The role of conventional Chest Radiography in the diagnosis of Acute Respiratory Failure in the Emergency Department

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Learning objectives

The aim of this work is to analyze the radiologic signs and the correct use of conventional Chest Radiography in the main conditions causing cardiac and pulmonary dyspnoea, as acute exacerbation of chronic obstructive pulmonary disease, acute pulmonary oedema, acute pulmonary trombo-embolism, pneumothorax, pleural effusion, and to focus indications and limits of this diagnostic tool.

Background

Dyspnoea is defined as an uncomfortable awareness of breathing.

NYHA (New York Heart Association) classified dyspnoea in 4 classes, according to the functional decrease of performance status in patients:

- class I dyspnoea appears after moderate physical effort
- class II dyspnoea appears during normal activities
- class III dyspnoea appears for lower physical efforts
- class IV dyspnoea is always present [1]

Causes of dyspnoea are various and may involve mainly cardiovascular and respiratory apparatus.

Dyspnoea, together with thoracic pain, are two of the most frequent symptoms of presentation of thoracic diseases in the Emergency Department (ED).

In the emergency setting, thoracic imaging by standard chest X-ray (CXR) plays a crucial role in the diagnostic process, because of its fast and cheap execution.

Although radiologists are responsible for the final interpretation of studies, many CXRs are first viewed by non-radiologists. All physicians should be able to quickly and accurately identify a wide number of critical findings to help identify patients who need subsequent emergency care.

The emergency physician should be aware that the sensitivity of CXR is rather low in the diagnosis of several causes of dyspnoea, such as pneumothorax, emothorax and pulmonary edema [2], particularly in bedside-obtained images. It has been shown a high inter-observer variability of reading that limits the diagnostic usefulness of bedside CXR and complicates the differential diagnosis.
ACUTE EXACERBATION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Chronic obstructive pulmonary disease (COPD) is a syndrome characterized by a progressive limitation of the air flow, poorly reversible and associated with an inflammatory response of airway epithelium. In this definition we can find both chronic bronchitis and emphysema.

Most exacerbations are due to infections of the upper airways [3]. In the most severe cases, it is common to observe co-morbidity with congestive heart failure, extrapulmonary infections or pulmonary embolism.

In patients with COPD, diagnosis of exacerbation is possible by evaluating clinical history, symptoms and physical signs, even if instrumental examination is crucial for confirmation and assessment of the severity. Chest x-ray is often used to detect some of the major causes of exacerbation and to rule-out associated conditions, mainly pneumonia and pulmonary congestion [4].

However, airway infections causing clinical worsening are very often not imaged in the CXR. During exacerbations, CXR demonstrates abnormal images only in 16% of cases, mainly as inflammatory infiltrates or signs of pulmonary congestion [5, 6].

For these reasons it is not recommended as a routine exam, but only in cases of highly suspected pneumonia, which could be decisive to decide for hospitalization, or to recognize other causes of dyspnoea as acute pulmonary oedema, massive pleural effusion, atelectasis or pneumothorax.

Usefulness of CXR in the diagnostic procedure of exacerbation of COPD has some important limitations, due mainly to the high inter-observer variability of reading. In case of pneumonia (figure 1), clinicians and radiologists agree on the diagnoses only in about 50% of cases [7].
Fig.: Fig-1. Posterior-anterior CXR in an emphysematous patient. It is possible to observe multiple bronco-pneumonic bilateral outbreaks, confluent in the right region. Left lateral costo-phrenic sinus is totally filled by pleural effusion.

References: E. M. Laugelli; Dipartimento di Scienze Cliniche e Biologiche, University of Torino, Orbassano (TO), ITALY

Variability exists also when CXR readings are performed by radiologists with different experience. Associated emphysema further limits the reading, because infiltrates and oedema may appear as atypical radiological patterns, with aspect similar to cavity due to hyperinflation zones and long-lasting course [3]. In these cases, a common finding could be the infection of an emphysematous bulla, with a hydro-aerial level, which usually is better analyzed using a CT scan with contrast-enhancement or through high resolution CT protocols [8].

