



The Airway and Lungs

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5.1 Inspection

5.1.1 Body Position

Extremely useful and relevant information can be obtained when analysing the position assumed by patients with dyspnoea. Relief of breathlessness in a sitting or standing position compared to the recumbent position is referred to as orthopnoea. While increased venous return in the supine patient is well tolerated in individuals with a preserved heart func-

tion, this leads to pulmonary venous congestion, an increase in interstitial lung water and a subsequent reduction of lung capacities with resultant shortness of breath in patients with impaired heart function. Accordingly, patients with heart failure prefer to sit upright (e.g. supporting their back with pillows to achieve a maximum upright position) (Fig. 5.1). Conversely, placing the patient into a supine position may be used as a stress test to exclude respiratory distress due to heart failure or (pulmonary) fluid overload. A history of paroxysmal nocturnal dyspnoea characterized by repeated awakening due to breathlessness while sleeping in the recumbent position is a typical symptom of heart failure.

Trepopnea is a phenomenon encountered in patients with heart failure (e.g. in those with right-sided pleural effusion), asymmetrical pulmonary disease (large atelectasis or total lung collapse, pleural effusion, pneumonia, patients post pneumonectomy) or mediastinal/endobronchial tumours. It describes the occurrence of dyspnoea in one lateral position as opposed to the other. As gravity causes blood to be redistributed in the chest, dyspnoea develops in the lateral position with the more diseased side of the lung placed downwards. In clinical practice, this effect can also be used therapeutically (“place the good lung down!” to improve oxygenation).

Patients with acute asthma or an exacerbation of chronic obstructive pulmonary disease (COPD) feel most relief from dyspnoea when sitting and leaning forward with their arms stemmed on their

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Fig. 5.1 Critically ill patient with acute heart failure and respiratory distress at admission to the intensive care. Courtesy of Martin W. Dünser, MD. Note the sitting position and the pillows placed under the back of the patient to relieve dyspnoea. Furthermore, note the tanned appearance of the patient (obviously from sunbathing) indicating the patient has been active until before this episode of acute illness



knees or the bed (Fig. 5.2). This position allows maximizing respiratory muscle contraction. Patients with COPD who regularly take this position may develop hyperkeratosis of the skin over the knees and distal thighs (Dahl sign).

Platypnea refers to breathlessness which occurs or increases in the upright position but is relieved with recumbency. In these patients, breathlessness is frequently accompanied by deoxygenation (orthodeoxia). This phenomenon can be observed in patients with right-to-left shunts through intra-cardiac or more often intra-pulmonary shunts [e.g. (bi)basal pneumonia, basal emphysema or arterio-venous shunts such as in patients with the hepatopulmonary syndrome or Osler disease]. Pathophysiologically, gravitational redistribution of the blood to more affected basal parts of the lungs can explain the occurrence of dyspnoea in the upright position in these patients.

5.1.2 Chest Form, Chest Wall Expansion and Symmetry

Visual inspection of the chest can reveal important clues about lung function. Chest wall deformities such as kyphosis, scoliosis, kyphoscoliosis, severe funnel (pectus excavatum) or pigeon-



Fig. 5.2 Typical body position taken by a patient with an acute asthma attack. Courtesy of Martin W. Dünser, MD

shaped (pectus carinatum) chests are associated with reduced lung capacities and resultant restrictive lung disease. A barrel-shaped chest is suggestive of the presence of underlying COPD and/or lung hyperinflation. Similarly, centripetal (abdominal) obesity may be associated with a reduction in chest wall compliance and lung capacities. Scars of previous thoracic surgeries indicate that the patient may have reduced lung capacities (e.g. due to lung resections). In patients

with COPD, lung apices may be seen and palpated in the supraclavicular region. Enlarged intercostal spaces with bulging lung tissue are less frequently noted over the lateral chest wall during acute exacerbation in asthenic patients. Significant deformities of the chest due to trauma (e.g. “stove-in chest”) are rare, but, if present, they are associated with life-threatening/fatal lung and/or mediastinal injuries.

The range of chest wall expansion during inspiration is a good clinical marker of tidal volume. Patients with barely visible expansions of the (lower) chest typically have (very) low tidal volumes and are at high risk of respiratory failure. Common causes are reduced pulmonary or chest wall compliance, COPD, respiratory muscle fatigue or neuromuscular diseases. In obese patients, the extent of chest excursions is difficult to assess and making conclusions about the size of tidal volume unreliable.

When assessing the symmetry of chest wall expansions, it is important to make sure that the

patient is lying flat so that asymmetry is not due to the patient’s position. Asymmetrical chest wall expansions reflect asymmetrical lung ventilation and can arise from pneumothorax, atelectasis or consolidation (e.g. pneumonia). While a pneumothorax results in elevation of the affected hemithorax, total lung collapse/volume loss leads to reduced chest wall expansion with the affected hemithorax lagging behind the contralateral side. In both pneumothorax and total lung collapse/lung collapse, chest wall expansions of the affected hemithorax are reduced. Rarely and only in asthenic patients result unilateral lung diseases (e.g. pneumonia) in reduced ipsilateral chest wall expansions.

Patients with multiple rib fractures can present with an unstable chest wall (“flail chest”). This is particularly common in patients in whom several adjacent ribs of the anterior or lateral chest wall have fractured into one or more free pieces (Fig. 5.3). This chest wall segment then moves inwards during spontaneous inspiration and outwards during expiration delicately compromising

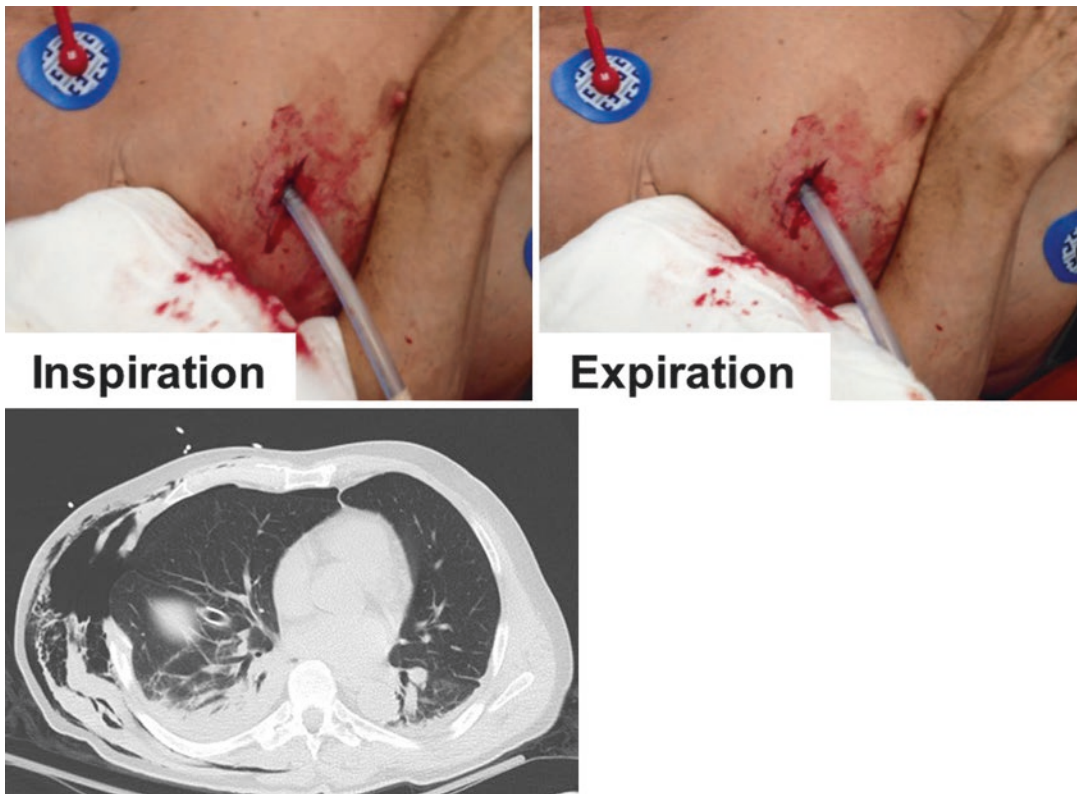


Fig. 5.3 Flail chest in a patient with serial lateral rib fractures with a large mobile chest wall segment and corresponding chest computer tomography scan. Courtesy of Martin W. Dünser, MD

chest wall mechanics and possibly gas exchange (which is usually impaired due to concomitant underlying lung damage or contusion; see Fig. 5.3). In patients with large freely moving chest wall components, a mediastinal shift (or “flutter”) may occur with changes of intrathoracic pressures over the respiratory cycle and cause additional hemodynamic instability. In most cases, an unstable chest can be recognized by inspection of the (anterior or lateral) chest wall. Palpation with the examiner’s palms placed over the anterior and lateral chest wall helps to detect smaller flail segments. In mechanically ventilated patients, positive airway pressure prevents the free chest wall part from moving inwards during inspiration, thus stabilizing the chest wall. In these patients, freely moving chest wall parts can only be detected by meticulous palpation. Abnormal or paradoxical chest movements may be observed in patients after cardiac surgery with sternal infection and instability. In some of these patients, partial or total sternectomy with muscle flap reconstruction is performed leaving them with a chronic unstable chest which is obvious on clinical inspection, as the muscle flap typically moves inwards during inspiration and outwards during expiration.

