- subject takes a full breath in and blows out as long, hard and completely as possible then takes a full breath in before resuming normal breathing
- repeated three times to ensure acceptable and reproducible results
- volume/times are recorded and compared to predicted (% expected)
- volume exhaled in 1 second (FEV₁) is compared to the volume of air that can be maximally forcefully exhaled (FVC, Forced Vital Capacity), giving a ratio
- results may be compared before/after bronchodilators ('reversibility')

Interpretation

Data presented as graphs and numerical (absolute numbers, % predicted)

Normal range/graph

- Normal FEV₁/FVC = 70-80%



Abnormalities

Obstructive lung defect: FEV₁/FVC = <70%

- the volume expired in 1 second is disproportionately small and the volume/time curve is flatter
- Example: COPD, asthma

Restrictive lung defect: FEV₁/FVC = >80%

- Due to smaller total lung volume
- Example: lung fibrosis, chest wall disease

Further investigations

- Flow/volume curves and Peak expiratory flow rate (PEFR) are usually measured at the same time

Limitations and complications

- Technique, recent use of bronchodilators, exercise, age height, gender and ethnicity can all affect results

Test: Flow/ Volume curve (dynamic)

Indications

- See volume/time curves

How it is done

- Principle similar to volume-time
- results may be compared before/after bronchodilators ('reversibility'): a change more than 20% is considered significant
- PEFR, Peak inspiratory flow rate (PIFR), FVC may also be measured

Interpretation

Data presented as graphs and numerical (absolute numbers, % predicted)

Normal range/graph

- Normal: see figure 2a

Abnormalities

- Intrathoracic airways obstruction
 - Airway compression during expiration produces a characteristic shape 'curvilinear' (fig 2b)
 - Increasingly severe disease lowers PEFR and FVC
 - Common causes: emphysema/bronchitis; asthma; bronchiectasis

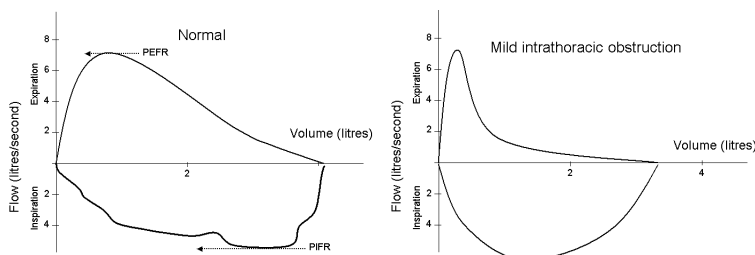


Figure 2a-b: Flow-volume loops in normal, mild and severe intrathoracic airway obstruction

- Extrathoracic obstruction (fig 3)
 - A fixed obstruction reduces both peak expiratory and inspiratory flow rates
 - On expiration, extrathoracic airway pressures are above atmospheric and hold the airway open: expiration is less affected
 - On inspiration, the decreased extrathoracic airway pressures narrow the airway: hence a greater effect on inspiration.
 - Causes include: tracheal stenosis, laryngeal paralysis, goitre
 - A variable obstruction may be held open during expiration by the above atmospheric extrathoracic airway pressures, so expiration is relatively unaffected.
 - Causes include pharyngeal muscle weakness of Obstructive Sleep Apnoea, laryngeal tumour

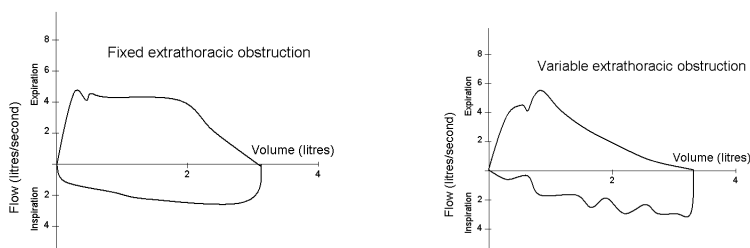


Figure 3 : Flow-volume loops in normal and extrathoracic obstruction (3a-b)

- Respiratory muscle weakness (fig 4)
 - Lower pressure/ slower rise of airway pressures cause
 - lower, later PEFR in expiration and lower flow throughout inspiration
 - loss of large airway flow changes ('expiratory spikes') when the patient is asked to cough (a test rarely done clinically)
- Restrictive Lung Disease
 - Reduced vital capacity, PEFR and accelerated emptying