ACUTE PULMONARY OEDEMA
Acute pulmonary oedema (APE) is a condition of increased fluid content in lung, at the expense of its content of air. It is classified into two main groups, depending on different mechanisms: cardiogenic APE, due to increased hydrostatic pressure in pulmonary capillaries during congestive heart failure and fluids excess; non cardiogenic or lesional APE, due to increased capillary permeability during acute respiratory distress syndrome (ARDS).

Differential diagnosis between cardiogenic and lesional oedema often is not very easy.

A clear correlation can be demonstrated between clinical-radiologic findings and pathogenesis.

Standard CXR represents the first line imaging exam in a patients presenting to the ED complaining of acute dyspnoea. The role of CXR is not only linked to the first diagnosis of APE, but also in the differentiation between cardiogenic and non-cardiogenic causes [9] and to manage treatment.

To these purposes, the radiologic signs and findings to be studied are:

- perfusion pattern and spatial distribution of the oedema,
- size of the vascular peduncle,
- cardiac volume,
- lung volume.

Moreover, it is crucial recognition of some specific signs like lung interstitial oedema, pleural effusion and air bronchogram.

The perfusion pattern of a normal standing individual observed by CXR shows a rate of distribution between basal and apical regions of the lung greater than 2. Values less than 1 represent the so called "redistribution pattern" of pulmonary blood flow, that is usually demonstrable in venous pulmonary hypertension. It is due to the rapid formation of oedematous infiltrates around vascular structures in declivous zones of the lungs. This phenomenon leads to a general vasoconstriction with increase in resistance of blood flow, which is consequently redistributed to the apical regions of the lungs.

The parenchymal opacities in APE may have variable spatial distribution, central, peripheral or declivous, and can present both with or without air bronchogram, accordingly with different ethiologies.

The width of the vascular peduncle is intended as the distance from the superior cava vein at right upper main bronchus crossing, to the vertical line tangent to the external profile of the origin of the left subclavian artery. The normal size of the vascular peduncle is 48 ± 5 mm while standing during forced inspiration, with posterior-anterior incidence of the X-ray beam. It is highly correlated to blood volume and it is increased during heart failure, high output syndromes, fluid overload and in case of hydrosaline retention.
Interstitial oedema is marked by the presence of reduced parenchymal diaphaneity, blurred vascular profiles, Kerley lines and pleural effusion.

However, all these signs are not decisive for the differential diagnosis between numerous diseases and particularly in the differentiation between cardiogenic and non cardiogenic APE. Instead, the air bronchogram and the signs of peripheral distribution of interstitial oedema are highly suggestive of lesional oedema.

Facing acute decompensated heart failure with consequent cardiogenic APE, four stages of altered pulmonary circulation can be described [3].

Initially there is a transient phase with few radiologic signs and circulation uniformly distributed.

A second stage follows with a slight increase of pulmonary capillary pressure (17-20 mmHg), visualized as redistribution of the pulmonary circulation when imaged by CXR performed in the upright position. During this phase, the supine CXR still fails to show redistribution because of lack of the gravity gradient between apical and basal lung areas. The hilar structures become prominent and show external convexity (figure 2).
Fig.: Fig-2. Posterior-anterior CXR demonstrating cardiomegaly, more pronounced at left chambers, with redistribution of lung circulation from bases to apex in a patient with acute decompensated heart failure.

References: E. M. Laugelli; Dipartimento di Scienze Cliniche e Biologiche, University of Torino, Orbassano (TO), ITALY

The third stage is secondary to a further increase of the pulmonary venous pressure over the oncotic plasmatic pressure (≥ 20 mmHg). At this time radiologic signs of interstitial oedema appear (figure 3), characterized by decreased parenchymal diaphaneity, blurred vascular profiles, Kerley lines, opacities due to sub-pleural oedema and pleural effusion. The Kerley lines are typical signs of interstitial involvement, but are not highly specific of interstitial oedema. They are usually distinguished in type A, B and C, depending on distribution and appearance. Pleural effusion, often presenting at this stage, can be meniscal, laminar, intrapulmonary, fissural, mono- or bilateral (more often on the right if mono-lateral).
**Fig.**: Fig-3. Posterior-anterior CXR in a patient with congestive heart failure and interstitial pulmonary edema. Note the large heart shadow, the thickening of the pulmonary perihilar shape, the pleural bilateral opacity due to effusion and the B Kerley’s lines.