5.1.3 Skin Colour

Central cyanosis characteristically affects the lips, oral/sublingual mucosa and tongue (Fig. 5.4). It reflects severe hypoxaemia but can

also be seen in patients with meth- (>1.5 g/dL, brownish hue or “chocolate” cyanosis) or sulfhaemoglobinemia (>0.5 g/dL). Central cyanosis becomes visible if the absolute quantity of capillary/venular blood in the lips or oral mucosa exceeds 4.25 g/dL (>2.38 g/dL in the arterial blood) of deoxygenated haemoglobin. In non-anaemic patients, this corresponds to an arterial oxygen saturation of approximately 80%. Clinical recognition of central cyanosis can be tricky with false-positive and false-negative results. In anaemic patients, hypoxaemia must be more profound before central cyanosis develops. For example, in patients with a haemoglobin concentration of 7.5 g/dL (e.g. as may occur in patients in the intensive care unit), central cyanosis only becomes detectable when oxygen saturation drops to values $<50\%$ (again corresponding to a concentration of deoxygenated haemoglobin of at least 4.25 g/dL in mucosal capillaries). In severe anaemia, central cyanosis may not be detected clinically despite the presence of severe hypoxaemia. Finally, skin complexion can affect the threshold at which central cyanosis is detected. In patients with dark complexed skin and lips, it is more practicable to inspect the oral (sublingual or buccal) mucosa and tongue for the presence of central cyanosis (Fig. 5.4b). Patients with polyglobulia (e.g. those with COPD) may appear cyanosed already at mildly reduced arterial oxygen saturations. Pseudocyanosis (central cyanosis without hypoxaemia) can result from chronic metal intoxication

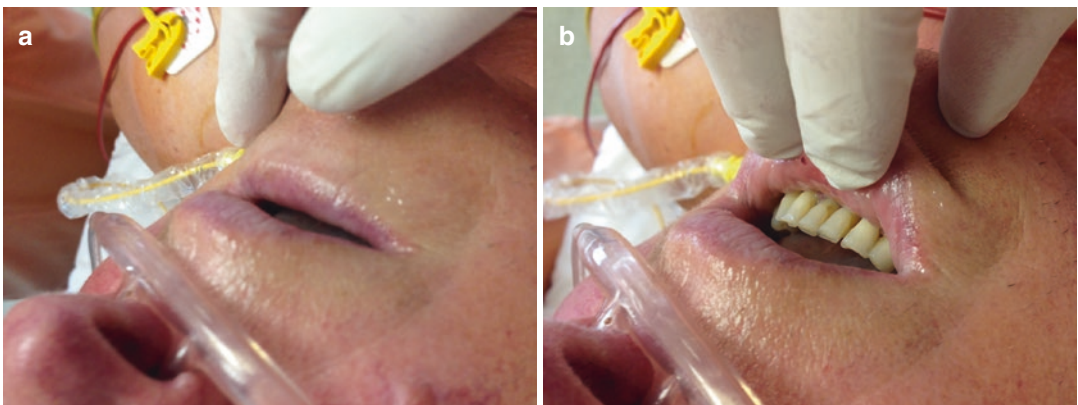


Fig. 5.4 Central cyanosis recognized by bluish discoloration of the lips (a) and oral mucosa (b) Courtesy of Martin W. Dünser, MD

(e.g. silver, gold) or complicate long-term drug therapies (e.g. amiodarone, minocycline, chloroquine, phenothiazines). Facial plethora with diffuse erythema of the upper chest is a clinical sign of acute hypercapnia (Fig. 5.5).

Chronic cyanosis can be detected more reliably than acute cyanosis as patients commonly have elevated haemoglobin levels. Other signs of chronic hypoxaemia include facial plethora, clubbing of fingers (drumstick fingers) and toes and nail bed cyanosis. Although clubbing is largely seen in patients with lung diseases leading to chronic hypoxaemia [e.g. pulmonary fibrosis, asbestosis, lung cancer (usually absent with small cell carcinoma of the lung), mesothelioma, suppurative lung disease including empyema, lung abscess and bronchiectases and only very rarely with COPD], it can be hereditary or result from non-pulmonary diseases such as cyanotic heart disease, inflammatory bowel disease or chronic liver disease. In some patients, ballotability of the nails or sponginess of the nail beds, an early stage of clubbing, has been observed within as short as 2 weeks after onset of pulmonary disease.



Fig. 5.5 Facial plethora with diffuse erythema of the upper chest in a patient with acute hypercapnia. Courtesy of Martin W. Dünser, MD

5.1.4 Respiratory Rate

The respiratory rate is determined by counting the number of chest wall expansions over 20–30 s and then multiplying it to attain the number of breaths per minute. A respiratory rate between 10 and 15 breaths per min is physiologically normal in the resting individual. Except in some elderly patients, in whom respiratory rates may physiologically reach 25 breaths per min, any increase >20 breaths per min must be considered abnormal and referred to as tachypnoea. The degree of tachypnoea is a solid but non-specific indicator of disease severity with respiratory rates >30 breaths per min often associated with life-threatening conditions. Tachypnoea is more valid to predict subsequent cardiac arrest in hospitalized patients than tachycardia or abnormal arterial blood pressure. A persistently normal respiratory rate is, conversely, a useful finding that makes certain pathologies (e.g. shock, significant pulmonary embolism) rather unlikely. Physiologically, a rise in respiratory rate increases alveolar ventilation, carbon dioxide elimination and alveolar oxygen tension. Tachypnoea can therefore not only be observed in (acute) lung disease but also in patients with reduced systemic oxygen delivery and metabolic acidosis. Despite this, tachypnoea, in clinical practice, correlates notoriously poorly with the degree of hypoxaemia. Furthermore, ventilation can be stimulated by increased sympathetic tone (e.g. pain), inflammation (e.g. sepsis) and cerebral dysfunction (e.g. cortical or midbrain lesions). Unlike most other patients, patients with metabolic acidosis first increase their alveolar ventilation by an increase in tidal volume and only later by an increase in respiratory rate. This form of tachypnoea is referred to as hyperpnoea and is physiologically the most effective way to eliminate carbon dioxide via the lungs as dead space ventilation is minimized. Although in some cases, an increase in tidal volumes may be evident as “Kussmaul” breathing, increased minute ventilation in patients with metabolic acidosis is difficult to recognize. It often

only becomes apparent when surprisingly low pH ranges have been reached. As very low (<7.1–7.2) pH values are most often caused by anion gap acidosis (mostly of diabetic origin), the most common causes of hyperpnoea include ketoacidosis, lactic acidosis, poisoning (e.g. salicylate, toxic alcohols, carbon monoxide, cyanide, isoniazid, paraldehyde, iron) and uraemia.

5.1.5 Respiratory Rhythm

Physiologic breathing is rhythmic. Characteristic changes to this rhythm can be observed and important clinical information be gleaned. The most commonly observed pathologic breathing rhythm in acute disease is Cheyne-Stokes breathing. It is characterized by alternating episodes of gradually increasing and fading hyperpnoeic episodes interrupted by spells of apnoea (Fig. 5.6). Apnoeic spells may persist for up to 45 s but usually last for 5–10 s only. While Cheyne-Stokes breathing can be physiologic in selected patient groups (e.g. infants) or certain conditions (e.g. during ascent to high altitude), it is a valid clinical sign of an acute cerebral or chronic cardiac pathology. It rarely results in apnoea.

This is in contrast to Biot breathing which resembles Cheyne-Stokes breathing at first sight but differs in that alternating episodes of hyper- and apnoea start and stop more abruptly. Overall, alterations of hyper- and apnoeic spells are less regular than in Cheyne-Stokes breathing. Biot breathing is fairly uncommon but a sensitive

indicator of pontine or brainstem pathology. This explains why patients with Biot breathing are at an increased risk of apnoea.

Apneustic or ataxic breathing is characterized by an irregular respiratory rate and tidal volumes. The patient typically holds the breath at the end of each inspiration before the next cycle of expiration starts at an irregular, slow rate. It reflects a preterminal sign (brainstem pathology or severe brain hypoperfusion) and usually precedes gasping and respiratory arrest (see Part I Sect. 4.3).

5.1.6 Breathing Pattern

Four breathing patterns are essential to recognize in the critically ill patient: the physiologic, paradoxical, obstructive and restrictive breathing pattern.

Physiologically, contraction of the diaphragm and intercostal muscles raises both the chest and abdomen during inspiration. Expiration occurs passively with the chest and abdomen descending. The normal time ratio of inspiration to expiration is 1:2.

Paradoxical breathing refers to the inward movement of the chest while the abdomen raises during inspiration. This breathing pattern is seen in patients with an obstructed airway (e.g. comatose patient in the recumbent position) and needs to be recognized without delay. Paradoxical breathing (or abdominal paradox) also occurs in patients with severe respiratory distress or diaphragmatic dysfunction (remember: abdominal respiratory movements indirectly indicate how the diaphragm is moving). In these patients, the abdomen moves inwards while the chest wall raises. It is a highly sensitive and alarming sign of impending respiratory decompensation. Furthermore, paradoxical breathing can be observed in patients with cervical spinal cord injury when only the diaphragm contracts moving the abdomen outwards and the chest inwards during inspiration.

In patients with an obstructive breathing pattern, exhalation of air is impaired. Clinically, this becomes evident by active abdominal con-

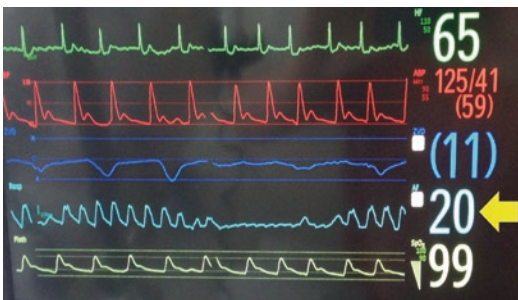


Fig. 5.6 Vital data monitor showing a respiratory curve suggestive of Cheyne-Stokes breathing/respiration (yellow arrow marks tracing of respiration). Courtesy of Martin W. Dünser, MD

traction during expiration. The chest often descends slowly and incompletely with the abdominal muscles contracting and moving the abdomen down- and outwards. The time used for expiration exceeds the time for inspiration. The most common clinical conditions leading to impaired expiratory airflow and an obstructive breathing pattern are asthma and COPD and pulmonary fluid overload/oedema which cause small airway collapse. Using the lips to generate a positive expiratory pressure (“pursed lip breathing”) is common in patients with an obstructive breathing pattern, particularly those with emphysema. It reduces respiratory rate, increases tidal volume (by up to 500–600 mL) as well as carbon dioxide elimination. Furthermore, the increase in end-expiratory pressure as mediated by “pursed lip breathing” shifts the diaphragm into a better position at the beginning of inspiration and thereby improves diaphragmatic function.

A restrictive breathing pattern is characterized by a prolonged and strenuous inspiration. Expiration usually follows a normal pattern. In contrast to obstructive breathing, the time required for inspiration exceeds that for expiration. Clinically, restrictive breathing is recognized by inspecting the upper chest wall. The most common clinical conditions leading to a restrictive breathing pattern are pulmonary diseases with a reduced lung or chest wall compliance (e.g. lung fibrosis, ARDS, early interstitial lung oedema). If expiratory flow limitation is severe and air trapping occurs in patients with asthma/COPD, restrictive and obstructive breathing pattern can be observed at the same time.

Being able to differentiate between the four types of breathing patterns requires experience and a long time of “active” clinical training [e.g. by paying specific attention to the breathing pattern in every critically and non-critically ill patient]. The information the clinician can get from correctly recognizing the breathing pattern is crucial. If one is unsure, it often helps to place one or both hands on the patient’s chest and/or abdomen to feel the (paradoxical) movements (Fig. 5.7).