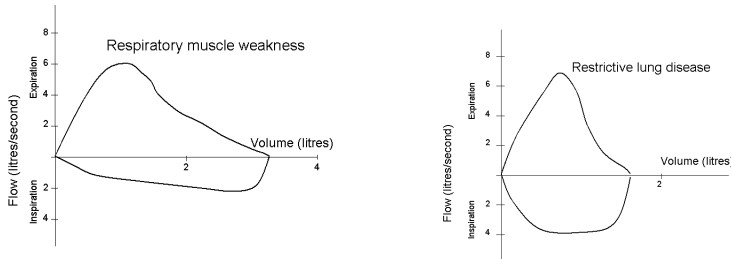


Figure 4 and 5 Flow-volume loop in respiratory muscle weakness and restrictive disease

Further investigations

- Volume/time curves (generating FEV₁/FVC) are usually measured

Limitations and complications

- See volume-time curves

Test: Transfer factor/Diffusing capacity

Indication

- To investigate disorders of the alveolar membrane

How it is done

- 0.03% carbon monoxide (CO) along with 10% helium (to measure alveolar volume) is held in a single breath for 10 seconds: the expired gas concentrations are measured
- If the subject is normal, then CO will be able to diffuse across the alveolus and the exhaled CO concentration will be appropriately low, resulting in a normal transfer factor
- The results are based on 3 factors
 1. the properties/surface area of the alveolar-capillary membrane
 2. the binding of CO to Haemoglobin
 3. the amount of Haemoglobin in pulmonary microcirculation
- The result may be expressed as a transfer factor or as a transfer coefficient per volume lung, KCO (Mmol / min / kPa /L)
- KCO helps differentiate conditions in which there is a reduction in surface area which is normal (eg pneumonectomy) from those where the surface area may be reduced, but there is abnormal alveolar membrane (eg emphysema)

Interpretation

Normal Value:

Normal T_{LCO} = 10-15 Mmol / min / kPa in a 25 year old male

Abnormalities

Reduced T_{LCO} but normal KCO:

- reduced lung volume with normal remaining gas transfer:
 - ↓ effort or respiratory muscle weakness, thoracic deformity, lung resection, anaemia

Reduced T_{LCO} but low KCO:

- reduced lung volume with abnormal gas transfer:
 - Emphysema, pulmonary emboli, interstitial lung disease (e.g. pulmonary fibrosis, sarcoidosis)
 - pulmonary hypertension, pulmonary vasculitis pulmonary oedema, excess carboxyhaemoglobin, pregnancy (12-26 weeks)

Increased T_{LCO}

- Polycythaemia, left-to-right shunt, pulmonary haemorrhage, asthma, exercise, pregnancy (up to 12 weeks)

Further investigations

- Performed with measurement of lung volumes, flow/volume curves and arterial blood gas

Limitations and complications

- dependent on age, sex and height
- Subjects must not have recently exercised, smoked, be anaemic or polycythaemic.

Sub-heading: Intra-operative respiratory monitoring

Test: Capnography

Indications

A requirement of minimum monitoring standards for patients undergoing general anaesthesia.

It gives useful information on:-

- CO₂ production and removal
- Lung perfusion
- Alveolar ventilation
- Altered airway dynamics
- Respiratory pattern and adverse respiratory events.

How it is done

Most commonly measured by infra-red (IR) spectography. Expired CO₂ is a polyatomic gas and absorbs IR rays (specific wavelengths around 4.8µm).

Interpretation

The amount absorbed is proportional to the concentration of the absorbing gas present. Concentration of CO₂ can be determined by comparing the measured absorbance with the absorbance of a known standard.

Data presented as

End tidal CO₂ (P_{ET}CO₂), expressed as partial pressure, measured in kPa or mmHg.

Physiological principles

During the respiratory cycle exhaled CO₂ produces a display of instantaneous CO₂ concentration versus time. In the healthy patient end-tidal CO₂ (P_{ET}CO₂) approximates to arterial CO₂ (PaCO₂). CO₂ in exhaled gas is dependent on its carriage from site of production in the tissues to the lungs via the right heart. Thus capnography also provides limited but useful information on cardiac output, pulmonary blood flow, and the diffusion of pulmonary capillary gases. A mismatch between P_{ET}CO₂ and PaCO₂ may occur due to both physiological and pathological processes.