**References**: E. M. Laugelli; Dipartimento di Scienze Cliniche e Biologiche, University of Torino, Orbassano (TO), ITALY

In the last or fourth stage, when pulmonary venous pressure increases over 30 mmHg, alveolar oedema may develop (figure 4). Very often heart size and cardio-thoracic ratio are markedly increased, but this sign is not highly accurate.
Indeed, cardiac enlargement at CXR can be lacking in decompensated heart failure with pulmonary congestion but normal systolic function (diastolic dysfunction) or acute myocardial illnesses, both ischemic and infectious. On the other hand, cardiac enlargement does not necessarily mean decompensation of a pre-existing chronic heart failure. Moreover, the bedside CXR performed in supine position, as commonly applied to critically ill patients, has indubitable low sensitivity in evaluating the real size of the heart [11].

**Fig.** Fig-4. Supine radiogram in a patient with cardiogenic alveolar edema. Note that the vascular perihilar structures are not defined because of the presence of
consolidation shadows, not well defined and confluent in peripheral territories. Cardiomegaly is not present.

**References:** E. M. Laugelli; Dipartimento di Scienze Cliniche e Biologiche, University of Torino, Orbassano (TO), ITALY

The oedema distribution often is declivous, bilateral and symmetric, but it can vary according to the decubitus: in supine position the lower regions are the posterior areas of the lung, without distinction between pulmonary apex and base, while in lateral decubitus oedema tends to distribute as unilateral manifestation.

Moreover, other pre-existing pulmonary pathologic conditions can modify the oedema distribution, giving asymmetric, unilateral, reticular, micro-nodular, or other atypical aspects, simulating bronco-pneumonia foci [12], limiting the diagnostic usefulness of the topographic criteria and complicating the differential diagnosis.

Distribution of oedema depends also from aetiology. For instance, in APE due to fluid overload, both spontaneous or iatrogenic, the perfusion rate between apex and base is almost 1, the oedema is central and the size of vascular peduncle is increased.

Moreover, dilation of superior vena cava and azygos together with an increase in thickness of lateral-thoracic chest wall due to fluid storage, can be seen. Interstitial oedema, pleural effusion and often enlargement of heart and pulmonary volumes can coexist.

In APE due to impaired capillary permeability, vascularization at bases and apex is mostly normal, and cardiac and vascular peduncle size appear of regular size. The consolidation areas are characteristically disposed to periphery and often associated with air bronchogram (figure 5 and figure 6).
Fig.: Fig-5. Supine CXR showing typical peripheral alveolar consolidation areas, prevalent in the basal zones, in a case of ARDS, but very similar to cardiogenic oedema.

References: E. M. Laugelli; Dipartimento di Scienze Cliniche e Biologiche, University of Torino, Orbassano (TO), ITALY
Fig.: Fig-6. High Resolution CT slice of the same patient of figure 5. Note the consolidation and "ground glass" areas with gravitational distribution, with air bronchogram. Air bronchogram is more consistent with a diagnosis of ARDS than cardiogenic edema.

References: E. M. Laugelli; Dipartimento di Scienze Cliniche e Biologiche, University of Torino, Orbassano (TO), ITALY

These signs represent very useful differential radiographic criteria and, if independently evaluated in a standardized diagnostic system, can lead to a correct diagnostic differentiation between cardiogenic and non-cardiogenic APE in over 90% of cases [9]. However, they can be unreliable in patients in supine position, if oedema is so large to
compromise a correct evaluation of cardiac and pulmonary vascular shadows, condition quite usual in critically ill patients evaluated by bedside CXR.