Fig. 5.7 A simple method to differentiate between the four essential breathing patterns involves placing the hands on the patient’s chest and abdomen to feel the movements of the chest wall and abdomen over the duration of the respiratory cycle. Courtesy of Martin W. Dünser, MD

5.1.7 Work of Breathing

Dyspnoea is the subjective feeling of breathlessness and cannot be clinically assessed but only relayed by the patient. On the other hand, the work of breathing can be assessed by inspection. Although clinical signs of an increased work of breathing usually correlate well with the degree of dyspnoea, some patients (e.g. those with COPD) surprisingly do not feel dyspnoea despite of an obvious increase in the work of breathing.

The work of breathing reflects the efforts of the respiratory muscles to generate airflow sufficient for alveolar gas exchange. It is critically determined by respiratory muscle strength, chest wall compliance and the underlying lung function. Inspiration is physiologically achieved by contraction of the diaphragm and intercostal muscles. At rest, expiration occurs passively. In respiratory distress, additional muscles are used for both inspiration and expiration. Accessory muscles for inspiration include (most importantly) the scalene and sternocleidomastoid muscles. In severe respiratory distress, the trapezius muscle and platysma are activated, the latter of which has, however, only minimal effects on inspiratory chest expansion. The oblique abdominal muscles are the predominant accessory muscles of expiration. Specific and general physical

signs of an increased work of breathing are summarized in Table 5.1.

Indirectly, respiratory distress and by that the work of breathing can be determined whether the patient can speak in full sentences. Patients with an increased work of breathing can only speak single words or speak in a staccato fashion (e.g. few words spoken with each breath). Assessment of the work of breathing and indirectly the vital capacity is particularly important in patients with acute neuromuscular diseases, first of all the

Table 5.1 Clinical signs of an increased work of breathing

Specific symptoms:

- Sitting/upright position (see Fig. 5.1)
- Inability to speak in full sentences
- Forced or laboured inspiratory and/or expiratory efforts
- (Twitchy) use of accessory respiratory muscles (e.g. scalene, sternocleidomastoid and trapezius muscles; Fig. 5.8)
- Arms stemmed on knees/thighs (tripod stance) (see Fig. 5.1)
- Intercostal, suprasternal or supraclavicular retractions (Fig. 5.8)
- Nose flaring (ala nasal flaring)
- Elevation of the shoulders and/or movements of the head synchronous with inspiration (Fig. 5.9)
- Elevation of eyebrows and eyelids synchronous with inspiration
- Inspiratory (downward) retractions of the trachea and larynx during inspiration
- Upward motion of the clavicles and shoulders during inspiration
- Backward movement of the head during inspiration
- Sucking sound during inspiration in intubated patients who breath spontaneously (DD: cuff pressure too low)

General symptoms:

- Diaphoresis, profuse (cold) sweating
- Agitation
- Restlessness
- Tremor or jerks (in hypercapnia)
- Anxiety, fear of death
- Reduced sensorium (e.g. talking/praying without responding to voice)
- Depressed mental state^a

^aAs a result of hypercapnia ($\text{PaCO}_2 > 80\text{--}90$ mmHg or $>10\text{--}12$ kPa) and/or hypoxia ($\text{PaO}_2 < 30$ mmHg or <4 kPa). Also note that patients with neuromuscular weakness, in contrast to those with cardiopulmonary compromise, often remain awake despite very high PaCO_2 levels

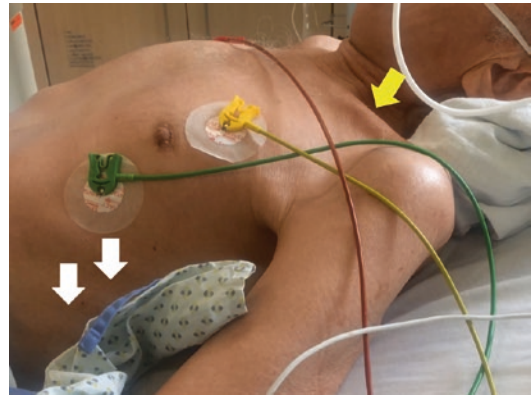


Fig. 5.8 Intercostal (white arrows) and supraclavicular retractions (yellow arrow) in a patient with COPD and respiratory distress. Courtesy of Martin W. Dünser, MD



Fig. 5.9 Use of accessory muscles (e.g. scalene and sternocleidomastoid muscles—white arrow) and involuntary elevation of the shoulders synchronous with inspiration (yellow arrow) in a patient with respiratory distress and an increased work of breathing. Courtesy of Martin W. Dünser, MD

Guillain–Barré syndrome. In these patients, inability to count to more than ten in a single breath is highly indicative of a critically reduced (forced) vital capacity (e.g. <1 L) and the need for endotracheal intubation. Conversely, patients who can count to 20 or higher with one breath usually have forced vital capacities within the safe range. Rapid progression of muscular weakness, particularly if it involves facial, neck and proximal extremity muscles, can highlight critical drops in vital capacity. A reduction in the volume of the voice and the inability to lift the head or elbow are further danger signs of impending respiratory failure.

In patients with a pleural drainage and a water seal in place, the swing of the water level (in the container) over the respiratory cycle reflects changes in pleural pressure. In patients with an increased work of breathing, typically large swings of the water level can be seen during inspiration and expiration.

5.1.8 Patient-Ventilator Dyssynchrony

Most episodes of patient-ventilator dyssynchrony are observed during assisted spontaneous breathing. However, they can also occur during controlled mechanical ventilation when patients “fight” against the ventilator. Clinically, this scenario is highlighted by the ventilator cycling off inspiratory efforts despite very low tidal volumes administered. The best way to detect whether a patient is fighting the ventilator is to touch the abdomen (Fig. 5.10). All patients who fight controlled mechanical ventilation contract their abdominal muscles and have a tender abdominal wall on palpation. Interestingly, certain patient populations are at specifically high risk for patient-ventilator dyssynchrony during controlled mechanical ventilation (e.g. cardiac surgical patients during the first postoperative hours). If abdominal muscles are not contracted and tidal volumes are low, other causes than patient-

ventilator dyssynchrony must systematically be excluded (e.g. partial/complete tube blockage, disconnection, ventilator dysfunction). Another form of patient-ventilator dyssynchrony observed during controlled mechanical ventilation is that of wasted efforts. Clinically, this can be recognized by the patient taking inspiratory efforts without the ventilator supporting them. Such “wasted” inspiratory efforts go along with only minimal chest wall expansion and can best be detected on the upper and anterior chest wall.

During assisted spontaneous breathing, patient-ventilator dyssynchrony can arise from the mismatch of patient and ventilator efforts with regard to timing of inspiration or expiration, as well as inspiratory flow and duration. Furthermore, inadequate flow trigger sensitivity can lead to patient-ventilator dyssynchrony. Ventilator waveform analysis is the best method to detect and interpret patient-ventilator dyssynchrony. General clinical signs are tachycardia, hypertension (sometimes also hemodynamic instability), sweating, anxiety and agitation. Respiratory symptoms of patient-ventilator dyssynchrony are often only subtle. When patient-ventilator dyssynchrony is suspected, it is particularly useful to continuously observe the patient’s breathing pattern for at least 1 min while trying to detect distinct patterns of patient-ventilator dyssynchrony (Table 5.2).



Fig. 5.10 Abdominal palpation to detect or exclude whether the patient is fighting the ventilator during controlled mechanical ventilation. Courtesy of Martin W. Dünser, MD

5.1.9 Tracheobronchial Secretions

Inspection of tracheobronchial secretions is an important part of the clinical examination of lung function and search for an infectious source. Foamy, frothy, pink or rose to meat water coloured secretions indicate pulmonary oedema. In severe pulmonary oedema, tracheobronchial secretions may be as extensive that they fill or exit the patient’s mouth. Before this occurs, the voice of the patient often becomes gurgling. In intubated patients with severe pulmonary oedema, foamy, frothy fluids can be seen in the endotracheal tube during each expiration or over the entire respiratory cycle (Fig. 5.11). Sometimes the amount of oedema fluid entering the tube is small, rapidly fades and can only be detected by

Table 5.2 Distinct patterns of patient-ventilator dyssynchrony with corresponding clinical findings and possible underlying problems

Type of patient-ventilator dyssynchrony	Clinical findings	Problem
Wasted efforts	Minimal chest wall expansions between breaths with greater chest wall expansion	Wrong ventilatory mode (e.g. controlled, unassisted), trigger sensitivity too low, dynamic hyperinflation
Flow asynchrony	Delayed chest wall expansion during inspiration, in severe cases can resemble the picture of an obstructed airway	Inspiratory flow rate is too low, inspiratory time is too long
Auto-triggering	Tachypnoea without signs of increased work of breathing	Trigger sensitivity too high, water/secretions in ventilator tubings
Double triggering	Two inspirations following each other without sufficient expiration in between	Too low pressure support, too short inspiration
Coughing, fighting, repeated inspiratory pressure alarms	Coughing or fighting the ventilator at the end of inspiration	Pressure support or inspiratory flow too high (e.g. leak around the mask during non-invasive ventilation), inspiratory time too long

careful inspection and recognition of small amber fluid streaks forming on the inner surface of the tube.

While the amount of tracheobronchial secretions correlates with the severity of pulmonary oedema, it is rather the appearance which is of relevance for clinical interpretation in other pulmonary conditions. In rare cases, tracheal secretions may appear normal despite the presence of pneumonia or even diffuse alveolar haemorrhage. Sometimes tracheal secretions only increase during the resolution of pulmo-



Fig. 5.11 Massive lung oedema with oedema fluid in the endotracheal tube and ventilation tubings in a patient with transfusion-related acute lung injury (TRALI) on extracorporeal membrane oxygenation. Courtesy of Martin W. Dünser, MD

nary infections. It is, however, a rule of thumb that the longer an infectious process in the lungs persists (e.g. chronic bronchitis or bronchiectasis), the more unlikely it is that tracheal secretions will remain unaltered. Large amounts of normally appearing rather liquid tracheal secretions can occasionally be seen in intubated or tracheotomized critically ill patients with no obvious lung pathology and may reflect a hypersecretory response to the tracheal foreign body.

Yellow, brownish, putrid or purulent tracheal secretions strongly suggest an ongoing pulmonary infection. Although not entirely specific, purulent secretions are mostly seen in bacterial infections of the distal airways and alveoli rather than the bronchi or trachea. Production of foul-smelling, yellow to dark green secretions should make the clinician consider bronchiectasis (Fig. 5.12) or a lung abscess. Although sporadically correct (e.g. rust-coloured purulent secretions in pneumococcal infection, greenish secretions in pseudomonas infection, whitish thickened secretions in viral or fungal infections), the colour of tracheal secretions does not allow definitive conclusions regarding the underlying pathogen or microorganism but is rather a clinical predictor of an underlying respiratory infection.