Conditions affecting arterial – end-tidal CO₂ gradient	
Increasing gradient	Decreasing gradient
Age	Increased cardiac output
COPD	Low frequency ventilation
Pulmonary embolism	Pregnancy
Reduced cardiac output	Infants under anaesthesia
Hypovolaemia	
General anaesthesia	

Normal range

In the healthy patient P_{ET}CO₂ approximates PaCO₂ (5 – 5.6 Kpa)

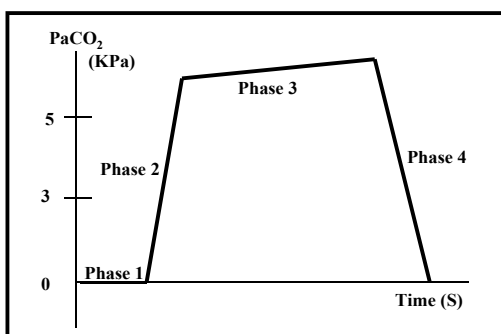


Fig 1 shows four phases of a normal capnograph trace

- Phase 1 - Baseline trace should read zero when no re-breathing occurs
- Phase 2 - Upstroke as exhaled dead space gas mixes with alveolar gas
- Phase 3 - Plateau represents alveolar CO₂ mixing
- Phase 4 - Inspiration occurs and CO₂ falls to zero

Abnormalities & Management principles

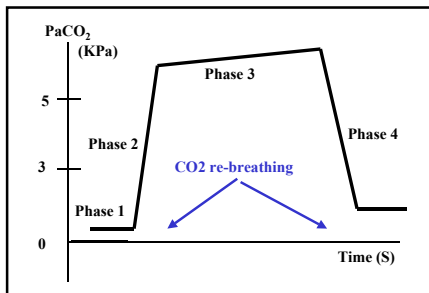


Fig 2. Baseline elevated suggests CO₂ re-breathing. **Check anaesthetic circuitry.**

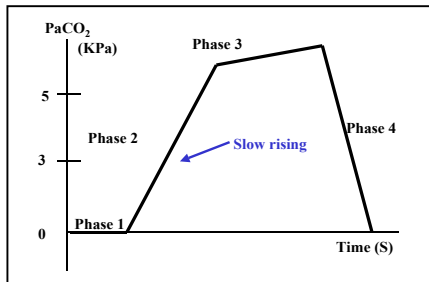


Fig 3. Slow upstroke phase 2 suggests obstruction to expiratory gas flow (e.g., asthma, bronchospasm, COPD, and kinked endotracheal tube or in the presence of leaks in the breathing system). **Examine patient and anaesthetic circuit**

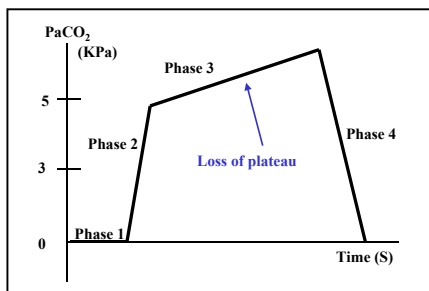


Fig 4 Loss of plateau in phase 3 suggests differing time-constants in the lung seen with COPD and emphysema. **Examine and optimise patient treatment.**

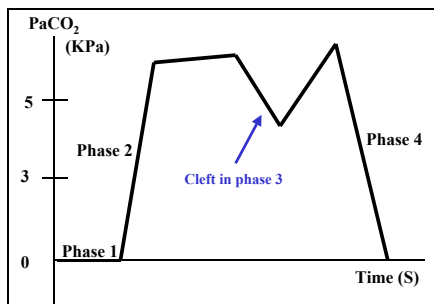


Fig 5 Cleft signifies return of respiratory effort during mechanical ventilation or artefact associated with abdominal movement during surgery. **Check adequacy of muscle paralysis.**

Shunt fraction

Indications

Hypoxaemia may be the result of shunting. Understanding its basis allows a rational approach to its correction.

How it is done

The total physiological shunt fraction can be calculated using the Shunt Equation, which allows calculation of the amount of venous blood bypassing ventilated alveoli and mixing with pulmonary end-capillary blood.

$$Qs/Qt = (CcO_2 - CaO_2) / (CcO_2 - CvO_2)$$

- CcO₂ = End capillary O₂ content.
- CaO₂ = Arterial O₂ content

- CvO_2 = Mixed Venous

Data presented as

When blood flow and oxygen content is known, the amount of shunt flow and its impact on systemic arterial oxygenation can be calculated. The shunt is described as a percentage of the cardiac output.