Chest x-ray usefulness in the first diagnosis and monitoring of patients with APE of whatever aetiology is well recognised and accepted. However, its accuracy is still debated; some authors showed great limitations in its sensitivity, suggesting that CXR cannot show increase of extra-vascular fluids inferior to 30%[13]. It should be stressed that diagnostic accuracy of CXR is often unreliable due to the emergency setting and forced bedside application in acute respiratory failure and critically ill patients. The posterior-anterior view obtained in the upright position, possibly completing the examination by a lateral view, considerably enhances sensitivity of CXR in the evaluation of pulmonary congestion.

ACUTE PULMONARY TROMBO-EMBOLISM

Acute pulmonary thrombo-embolism (APT) is secondary to sudden interruption or significant reduction of blood supply to the lung due to pulmonary circulation obstruction.

This pathologic condition is quite frequent and sometimes constitutes a cardio-respiratory emergency, leading to death in 30% of untreated cases [16, 17]. To date, APT is considered the third leading cause of death in western countries and the most misdiagnosed pathologic condition, being correctly detected only in 20% of cases [18].

Physical signs as well as routine diagnostic tests are not enough accurate to allow for final diagnosis, but only useful to hypothesize APT in the emergency setting and to define the pre-test probability according to the criteria published by Wells and co-authors [19].

CXR has a limited role in the diagnostic process of APT, primarily related to the exclusion of other common causes of respiratory failure and chest pain. Quite often, CXR is completely normal in APT.

Instead, spiral angio-CT (SCT) scan has a well defined role and it is the first level radiographic test when a clinical suspicion has been hypothesized [21, 22]. SCT has an high sensitivity (87% against 33% of CXR) and specificity (95% against 59% of CXR), and indubitable advantages due to its fast execution, broad view and objective interpretation, as well as its ability to allow for differential diagnosis in the event that the initial clinical suspicion is not confirmed [10].

Limitations of CXR are related to the lack of specific signs.

Some radiologic findings have been corroborated in many years of experience that are related to observations of CXR examinations in patients with APT, but rarely such signs are found altogether even in case of clear clinic presentations [23].
Nevertheless, many authors suggest that a careful observation of CXR images can show some specific abnormality in at least 90% of the cases [24-26].

The possible findings of standard CXR in APT are the following [18, 27]:

- **Pulmonary infiltrates**, due to haemorrhagic or oedematous infiltration of secondary lobules, often multiple, more often located to the right base, sometimes associated with atelectasis line or pleural effusion.
- **Atelectasis**, often sub-segmental, appearing as curved lines reaching the pleura, secondary to alveolar collapse (line of Fleishner), ought to bronchial mucosa congestion, alveolar collapse secondary to surfactant reduction and hypoventilation due to reduced diaphragmatic excursion.
- **Diaphragm elevation** secondary not only to reduction of pulmonary volume, but mainly to the dysventilation consequence of an antalgic respiration during pleural pain.
- **Pleural effusion**, mainly serous, bilateral and of slight entity, often in association with basal atelectasis.
- **Westermark sign**, uncommon but highly specific, corresponds to a region of impaired vascularisation in the lung region distally to the site of the embolism [28]. For a safe interpretation of this sign, it should be demonstrated the absence of it in an old radiogram to be used for comparison. Another limitation of this sign is linked to the difficulty in visualization when CXR is performed in the supine patient.
- **Right heart and azygos vein enlargement** are signs of severe pulmonary hypertension and right heart failure. They are invariably associated with symmetric enlargement of the ilar regions and other signs previously described. As for the Westermark, visualization of these signs should always be compared with previous images and they are unreliable when examination is performed in the recumbent position.
- **Hampton’s hump** is a triangular opacity with its apex pointing the hilar region, sometimes with blurred margins and irregular shape. It is a sign of interruption of blood supply from the systemic circulation in the lung region previously excluded by embolic obstruction of the functional circulation. Often, the differential diagnosis with an alveolar consolidation due to pneumonia is difficult.

Despite the numerous signs listed, the most useful and accurate radiologic finding is the normal appearance of CXR in the face of patients presenting with acute dyspnoea or thoracic pain. This observation has the value of excluding from the differential other conditions potentially causing acute respiratory failure and chest pain [18].