Pink or blood-stained tracheal secretions (haemoptysis) reflect haemorrhage in the tracheobronchial tree, distal airways or alveoli, but sometimes



Fig. 5.12 Aspirated tracheal secretions of a patient with bronchiectasis. Courtesy of Sirak Petros, MD

result from aspiration of the blood from the upper airways (nose, pharynx) or gastrointestinal tract. While small amounts of blood suctioned from the trachea of patients intubated for several days frequently arise from minor tracheobronchial tears (e.g. due to repeated suctioning, particularly when coagulation is impaired), it may indicate sentinel bleeding from a serious underlying lesion (e.g. tumour, pulmonary artery erosion, arteriovenous fistula). Fresh, bright red blood produced or suctioned from the airways is always an emergency and requires immediate attention. When compared to bleeding from other organs or tissues, pulmonary haemorrhage can rapidly result in death (due to suffocation) even with minor to moderate amounts of blood lost (e.g. >250–500 mL). In addition to the aforementioned pathologies, a myriad of clinical conditions can cause haemoptysis (e.g. pulmonary embolism, bronchitis, pneumonia, tuberculosis, lung abscess, bronchiectasis, bronchial carcinoma, bronchial adenoma, mycetomas, diffuse alveolar haemorrhage, trauma, recreational drugs such as crack or cocaine, congestive heart failure, mitral stenosis).

5.1.10 Miscellaneous

Further to what has been previously mentioned, the clinician can obtain additional information about lung function and pathology by simple inspection. For example, inspection of the fingers may reveal nail bed tarring or yellow distal finger



Fig. 5.13 Reactive herpes labialis infection in a patient with severe community-acquired pneumococcal pneumonia and associated pleural empyema. Courtesy of Martin W. Dünser, MD

segments as a sign of active often heavy cigarette smoking. Reddish discoloration of the cheek may be seen ipsilateral to an infectious pulmonary process (mostly community-acquired pneumonia). Similarly, the chest wall over a pleural empyema is often reddish and hyperaemic. Labial herpes infection (herpes labialis—herpes simplex virus type 1) is common in patients with or recovering from a pneumococcal infection such as pneumonia or meningitis (Fig. 5.13).

5.2 Listening

5.2.1 Without the Stethoscope

In some patients, especially those with severe respiratory dysfunction, a few distinct breathing sounds may be readily audible with the “naked” ear.

Stridor refers to a characteristic harsh, high-pitched sound which occurs during inspiration in patients with an upper/extrathoracic airway stenosis. It results from turbulent airflow generated by partial airway obstruction. As only significant narrowing of the airway (80–90% obstruction) results in stridor, it is a rather late sign of airway compromise and must be regarded as an absolute emergency. Once stridor can be heard, complete obstruction is of imminent concern, particularly in situations when stridor develops rapidly (e.g. in patients who re-bleed

after carotid surgery, have anaphylaxis, inhaled a foreign body or in whom subcutaneous emphysema spreads rapidly). Before stridor is heard, it can be perceived using a stethoscope placed over the neck (Fig. 5.14). The volume of an inspiratory stridor does not correlate with the degree of airway obstruction as it also depends on the velocity of inspiratory airflow. This is essential because the volume of a stridor often decreases as the patient decompensates and can no longer generate large enough airflows to pass through the airway stenosis. Clinical symptoms frequently preceding stridor include problems swallowing, a feeling of neck swelling and/or that something is blocking the airway and a change in voice. Notably, any change in voice in an acutely unwell or critically ill patient must be taken seriously as it may be a symptom of a potentially life-threatening condition (e.g. airway compromise, recurrent laryngeal nerve palsy, aortic aneurysm). Conversely, a normal voice suggests that the upper airway is patent and that the patient protects her/his airway.

An expiratory, high-pitched sound may be heard without a stethoscope in patients with severe intrathoracic (e.g. foreign body), mostly distal airway obstruction. Loud expiratory wheezing is a symptom of an asthmatic attack or COPD exacerbation. Identical to inspiratory stridor, the volume of the expiratory stridor does not correlate with the degree of airway obstruction.



Fig. 5.14 Placing the stethoscope over the neck can help to recognize stridor that cannot (yet) be heard without a stethoscope. Courtesy of Martin W. Dünser, MD

Impending respiratory decompensation is heralded by a decrease in the volume of expiratory wheezing. A “silent chest” describes the condition when airflow has decreased to such an extent that expiratory wheezing is not audible even with a stethoscope. Occasionally, a monophonic wheeze is heard during inspiration and expiration (mostly only with the stethoscope). This is caused by partial obstruction of the distal trachea or tracheal bifurcation (e.g. by a tumour, mediastinal mass or foreign body). Polyphonic wheezing (differing tones of wheeze) is heard in patients with varying reductions in airway diameter or calibre.

In patients with severe, life-threatening pulmonary oedema, usually of cardiogenic origin, crackles may be heard without a stethoscope. Crackles are then heard during expiration and resemble the sound heard when air is blown through a straw into a glass of water. This is in contrast to auscultation using the stethoscope when crackles are primarily heard during inspiration. Only when oedema floods the alveoli and reaches the distal airways can crackles be heard during expiration as well. The more oedema fluid enters small and larger airways, the louder the crackles become until they can be heard with the “naked” ear. Another sound which can occasionally be heard in patients with pulmonary oedema is grunting. Grunting arises from vocal cord closure during expiration followed by their sudden and short opening. Physiologically, vocal cord closure increases end-expiratory pressure and by that functional residual capacity and oxygenation. Although grunting has been reported as a sign of respiratory muscle fatigue, grunting breathing is usually associated with a rather low respiratory rate (approximately 20 breaths per minute).

A sound which is heard more commonly is that of airway secretions. They result in coarse, loud crackly and gurgling sounds during inspiration and especially expiration. While secretions in the trachea and bronchi sound more muffled and distant (like small water bubbles in a closed container), secretions in the pharynx and glottis generate a louder, less muffled and “closer” sound. One study found that the presence of gur-

gling breath sounds during quiet breathing or speech in hospitalized patients was independently associated with hospital-acquired pneumonia [1]. In dying patients, inspiratory and expiratory sounds due to airway secretions are often heard and have been referred to as the “death rattle”.

Partial airway obstruction is typically associated with gurgling and/or snoring sounds (see Part I Sect. 3.1). Gurgling sounds can be heard during inspiration and sometimes also expiration. They indicate that secretions or semi-solid materials are obstructing the larynx or pharynx. Snoring, on the other hand, is heard only during inspiration and results from partial occlusion of the pharynx by the tongue, soft palate and/or epiglottis. The absence of any breathing sounds is not helpful to verify airway patency as both complete obstruction and full airway patency produce no breathing sounds.

Cough is a common but non-specific symptom of pulmonary disease. Only in exceedingly rare cases are the characteristics of a cough specific enough to allow diagnosis of a certain pulmonary disorder (e.g. whooping cough). Barking or croupy cough is suggestive but not specific for viral upper airway disease. Productive or “chesty” coughs allow for inspection and interpretation of tracheobronchial secretions. Although commonly associated with pulmonary infection, a relevant number of patients with chest infection may present with non-productive cough. This appears to be more common in atypical or nonbacterial pulmonary infections. The history is essential to interpret cough in subjects with community-acquired critical illness. For example, cough accompanied by fever, sweats and productive sputum makes acute pulmonary infection likely. Increased coughing in a patient with a history of smoking, prior cough, productive sputum and wheezing is highly suggestive of a COPD exacerbation. A personal/family history of atopic disease together with new or worsening cough (particularly nocturnal) and wheezing is strongly indicative of an acute asthma attack. A drug history should also always be taken to exclude drug-induced coughing in patients with persistent cough (angiotensin-converting enzyme inhibi-

tors, beta blockers including topical usage of these agents, e.g. in glaucoma). Symptoms of dyspepsia, particularly gastro-oesophageal reflux disease, should also be inquired about in such patients.

In critically ill patients with a cough of new onset, it is important to exclude lung infection and pulmonary oedema. Acute pulmonary oedema is, in its early stages, often associated with a quite distinctive non-productive, superficial, staccato-like cough. With increasing severity of pulmonary oedema, coughing occurs following almost each inspiration. Physiologically, cough in patients with pulmonary oedema is thought to arise from stimulation of interstitial juxtacapillary receptors (J-receptors) by increased lung water. These receptors may also be involved in the coughing response to pulmonary embolism, barotrauma/pneumothorax, lung hyperinflation (e.g. recruitment manoeuvre) or re-expansion of atelectasis (e.g. after drainage of a large pleural effusion). A similar cough as with early pulmonary oedema can be heard in patients with dysphagia who aspirate saliva or liquids (e.g. during the water swallow test). In patients with shock, particularly heart failure, the haemodynamic response to coughing can be used as an indicator of the underlying cardiovascular pathology. Prolonged arterial hypotension or even cardiovascular collapse in response to coughing or more frequently “fighting the ventilator” in a patient on catecholamine support is highly suggestive of right heart failure. Less commonly, patients with severe left heart failure or hypovolaemia may experience aggravation of haemodynamic instability following coughing or “fighting the ventilator”.

5.2.2 Auscultation: Listening with the Stethoscope

The stethoscope allows the examiner to perceive acoustic phenomena of the trachea, large bronchi and lung peripheries. As only sounds generated by airflow in the immediate sub-pleural tissues can be heard, the sensitivity of auscultation to diagnose specific pulmonary diseases (e.g. pneumonia) is traditionally low. Auscultation in

the critically ill is challenged by the fact that patients usually cannot sit up so that it needs to be performed with the patient in a recumbent position (Fig. 5.15). This implies that fewer parts of the lung can be auscultated than in the standard sitting position. Controlled mechanical ventilation results in different pulmonary air distribution and airflows in the lung as compared to (assisted) spontaneous breathing. Critically ill patients frequently cannot take deep breaths or cooperate so that transmitted voice sounds could be assessed. Finally, the settings where critically ill patients are cared for (e.g. pre-hospital scene or hectic resuscitation room) are noisy making it difficult for the examiner to recognize subtle acoustic phenomena.