Physiological principles

Anatomical shunt exist with normal anatomy e.g. Thebesian and bronchial veins contribute a small degree of shunt in all humans.

Abnormal anatomical shunts are best divided into pulmonary and/or extra-pulmonary e.g. pulmonary A-V fistula or a atrial septal defect.

Physiological shunt = V/Q inequalities + Anatomical shunt.

Abnormalities & Management principles

A degree of shunting may be seen due to altered V/Q matching associated with anaesthetic agent use, IPPV, patient positioning and hydration status. However shunt is commonly the result of pulmonary pathology including, pneumonia, atelectasis and pulmonary oedema, which during anaesthesia may prove difficult to treat. True shunts respond poorly to increased oxygen concentration, but improvement in oxygenation may be seen with attention to the conduct of anaesthesia and optimising fluid balance, ventilatory settings including PEEP and patient positioning, thus correcting V/Q inequalities.

Further investigations

Shunt estimation 1: A-a gradient

The degree of shunt can be estimated by comparing the partial pressure of O_2 in the alveoli (A) to that in the artery (a). The A-a gradient is $(PAO_2 - PaO_2)$

$PAO_2 = (\text{barometric pressure} - \text{saturated water vapour pressure}) \times FiO_2 - PaCO_2 / \text{respiratory quotient}$

$PAO_2 = (101 - 6.3) \times FiO_2 - PaCO_2 / 0.8$ (or 1) (Alveolar gas equation)

Normal A-a gradient is 20 in a healthy young person and a rough estimation is $(\text{Age}+10) / 4$ (mmHg) A-a increases 5 to 7 mmHg for every 10% increase in FiO_2

Shunt estimation 2: a/A ratio

Another useful tool for estimating shunt is the **a/A ratio**. Here the same values as A-a gradient are divided, rather than subtracted. (PaO_2 / PAO_2) . Further simplification by substituting PAO_2 for FiO_2 offers a rough but useful estimate of shunt.

"P/F" Ratio (PaO_2 / FiO_2)

Advantage - doesn't use the alveolar gas equation.

Example:

Pa O ₂ KPa (mmHg)	FiO ₂	P/F ratio
12.5 (93.7)	0.21	59 (446)
10 (75)	0.30	33 (250)
8 (60)	0.70	11 (86)

Subheading: Pressure-Volume (P/V) curve analysis

Test: Static airway compliance – a measure of lung distensibility

Data presented as

Compliance is defined as the volume change per unit pressure change (DV/DP) (ml/cmH₂O)

Physiological principles

Many diseases result in altered pulmonary elasticity, which can be measured by changes in compliance (DV/DP), or elastance (DP/DV).

During spontaneous ventilation the total compliance of the chest wall and lungs, is approximately 100 ml/cmH₂O. The lung compliance is approximately 200 ml/cmH₂O. During ventilated anaesthesia, total compliance of the respiratory system is approximately 70–80 ml/cmH₂O.

Indications for measurement

1. A tool to analyse the mechanical properties of the respiratory system.
2. To guide ventilatory adjustments to optimise mechanical ventilation.
3. As a model to appreciate lung protective ventilation strategies.

How it is done

Static lung compliance is measured by sequentially inflating the lungs with a known volume of gas and measurement of the transmural pressure. The inspiratory occlusion technique involves the sequential measurement of plateau airway pressures corresponding to different tidal volumes during successive end inspiratory occlusions.

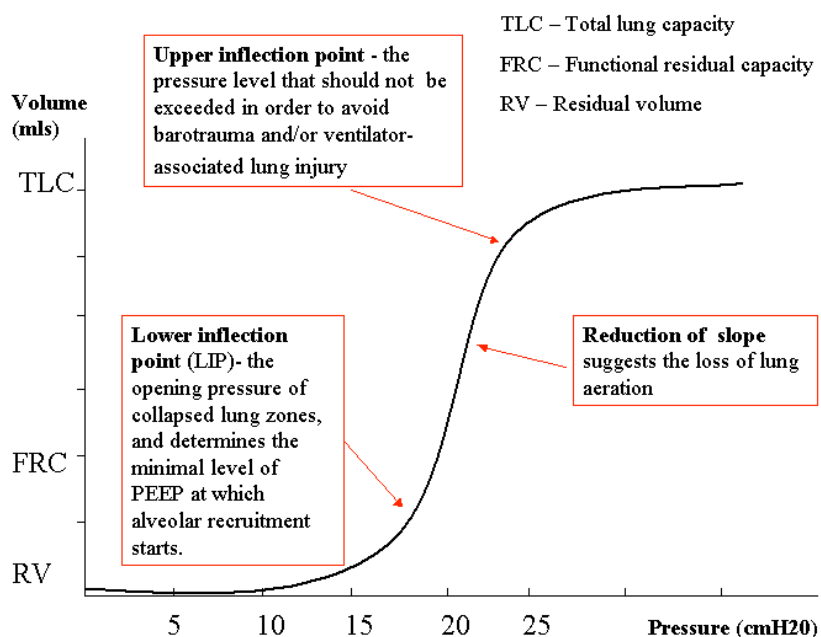
The quasi-static method uses a continuous inflation at a constant gas flow. Here the change in airway pressure is inversely proportional to the compliance of the respiratory system. A simple technique, the graphic is often incorporated into ventilator screen settings.

Interpretation

The lung at residual volume requires an opening pressure before inflation takes place. A lower inflection point indicates the pressure at which many collapsed alveoli are opening at the same time. Application of a PEEP that is equal to or greater than the pressure corresponding to the lower inflection point results in significant alveolar recruitment and decrease in pulmonary shunt. This approach may avoid mechanical ventilation-induced lung injury resulting from the repeated opening and closure of the terminal bronchioles during each respiratory cycle.

V-P relationship is linear around FRC until TLC is approached (UIP) (see figure). Above UIP overdistension of alveolar units occurs and no more recruitment achieved. On the P/V curve this point is situated around 30cmH₂O. A stiff lung (ARDS) has a low compliance, whereas a highly distensible lung (emphysema) has a high compliance.

Compliance is affected by posture, and will be increased with age and emphysematous lung disease. Increases in extravascular lung water, consolidation, poorly adjusted mechanical ventilation and fibrosis are common causes of reduced compliance.



Limitations of technique

Super syringe technique requires a patient to be disconnected from the ventilator and inspiratory hold techniques are time consuming. Both may be subject to error when intrinsic PEEP is present. The quasi-static technique may be of limited use where high gas flow rates result in high airway resistance.

Subheading: Arterial Blood Gas

Test: Arterial Blood Gas

Indications

- To diagnose acid-base disorders and monitor treatment
- To assess adequacy of ventilation and oxygenation

How it is done

- Anticoagulated blood from an arterial line or arterial puncture is used.
- Electrodes measure the pH, pO₂ (Clark) and pCO₂ (Severinghaus).
- The bicarbonate, base excess/deficit are calculated.
- Some machines may also measure Hb photometrically as well as electrolytes and lactate.
- Co-oximeters haemolyse the blood and measure total haemoglobin, fetal haemoglobin, oxyhaemoglobin, deoxyhaemoglobin, carboxyhaemoglobin and methaemoglobin by using absorbance at 6 different wavelengths, which is more accurate than photometric methods

Interpretation

Normal ranges

Abbreviation	Meaning	Normal value
pH	A measurement of the hydrogen ion concentration (log scale) $pH = pK + \log \left(\frac{[HCO_3^-]}{[CO_2]} \right)$	7.35-7.45
pCO ₂	Partial pressure of CO ₂	4.8-5.9 kPa
pO ₂	Partial pressure of O ₂ ; the FiO ₂ must be known.	11.9-13.2 ¹ kPa
BEx	Base Excess, a measure of the metabolic component of acid base disorders: the calculated amount in milliequivalents of strong acid required to restore 1 litre of fully saturated blood to pH 7.4, at a PCO ₂ of 5.3kPa. More than +2 = a metabolic alkalosis, less than -2 = a metabolic acidosis.	-2 to +2
sBEx	Standard Base Excess: the calculated base excess after the sample has been equilibrated ('standardised') with CO ₂ at 5.3kPa at 37 ⁰ c, saturated with oxygen and a haemoglobin of 5g/dL	-2 to +2
BDef	Base Deficit, a measure of the metabolic component of acid base disorders, is the opposite of base excess	-2 to +2
Total CO ₂	= CO ₂ + HCO ₃ ⁻	22-32 mEq/L
sHCO ₃	Standard Bicarbonate: the calculated bicarbonate concentration after the sample has been equilibrated ('standardised') with CO ₂ at 5.3kPa at 37 ⁰ c and saturated with oxygen. Like base excess, a measure of the purely metabolic component.	22-26 mmol/L
aHCO ₃	Actual Bicarbonate ie the bicarbonate calculated from the measured CO ₂ and pH: values vary if the CO ₂ is abnormal.	22-26 mmol/L