5.2.2.1 Normal Breath Sounds

Breath sounds are generated by airflow turbulences. During inspiration, when air moves from large to small airways thereby colliding with multiple bronchial walls and bifurcations, more airflow turbulence is produced and breath sounds are louder. During expiration, when air moves from small to large airways, less airflow turbulence occurs and breath sounds are typically less intense in volume, sometimes even difficult to hear. Depending where the examiner places the stethoscope, normal breath sounds differ in their

character. Placing the stethoscope directly over the trachea (e.g. over the neck, jugulum or *manubrium sterni*) allows the examiner to hear the harsh and loud character of tracheal breath sounds during both inspiration and expiration. By placing the stethoscope over the bronchial system (e.g. lower part of the sternum or parasternal area), loud, high-pitched sounds with a short pause between inspiration and expiration are heard and referred to as bronchial breath sounds. Vesicular breath sounds are perceived when placing the stethoscope over other parts of the chest wall and auscultating the lung peripheries. Vesicular breath sounds are soft, low-pitched and breezy. They can be heard throughout inspiration. After a short end-inspiratory pause, vesicular breath sounds can physiologically be heard during early expiration but are usually louder during inspiration (Table 5.3).

5.2.2.2 Bronchial Breath Sounds Auscultated over the Lung Peripheries

Bronchial breath sounds are present when sounds are heard during inspiration and expiration and are of the same intensity and duration. It is distinctly abnormal to hear bronchial breath sounds instead of vesicular breathing over the lung peripheries. Bronchial breathing occurs when patent airways are surrounded by fluid-filled or consolidated adjacent lung tissue, thus allowing for better transmission of breath sounds from the large bronchi to the lung peripheries. Common conditions in which vesicular breath sounds are replaced by bronchial breath sounds are pneumonia, the acute respiratory distress syndrome (ARDS) or fluid overload states.

5.2.2.3 Diminished or Absent Breath Sounds

The volume or intensity of breath sounds physiologically depends on several factors including the thickness of the chest wall as well as the velocity and amount of air entering the lungs. This explains, for example, why only diminished breath sounds may be heard in obese patients or those with a small tidal volume [e.g. during shallow breathing or (lung protective) mechani-

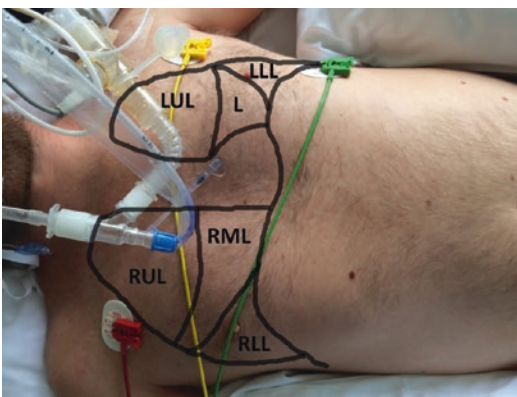


Fig. 5.15 Schematic overview of the lung anatomy in relation to the skin when auscultating a patient in the recumbent position. Courtesy of Martin W. Dünser, MD. LUL left upper lobe, L lingula, LLL left lower lobe, RUL right upper lobe, RML right middle lobe, RLL right lower lobe

Table 5.3 Summary of abnormal breath sounds

Breath sound	Description	Timing	Interpretation
Bronchial breath sounds over lung periphery	Harsh and loud breath sounds instead of normal vesicular breathing	Inspiration and expiration	Fluid-filled lung such as in pneumonia/consolidation, ARDS or fluid overload
Diminished or absent breath sounds	Breath sounds diminished in volume or completely absent	Inspiration and expiration	Bilateral: shallow breathing, lung protective ventilation, emphysema, dynamic hyperinflation (“silent chest” in asthma or COPD), bilateral pneumothorax (rare), thick chest wall (e.g. obesity) Unilateral: bronchial intubation, atelectasis, pleural effusion, pneumothorax
Crackles	High-pitched, clicking or crackling	Early inspiration	Bronchopneumonia, bronchitis, COPD or bronchiectasis (typically coarse)
	Like Velcro being pulled apart	Pan- or late inspiratory	Lung fibrosis, interstitial lung disorders
	Like strands of hair being rolled between the fingers	Pan- or late inspiratory	Bilateral (from base to top): lung oedema (typically fine and late inspiratory) Unilateral or localized: pneumonia
Rhonchi (retained secretions)	Low-pitched (coarse), snoring, vibrating and sometimes gurgling	Inspiration and expiration	Liquid or semi-solid materials in the tracheobronchial tree (e.g. tracheobronchial secretions)
Wheeze	Continuous musical, squeaking or whistle-like sound	Expiration	Localized and monophonic: tumour, mucus plug, foreign body generalized and polyphonic: asthma, COPD, fluid overload, pulmonary congestion
		Inspiration and expiration	Partial obstruction of the distal trachea or tracheal bifurcation (e.g. by a tumour or foreign body)
Pleural friction rub	Brushing sound like walking on snow	Inspiration and expiration	Inflammation or irritation of the pleura

ARDS acute respiratory distress syndrome, COPD chronic obstructive pulmonary disease

cal ventilation]. As often painfully experienced, the quality and type of the stethoscope can dramatically influence the perceived volume of breath sounds, too. When auscultating patients in the recumbent position, it is physiologic that less air enters the basal versus the upper parts of the lung physiologically, resulting in diminished breath sounds in dependent lung fields. This is especially pronounced during controlled mechanical ventilation where inspiratory airflow is generated by positive instead of negative pressure and air is mostly distributed to non-dependent lung areas.

Furthermore, several pathologic conditions (e.g. emphysema) can reduce the volume of breath sounds. As no air enters collapsed lung tissues (atelectasis), (bronchial) breath sounds over these lung areas are diminished. As the breath sounds heard are transmitted from adjacent lung

segments, substantial parts of the lungs (e.g. large parts of a lobe) need to be collapsed and un-aerated before breath sounds cannot be heard anymore. Air or fluid in the pleural space also diminishes the intensity of breath sounds. In the majority of patients with a pneumothorax, pleural air evenly encases (and compresses) the lung. This leads to diminished and distant or, in a large pneumothorax, absent breath sounds over the affected hemithorax. Due to the aforementioned fact that breath sounds are transmitted from adjacent lung segments, breath sounds from the contralateral lung can be heard over the parasternal chest wall even in patients with a pneumothorax. Another important cause for absent breath sounds over one hemithorax is (unnoted) bronchial intubation and one-lung ventilation. Even though the amount of pleural fluid collections may be massive (e.g. in severe haemothorax), it is

unusual that breath sounds are diminished or absent over the entire hemithorax. As fluid collects in the dependent areas of the pleural cavity, breath sounds are typically diminished or absent over these parts. Compression atelectasis commonly accompanies pleural effusions. In some patients, fine crackles are heard over the transition zone between compressed and aerated lungs and are indicative of the lung segments which open and collapse during each respiratory cycle. Differentiating between pleural effusion and atelectasis by auscultation alone is difficult. In awake and cooperative patients, it may help to test for transmission of voice. If the patient says “eee” and the examiner hears “aaa” over the affected lung part, atelectasis rather than effusion is likely present. This phenomenon is referred to as aegophony and results from enhanced transmission of sound through consolidated or non-aerated lung tissue, in contrast to pleural fluid (may also be heard where fibrotic lung is present).

5.2.2.4 Crackles

Crackles have formerly been referred to as crepitations or rales. Currently, the term crackle is the preferred terminology. Crackles refer to high-pitched, clicking or crackling non-musical breath sounds which are heard during inspiration. Depending on their occurrence during inspiration, they are divided into early or late crackles. Generally, crackles are produced by explosive opening of alveoli or distal airways during inspiration. Crackles heard during early inspiration result from the popping open of airways >2 mm in diameter and are lower-pitched than late or pan-inspiratory crackles. Early inspiratory crackles sound coarse and are encountered in patients with bronchopneumonia, bronchitis, COPD or bronchiectasis. Late- or pan-inspiratory crackles are finer, higher-pitched and result from opening of collapsed alveoli during inspiration. Alveolar diseases such as pneumonia, lung oedema or interstitial lung disease/fibrosis are characteristic pathologies resulting in late- or pan-inspiratory crackles. In contrast to lung oedema and interstitial lung disease/fibrosis, pneumonia results in localized crackles often accompanied by rhon-

chi (or retained secretions). While crackles resulting from alveolar pulmonary oedema have been compared to the sound of a strand of hair being rolled between the fingers, crackles in patients with interstitial lung diseases/fibrosis (e.g. idiopathic pulmonary fibrosis) have been compared to the sound made by Velcro being pulled apart. Interestingly, crackles cannot be heard in all patients with pulmonary fibrosis. This seems to depend on the underlying pathology of fibrosis, as crackles are heard in almost every patient with idiopathic pulmonary fibrosis (which causes fibrotic changes of the terminal bronchioli and alveoli) but only a minority of those with pulmonary fibrosis from sarcoidosis (which causes fibrotic changes alongside the bronchovascular bundle but not the terminal small airways).

The extent and severity of lung oedema can be determined by auscultating the lateral chest wall at different levels in the recumbent patient (Fig. 5.16). The more anterior crackles can be heard, the more severe lung oedema usually is. In patients with severe disease in whom oedema fluid spreads from the alveoli to more proximal airways, crackles together with expiratory rhonchi can be heard. In some patients with heart failure, basal crackles can be induced by changing the position from semi-recumbence to the supine position. In patients with pulmonary venous congestion, crackles can often be heard within a few minutes in the supine position. Before crackles are heard, wheezing frequently occurs as the lungs fill with water, gain weight and compress basal distal airways thus compromising expiratory airflow.

5.2.2.5 Rhonchi

Rhonchi are low-pitched (coarse), snoring, vibrating and sometimes gurgling breath sounds. They are typically heard during both inspiration and expiration but are usually louder during expiration. Partial obstruction of medium-sized and large airways by liquids or semi-solid materials (mostly thick secretions) is the most common pathology resulting in rhonchi. These sounds are sometimes also referred to as retained secretions.

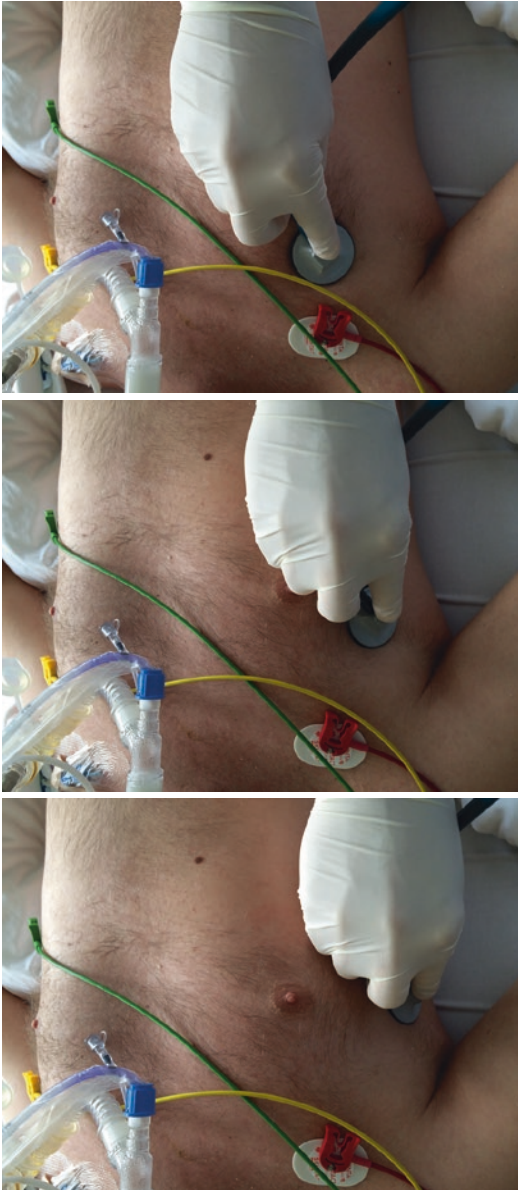


Fig. 5.16 Assessing the severity of (cardiogenic) lung oedema by auscultating at different levels of the lateral chest wall (see text for further explanation). Courtesy of Martin W. Dünser, MD

5.2.2.6 Wheeze

A wheeze is a highly characteristic continuous musical, squeaking or whistle-like breath sound heard during expiration. Wheezing results from significant (>50%) narrowing of smaller airways. It can be localized or heard over both lungs. A localized wheeze is commonly monophonic

(produced by a single tone) and results from the obstruction of a single (larger) airway, for example, by a tumour, mucus plug, foreign body or compression by a mediastinal mass. In few but notable cases, the distal opening of the endotracheal tube (without a Murphy's eye) directly faces the posterior wall of the trachea which intermittently obstructs the tube during expiration resulting in severe prolongation of expiratory airflow and a monophonic wheeze. Similarly, a monophonic wheeze can be heard over both lungs in patients with vocal cord dysfunction who present with asthma-like symptoms. A wheeze that can be heard over both lungs is usually polyphonic as it results from several tones due to narrowing of different sized and located airways ("concertus asthmaticus"). Multiple conditions can cause airway narrowing, of which the archetypical is bronchoconstriction due to asthma or anaphylaxis. In critically ill patients, the most common conditions associated with a wheeze are COPD, pulmonary fluid overload and left heart failure. It is important to note that in severe small airway obstruction (e.g. severe asthma attack or COPD exacerbation) or with very low airflows (e.g. low tidal volumes in respiratory decompensation or (ultra)lung-protective ventilation), the volume of wheezing is reduced or even absent ("silent chest"). However, pitch and length of the wheeze correlate with the severity of expiratory airflow obstruction.

5.2.2.7 Pleural Friction Rub

The pleural friction rub is a characteristic brushing sound which resembles the sound that occurs when walking on snow or rubbing two pieces of leather together. It is caused by inflammation (pleuritic) or irritation of the visceral and/or parietal pleura. In contrast to crackles, the pleural friction rub is heard during both inspiration and expiration and usually localized to a rather small area. A pleural friction rub is rarely encountered in critically ill patients but can occasionally be heard in patients with pneumonia or those with a recent pulmonary embolism. In patients with pleural drains of larger size (>20 Charrière) and under negative pressure, a squeaking friction rub may be heard. In patients with bronchopleural fistula and a chest drain in place, the bubbling of

the air exiting over the water seal is often heard distally on auscultation.

5.3 Palpation

Chest palpation can reveal important additional information. A common palpation technique in critically ill mechanically ventilated patients is to place the examiner's palm on the upper sternum (Fig. 5.17). When large enough in amount, tracheobronchial secretions cause bubbling vibrations that can be felt through the chest wall, particularly during expiration. Less frequently and mostly only in asthenic patients can the much finer vibrations of lung oedema be felt when the palm is placed over the lower lateral chest wall (Fig. 5.18). Palpation is also useful to evaluate the symmetry of chest wall expansions. This can be done by placing both hands around the chest or over the subcostal angle (Fig. 5.19). Tactile fremitus refers to vibrations felt by the examiner with both palms placed over the chest as the patient speaks (e.g. saying "99"). As non-aerated lung tissue transmits vibrations better than aerated lung parts, tactile fremitus is more pronounced over consolidated as compared to aerated lung fields. Tactile fremitus is absent over areas of pleural effusion or pneumothorax. This examination technique only detects large consolidations and is rarely applicable in critical care.

Palpation of subcutaneous air is a unique sensation and often generates a distinctive sound ("crepitus"). In severe forms of subcutaneous (or surgical) emphysema, this "crepitus" may lack as subcutaneous tissue layers are splinted too far apart. It is specific for the presence of subcutaneous emphysema which may accompany pulmonary barotrauma. Significant amounts of air have already collected in the subcutaneous tissues, once it can be palpated first. Subcutaneous emphysema can develop rapidly, occasionally even within only a few minutes. Development of subcutaneous emphysema in trauma patients (e.g. already at scene) or after surgery is often caused by a bronchial injury and significant air leak. Extensive subcutaneous emphysema is an obvious clinical diagnosis as air commonly distributes to areas of the body where there is soft subcutaneous tissue (e.g. the face and in particular the eyelids—Fig. 5.20). Pneumomediastinum can be felt by palpation of the jugulum and supraclavicular tissue. In clinical practice, it is most frequently encountered in postoperative cardiac or thoracic surgical patients. Usually, subcutaneous emphysema subsides within a few hours to days following pleural or mediastinal drainage. Persistence or progression must always be considered as a sign of an ongoing air leak.

Another feature that may be detected by chest wall palpation is a warmer skin over a pleural empyema. In patients with chest pain, palpation of the costal cartilages may reveal the costochondritis.



Fig. 5.17 Palpation for the presence of large amounts of tracheobronchial secretions. Courtesy of Martin W. Dünser, MD



Fig. 5.18 Palpation for the presence of alveolar lung oedema. Courtesy of Martin W. Dünser, MD

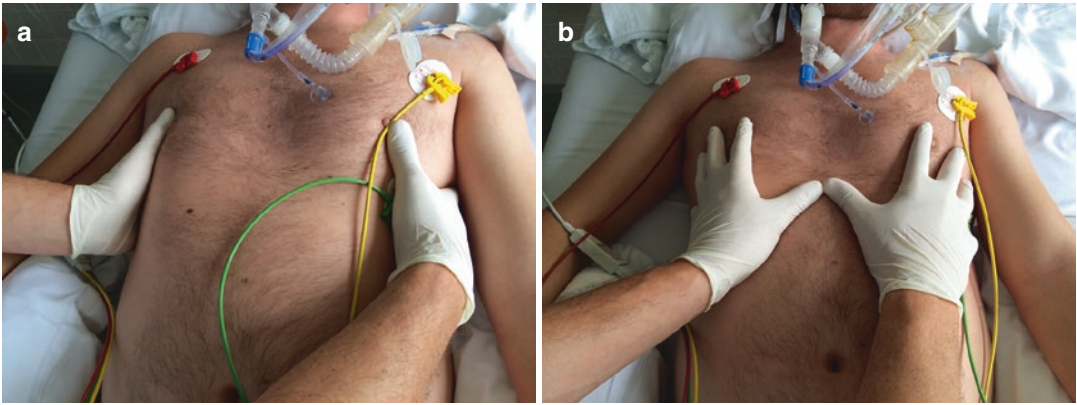


Fig. 5.19 Assessing the symmetry of chest wall expansions by palpation. Courtesy of Martin W. Dünser, MD. (a) Palpating both lateral chest walls with the examiner's hands; (b) placing the examiner's hands over the subcostal angle



Fig. 5.20 Typical features of subcutaneous/surgical emphysema in a patient with chest trauma and pulmonary barotrauma. Courtesy of Martin W. Dünser, MD



Fig. 5.21 Palpation of the position of the trachea. Courtesy of Martin W. Dünser, MD

dral junction or Tietze syndrome as an important differential diagnosis in patients with acute chest pain. Finally, palpation of the trachea and its position can assist in the recognition and assessment of severe barotrauma (Fig. 5.21). In patients with tension pneumothorax or large pleural effusion, the trachea can be deviated to the contralateral side; in patients with total lung collapse/volume loss/atelectasis, or fibrosis, to the ipsilateral side.

Palpation is one of the key techniques to examine the chest in patients sustaining severe trauma. In order to detect rib and sternal fractures, the chest wall is palpated for bony crepitus over both infraclavicular areas and the sternum (Fig. 5.22). Bimanual lateral compression of the

mid- and lower chest induces localized pain in awake patients with rib fractures (Fig. 5.23). In unconscious or sedated patients, fractures of single ribs are difficult to diagnose, particularly if fractures are posterior. In spontaneously breathing patients, flail chest wall segments are discerned by placing the examiner's hands over the lateral and anterior chest wall while feeling for paradoxically moving segments (Fig. 5.24). In patients who are mechanically ventilated, flail chest wall segments cannot be palpated since flail segments do not move paradoxically as pleural pressure remains positive over the respiratory cycle. In these patients, all areas of the chest wall need to be meticulously examined by digital compression. Flail segments are identified by mobility and inward movement on compression.



Fig. 5.22 Palpation of the sternum to detect sternal fracture and/or instability. Courtesy of Martin W. Dünser, MD



Fig. 5.23 Lateral chest compression to screen for rib fractures and/or a flail chest. Courtesy of Martin W. Dünser, MD

In case a mini-thoracotomy/thoracostomy is performed (Fig. 5.25), the lung can be palpated with the finger that enters the pleural cavity. The first information obtained is whether the lung is up or down (e.g. confirming the presence of pneumothorax). Although taking into account that only a very small area of the lung is palpated, the texture of the lung can give the examiner a rough idea of the lung injury. An uninjured or mildly traumatized lung feels tense like a balloon and quickly expands upon release of the pneumothorax. A severely contused lung feels slippery and has the consistency of a blood clot. Despite release of air from the pleural cavity, it does not expand at all or only incompletely and with a significant delay.

5.4 Percussion

Percussion of the chest is only a meaningful examination technique when it can be performed in an acceptably quiet setting. It requires experience to correctly interpret its findings. Percussion is performed by placing the (second or) third finger of the examiner on the anterior chest wall and firmly (=firmly!) tapping its distal segment with one finger of the other hand (Fig. 5.26). The sound produced by this can be classified as normal, hyperresonant or dull. A hyperresonant sound is higher in pitch, somehow “hollow” (tympanic) and can be heard over a pneumothorax, large bullae or in patients with COPD. It is extremely helpful to confirm hyperresonance by comparing it to the percussion sound of the contralateral hemithorax. Percussion over pleural effusions or large lung consolidations results in a dull and short resonance. A “stony dull” percussion note typically implies a pleural effusion. While percussion to test for pneumothorax or total lung atelectasis is performed over the anterior chest wall, the posterior or lateral chest wall (in the sitting position) is percussed in case a pleural effusion or lung consolidation is suspected. Percussion sounds in patients with lung emphysema and/or large anterior bullae are equally hyperresonant but less tympanic than in patients with a (large) pneumothorax.