Table 10: definitions and normal ranges of measured and calculated acid-base parameters
¹breathing room air at sea level

Physiological principles

- Disorders may be classified according to the pH (acidosis or alkalosis) and whether the cause is respiratory (CO₂) or metabolic (bicarbonate)
- Each disorder may be compensated (tending to normalize the pH) or mixed (a combined metabolic/respiratory disorder)

Abnormalities

- The disorder can be calculated by working through Figure 20 (below).
- Respiratory compensation: for metabolic disorder minute volume will change in minutes to alter CO₂
- Metabolic compensation: for a respiratory disorder renal bicarbonate reabsorption will change within 12 hours, but complete correction takes some days.

Low pH with low sHCO₃ (<22) or BEx (<-2.2) - Metabolic Acidosis

- Acid ingestion eg Aspirin overdose
- ↑ Acid production
 - Tissue Hypoperfusion: lactic acidosis
 - Eg hypovolaemia, sepsis, cardiac failure
 - Diabetic Ketoacidosis: β hydroxybutyrate and acetoacetate
 - Hyperchloraemia- excess saline containing fluids (eg most colloids) especially with hypernatraemia
 - Hepatic failure
 - Glucose-6-Phosphate dehydrogenase deficiency
 - Drugs: Metformin, alcohols, reverse transcriptase inhibitors
 - Thiamine deficiency
- ↓ Acid elimination
 - Renal Failure: organic acids eg sulphuric acid
 - Distal renal tubular acidosis
- ↑ Bicarbonate loss
 - Diarrhoea, Large ileostomy losses, Small bowel fistulae
 - Urethroenterostomy, proximal renal tubular acidosis

Low pH with high pCO₂ (> 6.2 kPa) - Respiratory Acidosis

Chronic hypoventilation is compensated by HCO₃ retention

- Acute Hypoventilation
 - Central control- eg CNS depressant drugs, fatigue, CO₂ narcosis, encephalitis, brainstem disease, trauma
 - Airway obstruction – large (eg COETT) or small airways (eg Bronchitis/emphysema)
 - Respiratory muscle weakness or paralysis

- Reduced lung volume – eg pneumonia, reduced artificial ventilation, structural chest abnormalities
- Excess CO₂ production/administration
 - Hypermetabolism (eg malignant hyperthermia)
 - Failure of CO₂ absorber – rebreathing
 - Inadvertant CO₂ administration

High pH with high sHCO₃ (>26) or BEx (>+2.2) - Metabolic Alkalosis

- ↑ acid loss
 - Prolonged vomiting/ loss of gastric fluid
 - Conns, Cushings, Bartter's syndrome
- ↑ Base administration, retention or concentration
 - Excess bicarbonate
 - Excess citrate (eg blood transfusion)
 - Excess buffer in renal haemofiltration fluid (eg lactate) Loss of Cl⁻ (eg diuretics)
 - Renal retention of bicarbonate

High pH with low pCO₂ (< 4.2 kPa) - Respiratory Alkalosis

This is caused by hyperventilation

- Excess external mechanical ventilation
- Central nervous system
 - Pain, anxiety, fever, cerebrovascular accident, systemic inflammatory response, meningitis, encephalitis
- Hypoxaemia
 - high altitude, severe anemia, right-to-left shunts
- Drugs: eg doxapram, aminophylline, salicylate, catecholamines, stimulants
- Endocrine: pregnancy and hyperthyroidism
- Stimulation of chest receptors
 - pneumothorax/hemothorax, pulmonary infection / oedema / aspiration / embolism, interstitial lung disease

Management principles

- Identify and treat the cause
- Intravenous Sodium Bicarbonate (8.4% 50ml/hr) may worsen intracellular acidosis, but may buy time if there is a rapid rise in serum K⁺, the acidosis is severe (eg pH< 7.1) or if definitive treatment is awaited

Further investigations

- Anion Gap = ([Na⁺]+[K⁺]) - ([Cl⁻]+[HCO₃⁻])