5.5 The Physical Examination in Relation to Intubation and Extubation

5.5.1 Recognition of the Anatomically Difficult Airway: The LEMON Approach

There are multiple reasons why establishing a safe airway (anatomical, physiologic, process-related) can be (terrifyingly) difficult. The clinical examination is crucial to recognize the anatomically and physiologically difficult airway and thus induce appropriate subsequent preparations. No clinical sign alone is sensitive or specific enough though. A reliable prediction of an anatomically difficult



Fig. 5.24 Palpation techniques to detect a flail chest in the spontaneously breathing (a) and mechanically ventilated patient (b) Courtesy of Martin W. Dünser, MD



Fig. 5.25 Digital palpation of the lung in a patient with severe chest trauma during thoracotomy and placement of a chest drain. Courtesy of Martin W. Dünser, MD



Fig. 5.26 Percussion of the anterior chest wall to differentiate between a pneumothorax and (total) lung collapse. Courtesy of Martin W. Dünser, MD

airway can be achieved by using the LEMON approach. This mnemonic stands for Look, Evaluate, Mallampati, Obstruction and Neck mobility. Clinical signs to be looked for are summarized in Table 5.4. Evaluation includes the 3-3-2 examination technique (Table 5.5 and Fig. 5.27). Although the Mallampati score was first described in the sitting patient during preoperative evaluation and has not been validated in critically ill patients, it is worthwhile assessing. The patient is asked to protrude the tongue as far as possible while saying “aah”. The visibility of the soft palate and uvula is then inspected and graded into four classes (Fig. 5.28). While the chances for uncomplicated laryngoscopy and intubation are high in class one, the risk of a difficult

Table 5.4 Anatomical signs suggestive of a potentially difficult airway

Difficult mask ventilation	Difficult laryngoscopy and intubation
<ul style="list-style-type: none"> • Age > 55 years • Body mass index >26 • Lack of teeth • Presence of beard • History of snoring • Airway obstruction 	<ul style="list-style-type: none"> • Prominent upper incisors • Large tongue (Fig. 5.25) • Short and thick neck • Facial trauma or burn • Previous tracheostomy • Previous airway surgery/radiation • Upper airway obstruction (stridor!) • Oropharyngeal or neck masses • Pregnancy • Craniofacial syndromes

Table 5.5 The 3-3-2 examination technique

Examination step	Assessment of	Question	Interpretation if “no”
1	Mouth opening	Do THREE (patient-sized) fingers fit between the incisors?	Insertion of laryngoscope and laryngoscopy likely difficult
2	Volume of submandibular space	Do THREE (patient-sized) fingers fit between the mentum and hyoid bone?	Laryngoscopy likely difficult
3	Location of the larynx	Do TWO (patient-sized) fingers fit between the hyoid bone and thyroid cartilage?	Laryngoscopy and intubation likely difficult

Be aware that the 3-3-2 rule has been developed to predict difficult laryngoscopy and intubation with the use of a conventional laryngoscope. With the advent of video laryngoscopes, new challenges arose. While visualization of the vocal cords (laryngoscopy) became easier, intubation (passing the tube through the vocal cords) can remain a challenge (remember: visualization is not intubation, S. Seidl, 2015)

laryngoscopy is significant in classes three and four. Neck mobility must only be assessed in patients without cervical spine instability and thus not in all critically ill patients, particularly not those in the pre-hospital setting or emergency department. The simplest way to confirm adequate neck mobility for laryngoscopy is to passively move the patient’s head. Alternatively in awake patients, the patient can be asked to bend his head so that his/her chin touches the chest wall.

5.5.2 Clinical Indicators of Endotracheal Tube Position

The only reliable methods to confirm correct endotracheal tube position are direct visualization (bronchoscopy or direct laryngoscopy) and end-tidal carbon dioxide measurement. The clinical examination can only suggest correct and more importantly incorrect tube placement. It can be used in addition to or in the event that the aforementioned techniques are not available or have not yet been installed.

Bilateral chest wall expansion and/or auscultation of bilateral breath sounds in synchrony with mechanical ventilation usually indicates correct endotracheal tube position in the non-breathing patient but is unreliable if the patient maintains spontaneous breathing over the intubation process. In the latter patient, expiratory air-flow can be felt at the end of the tube to confirm endotracheal tube position. A fast screening method involves firmly placing the examiner’s fingers into one anterior intercostal space of the

left hemithorax (Fig. 5.29). Delivery of a firm breath with a ventilation bag results in synchronous lung expansion which can be felt with the finger tips (yellow arrow).

If the endotracheal tube is advanced too far, its tip usually enters the right main stem bronchus resulting in hypoventilation of the left lung. It is difficult to determine the endobronchial position of the tip of the tube by clinical examination alone. As a small air leak between the inflated balloon and the tracheal bifurcation often exists despite of the tube’s tip being positioned in the right bronchus, diminished breath sounds can often be heard over the contralateral (mostly left) lung on auscultation. Only in rare instances, when the tube is advanced far enough for the balloon to obliterate the entire right or left main stem bronchus, are contralateral breath sounds absent. In addition to observation of chest movements and bilateral auscultation, verifying the insertion depth of the tube as 20–21 cm in females and 22–23 cm in males rendered the highest sensitivity and specificity to detect endobronchial intubation [2].

Importantly, the clinical examination can help to recognize oesophageal tube misplacement. Unless spontaneous ventilation is maintained during intubation, absence of chest movements and development or worsening of cyanosis despite ventilation must primarily be considered a sign of oesophageal tube misplacement. While delivery of breaths to the oesophagus with a ventilation bag can feel similar to delivery of breaths to the lungs, air is not expired or only at a much slower rate from the stomach/oesophagus than



Fig. 5.27 Examination steps of the 3-3-2 examination technique to recognize the anatomically difficult airway. Courtesy of Sirak Petros, MD. See Table 5.5 for explanations



Fig. 5.28 Mallampati classification



Fig. 5.29 Rapid screening technique to assess endotracheal tube placement at the first breath with the ventilation bag (see yellow arrow and text for explanation). Courtesy of Martin W. Dünser, MD

from the lungs. This is then typically associated with gurgling sounds. Entrance of gastric juice into the tube (be sure not to mistake it for lung oedema!) during expiration is another sign that is highly suggestive of oesophageal intubation (unless tracheal aspiration of gastric content has

occurred before). Expiratory misting of the tube is usually absent in oesophageal intubation but has anecdotally been observed after large amounts of gastric air exit through the tube during release of positive pressure.

5.5.3 Assessing Preparedness for Extubation

Certain absolute and relative criteria need to be present so that a patient can be extubated safely. The absolute criteria include sufficient spontaneous gas exchange, the presence of upper airway reflexes and adequate cough strength. Although several scores and cut-off values have been suggested to predict successful extubation, it is clinically difficult to apply single values to all patients. While it is fairly easy to assess adequate oxygenation with the use of arterial oxygen saturation and/or blood gas analysis on reasonable ventilator settings ($\text{FiO}_2 < 0.45(-0.5)$, $\text{PEEP} < 7 \text{ cm H}_2\text{O}$), evaluation of adequate spontaneous ventilation primarily relies on clinical skills. In clinical practice, this is achieved by close observation of the patient for signs of respiratory distress, an increased work of breathing or a pathologic breathing pattern either during a spontaneous breathing trial or on an augmented spontaneous ventilation mode. It is advisable to take at least 1 min or longer to observe the patient, particularly those who have been ventilated for several days. Although a respiratory rate < 25 breaths per minute on reasonable ventilatory support should be present in the majority of patients, some individuals can be extubated successfully at higher respiratory rates. These subjects are typically alert and have a poor tube tolerance or an underlying restrictive lung process necessitating higher respiratory rates. Finally, the patient's own estimation whether he or she can breathe without the tube often helps to predict whether the extubation will be successful or not.

The presence of reflexes to maintain upper airway patency and clear secretions from the airways is another prerequisite for safe extubation. The awake patient should be able to show his or her tongue. Indicators of an inadequate swallow-

ing reflex in the non-responding patient are the presence of a saliva pool in the mouth, the frequent need for oral suctioning and/or saliva drooling from the mouth. Important to note is that these clinical methods only assess whether upper airway control is present but cannot be used to predict post-extubation dysphagia (see Part II Sect. 5.5.4). Spontaneous coughing is a positive predictor of extubation success. The cough force can be tested by disconnecting the patient from the ventilator and asking him or her to cough forcefully (Fig. 5.30). Alternatively, coughing can be assessed by endotracheal suctioning. If the patient does not cough or coughs only with little force, clearance of secretions following extubation is likely to be insufficient. Finally, the frequency at which endotracheal suctioning is required needs to be taken into account.

Although it is preferable to have the patient alert and cooperative at extubation, this is—against common belief—no absolute criterion for extubation. In some patients with a rapidly reversible neurological condition (e.g. mild or moderate brain trauma, intoxication, delirium) and well-maintained upper airway reflexes, prolonged sedation and ventilation may carry higher risks than early extubation. However, this must be individually evaluated and a risk-benefit assessment considered.

While “overhang” of opioids or sedatives is indicated by a depressed mental state or a distinct

breathing pattern (opioids: delayed onset of spontaneous breathing followed by bradypnea with high tidal volumes), it is more difficult to recognize residual neuromuscular blockade. Recognition of the latter is essential as it commonly results in immediate respiratory decompensation after extubation or impaired airway control with an increased risk of delayed tracheal (micro)aspiration and pneumonia. The population at highest risk for residual neuromuscular blockade is critically ill patients after surgery. Important determinants are timing, type and dosage of the neuromuscular blockade agent administered. Hepatic and renal dysfunction can delicately impair the clearance of neuromuscular blocking agents and lead to a prolonged time to full recovery of neuromuscular capacity. Similarly, obesity is a clinical risk factor as patients typically receive higher doses although their muscle mass is only slightly increased. In addition, obese patients are specifically sensitive to any degree of residual neuromuscular blockade. Before extubation, an in-depth physical evaluation alone is not sensitive enough to detect mild degrees of residual neuromuscular blockade. This can only be achieved by quantitative neuromuscular monitoring (e.g. the train-of-four testing). When assessing the patient’s muscular force, it needs to be taken into account that the diaphragm and pharyngeal musculature are exclusively sensitive to neuromuscular blockade and recover last. Therefore, recovery of full peripheral muscle strength (e.g. strong hand squeeze or ability to move extremities particularly in response to external stimuli such as suctioning) must not be used to exclude residual neuromuscular blockade. Similarly, an adequate tidal volume is a highly unreliable marker of full neuromuscular recovery. The best clinical method appears to be the ability to lift the head off the cushion or pillow for at least 5 s. If the patient is extubated despite (unrecognized) residual neuromuscular blockade, a typical clinical picture is seen. Patients immediately obstruct their airways or take shallow breaths which can only be elicited by twitchy movements of the accessory respiratory muscles. This often leads to synchronous small forward movements of the head and



Fig. 5.30 Testing for cough force in a patient being evaluated for preparedness for extubation. Courtesy of Martin W. Dünser, MD

chin. Patients are awake, typically frightened and unable to speak.

If upper airway obstruction is considered a risk, the cuff leak test can be performed with the patient still intubated. Given the high odds of false-negative results and the associated risk of unnecessarily prolonging mechanical ventilation, the cuff leak test should only be performed in a highly selected group of critically ill patients. During positive pressure ventilation, the cuff of the endotracheal tube is deflated and the examiner listens for gurgling sounds of air exiting next to the tube. It is important to make sure that the patient's head is placed in a neutral position as otherwise the position of the head can obstruct the upper airway, particularly in obese patients. An air leak should be audible at peak inspiratory pressures of 20 mbar or lower. The absence of an audible air leak during a correctly performed cuff leak test is a sensible indicator of upper airway compromise or obstruction. If an air leak can only be heard when the patient coughs (which is frequent after deflating the cuff) but not during assisted breathing, the cuff leak test must be considered negative. It should be noted, however, that if an air leak is present, it cannot be safely assumed that the upper airway will be uncompromised after extubation. Performance of the tube occlusion test can increase the predictive value of a positive cuff leak test. During this test, the tube is occluded while the balloon of the tube is still deflated. The patient is then asked to take a deep breath. If inspiration results in visible chest wall expansion, the risk that the upper airways are unobstructed is minimal.

5.5.4 Screening for (Post-extubation) Dysphagia

Oropharyngeal dysphagia is frequent in critically ill patients, particularly after long-term (>48 h) intubation, in the elderly or those with a neurological disease (e.g. stroke) or neuromuscular disease. It is an important contributor to morbidity (hospital-acquired pneumonia) and mortality in these patients. Early recognition of dysphagia is essential. The clinical examination plays a crucial role as a screening tool to trigger definite diagnostic procedures typically including fiberoptic

endoscopic evaluation of swallowing or barium swallow. Inability of the patient to swallow resulting in retained oral saliva and secretions is a straightforward diagnosis. However, in the majority of patients with swallowing disorders, symptoms are more subtle. The water swallow test is a valid method with a moderate sensitivity but an acceptable specificity to diagnose dysphagia. The alert patient is placed in the sitting position and offered one or more sips (<20–30 mL) of water to drink. The swallowing process is closely observed for obvious dysfunction (e.g. multiple swallow attempts, water drooling from mouth, non-elevation of larynx) and the patient monitored for coughing, choking, change in voice or throat clearing. Any of these signs strongly suggests the presence of dysphagia and should lead to definite diagnostic tests. In patients with a tracheostomy, the water-swallowing test can be modified. The balloon of the tracheal tube is deflated and the patient offered coloured (e.g. with methylene blue) water to drink. The same observation as described with the standard water-swallowing test is then performed. At the end of the test, the patient is suctioned endotracheally and secretions inspected for the presence of coloured secretions. Sometimes the coloured water also exits from the tracheostoma site and confirms the diagnosis dysphagia.

5.6 Clinical Evaluation of the ECMO Circuit

Inspection and palpation are important techniques to monitor the extracorporeal membrane oxygenation (ECMO) circuit. A detailed daily inspection of all circuit components for integrity and adequate function is obligatory. Similarly, dressings and wounds must be inspected for signs of oozing or bleeding as indicators of (acquired) coagulopathy. Relevant haemolysis can rapidly be recognized at the bedside by checking whether the urine or haemofiltrate are rose coloured. Even though the ECMO circuit may be looked after by a dedicated perfusionist, the (intensive care) physician must be able to perform this examination, too.

Irrespective of the mode of ECMO (veno-venous or veno-arterial), the colour of the blood

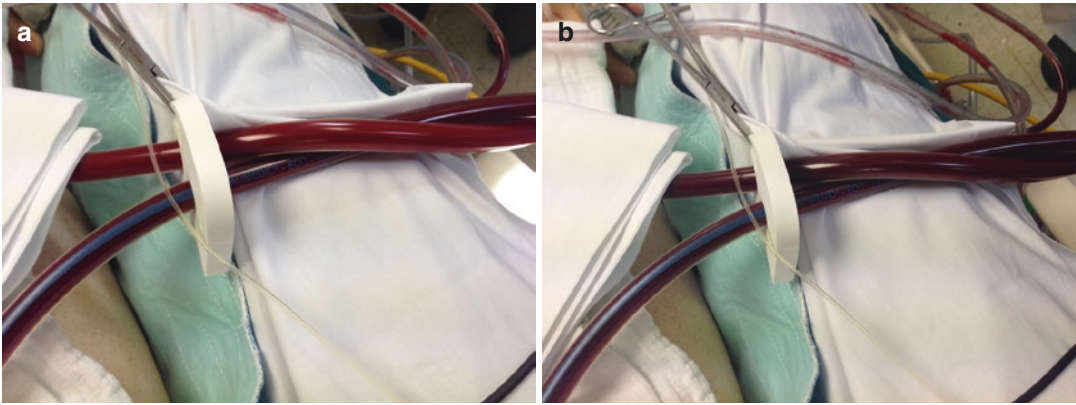


Fig. 5.31 Inspection of the blood's colour before and after the oxygenator of an ECMO circuit. Courtesy of Martin W. Dünser, MD. (a) Normal: deoxygenated/venous blood before the oxygenator and bright red blood

after the oxygenator. (b) Danger: deoxygenated/venous blood before and after the oxygenator indicating loss of oxygenator function or oxygen supply



Fig. 5.32 Inspection of the oxygenator for the presence of thrombi. Courtesy of Martin W. Dünser, MD

after the oxygenator must be bright red. Visual comparison to the pre-oxygenator blood's colour is helpful (Fig. 5.31). Reasons for an acute change in colour of the post-oxygenator blood are usually accidental changes in the ECMO FO_2 or failure of the oxygen source. A deterioration in oxygenator function is, however, usually detected by a drop in post-oxygenator PO_2 before any visual changes in blood colour gradually occur. The outer surface of the oxygenator should be inspected with a light source (Fig. 5.32). Small, usually dark-coloured fibrin strands or thrombi can be observed and, together with a reduced post-oxygenator PO_2 , indicate oxygenator dysfunction due to clotting.

If the pump revolutions are set too high, venous filling is too low or the draining cannula

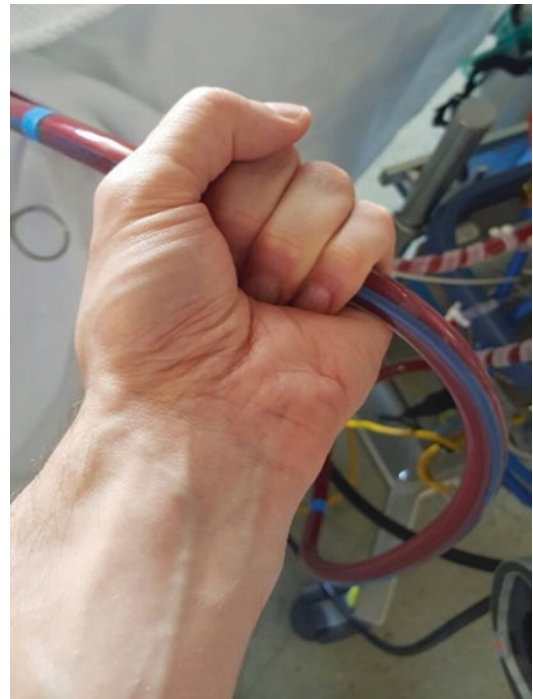


Fig. 5.33 Palpating for vibrations of the venous tube indicating impaired drainage of venous blood. Courtesy of Daniel Dankl, MD

malpositioned, stroking of the venous tube occurs, often before a drop in blood flow takes place. Before stroking or shaking of the venous tube can be seen, vibrations of the tube can be felt with the hand (Fig. 5.33). Air bubbles (or in severe cases even foam) are a common cause for

an acute halt of the circuit pump and need to be urgently recognized by visual inspection of the circuit.

Clinical Practices

Box 1 Schamroth Sign

Putting the dorsal aspects of the distal segments of the finger and nails together should normally leave a diamond-shaped aperture at the nail bed. The absence of this aperture is considered a positive Schamroth sign and indicates finger clubbing.

Box 2 Definitive Features of Clubbing

Five components of clubbing are recognized:

- Increased nail bed fluctuation
- Loss of the nail bed angle (normally <math><140\text{--}160^\circ</math>) (Lovibond angle)
- Increased curvature of the long axis of the nail
- Soft tissue swelling at the ends of the digit (which when marked produces a drumstick appearance)
- Hypertrophic pulmonary osteoarthropathy may develop and is recognized by painful wrists and periosteal elevation demonstrated radiologically

Box 3 Clinical Signs Indicative of a Severe Asthma Attack

- Increased work of breathing (see Table 5.1)
- Altered mental state (incl. agitation)

- Diaphoresis
- Decreasing volume of breath sounds and by that also of the expiratory wheeze
- Paradoxical pulse
- Patient assumes the tripod position (patient leaning forward with both arms stretched and stemmed against the thighs)
- Severe tachycardia (adults >120–130 bpm)

Box 4 Differences Between Two COPD Subtypes

	Emphysema subtype“pink puffer”	Bronchitis subtype“blue bloater”
Comorbidities		
Body mass index	Low	High
Cardiovascular disease	+	+++
Metabolic disease	+	+++
Obstructive sleep apnoea	–	+++
Pulmonary cachexia	+++	–
Lung function		
Emphysema	+++	+
CO diffusion capacity	Low	Normal or mildly reduced
Dyspnoea	+++	+
Exercise capacity	Severely reduced	Mildly/moderately reduced
Exacerbation management		
Risk of hyperinflation	+++	+
Risk of post-intubation cardiovascular collapse	+++	+
Complications of MV	+++	++

CO carbon monoxide, MV mechanical ventilation

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