Strictly, it refers to the venous electrolyte concentrations

In the context of a metabolic acidosis:

- 8 - 16 mEq/L = renal/GI HCO₃⁻ losses, or ↓renal acid excretion
- >16 mEq/L = an unmeasured anion eg lactic, methanol, ethanol, ketoacids, paraldehyde, renal failure, etc
- < 8 mEq/L = hyponatraemia, hypoalbuminaemia, paraproteinaemia
- Strong Ion difference = the difference in concentration between strong cations and strong anions
 - Normal = ~40 mEq/L
 - = [Na⁺] + [K⁺] + [Ca²⁺] + [Mg²⁺] - [Cl⁻] - [Other Strong Anions]
 - A strong ion is a highly dissociated cation or anion
 - Concept developed by Peter Stewart (1981)
 - Stewart showed that metabolic changes in acid base disorders were due to
 - the strong ion difference or
 - [A_{TOT}], total plasma concentration of the weak non-volatile acids - inorganic phosphate, or serum proteins such as albumin

Limitations and complications

- Heparin (an acid) lowers the pH: expel the heparin before taking the sample
- Large air bubble in the syringe may raise pO₂ and pH and lower pCO₂: expel the air
- Abnormal plasma protein levels affect the Base Excess/Bicarbonate
- Intravenous lipid may affect pH

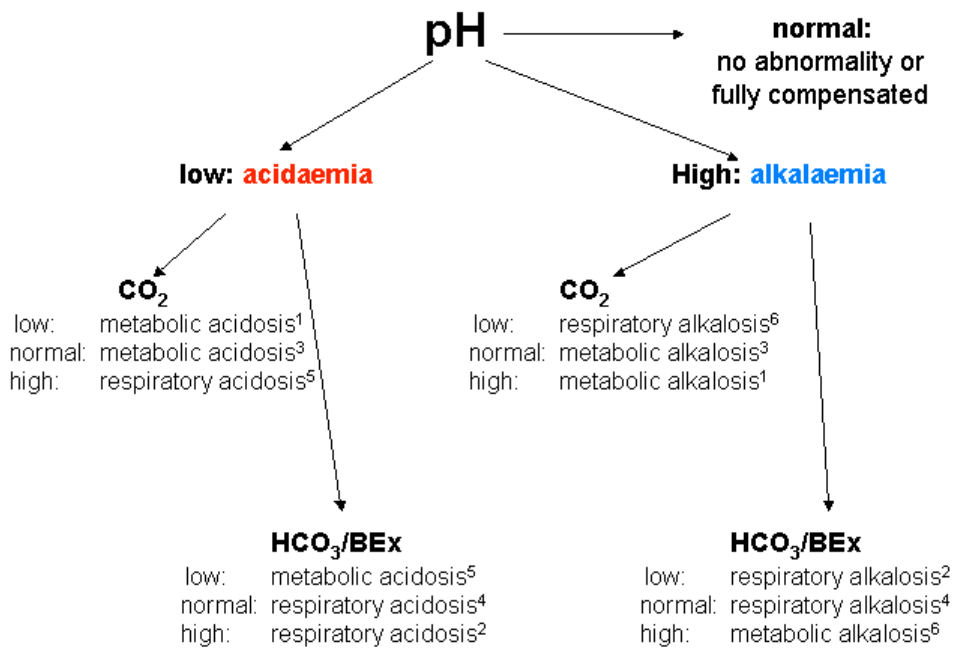


Figure 20: a flow diagram for interpretation of acid-base abnormalities

¹ = with attempted respiratory compensation (either acute or chronic).

² = with attempted metabolic compensation (ie treated with bicarbonate or longer than ~ 6 hours).

³ = with no respiratory compensation,

⁴ = with no metabolic compensation.

⁵ = mixed respiratory/metabolic acidosis if CO₂ high *and* HCO₃/BEx low.

⁶ = mixed respiratory/metabolic alkalosis if CO₂ low *and* HCO₃/BEx high.

⁷ acid base disturbances are rarely fully compensated by the patients natural mechanisms

Further reading:

1. NIHC guidelines on chest X-ray: <http://www.nice.org.uk/guidance/CG3/guidance/pdf/English>

2. www.acid-base.com

3. Oxford Textbook of Medicine: Chapter 11 section 12 Disturbances of acid–base homeostasis, OUP 2004 D Warrell, TM Cox, JD Firth, EJ Benz.