**PNEUMOTHORAX**
Pneumothorax (PNX) is defined as the presence of air in the pleural cavity, which comes from the break of the visceral or parietal pleural layers [29]. The main effect of this phenomenon is the collapse of the lung. The extent of the air layer affects the severity of the clinical picture. Moreover, clinical consequences are strictly connected with the pre-existing condition of the patient.

Standard CXR, acquired in orthostatic position, is the elective exam for diagnosis. Sign used for the diagnosis is better visible using a forced-expiration acquisition (figure 7).

**Fig.** Fig-7. Inspiration and expiration CXR in a case of right sided spontaneous pneumothorax. Note that the extension of pneumothorax is larger during expiration than inspiration and the expansion of the affected hemi-thorax is more evident in the affected side.

**References:** E. M. Laugelli; Dipartimento di Scienze Cliniche e Biologiche, University of Torino, Orbassano (TO), ITALY

The visceral pleura is visualized as a thin line, with no bronco-vascular texture beyond it. Although highly specific, the radiologic performance targeted to detection of this sign has an incredibly low sensitivity.

A large number of PNX (probably more than 30%) are not diagnosed by conventional CXR, particularly when expiration and orthostatic radiograms cannot be obtained [30].

When supine patient imaging is evaluated, diagnosis is more difficult because there is the possibility to misdiagnose even severe PNX because air move up and medially between lung and heart. Only after filling these spaces, free air can gather the usual apical-lateral position (only 20% of cases in supine CXR) [3].
When a CXR is not acquired in an orthostatic posterior-anterior view, there are some other signs that can be important for diagnosing PNX. These are the emphasized transparency of ipocondrium, the *deep sulcus sign* [31], the appearance of sharp edges of mediastinum, heart and subcutaneous tissues, or the visibility of the anterior-inferior edge of the lung [32]. Anyway, these signs are pathognomonic but not constant.

When possible, in doubtful cases acquisition of a radiogram in the lateral view (Hessen position) or during a forced expiration, can be useful [10, 12]. In these cases, it is sometimes possible to demonstrate even the thinner layer of PNX.

Free air can also gather in a fissure or behind the triangular ligament, or it can distribute around an atelectasis or a consolidate lobe, sometimes with unusual aspects against the expected gravity distribution. This is due to variations of intra-pleural pressure in presence of various chronic pulmonary diseases (figure 8).

**Fig.**: Fig-8. CXR of a patient affected by fibrothorax consequence of tuberculosis. Note a limited layer of pneumothorax visible in the left lateral inferior lobe.
In these cases CXR differential diagnosis between pneumothorax, pneumopericarium and pneumonediastinum can be very difficult.

The main radiologic signs of tension PNX are the lateral shift of heart and mediastinum, the lowering of the hemi-diaphragm, the flattening of the cardiac profile, the reduced size of the superior vena cava and the protrusion of the parietal pleural layer between the intercostal spaces.

The underused thoracic sonography has been widely showed to be of great usefulness in the emergency diagnosis of PNX and even in the detection of radio-occult PNX, being far more accurate than CXR and equivalent to CT scan [33].

**PLEURAL EFFUSION**

Pleural effusion is defined as the presence of liquid in excess inside the pleural cavity. A thin fluid film is regularly present between the two pleural layers, thus facilitating respiratory sliding.

A minimal amount of pleural fluid can be detected in 10% of healthy subjects, and it is physiologically increased after laparatomy or in post-partum [34, 35].

Numerous different conditions can cause pleural effusion, as cardiovascular diseases, hyper-expansion of body fluids due to renal and hepatic failure, infections, autoimmune diseases, cancer and traumas.

CXR is the first line diagnostic tool to be used in the diagnosis an quantification of pleural effusion. Orthostatic standard CXR in two views is able to detect even a minimum amount of pleural effusion (about 25 ml), which are usually visualized at lateral view only in the posterior costophrenic angle. When some fluid is visualized also in the lateral costophrenic angle at the posterior-anterior view, it is possible to calculate a total amount of about 100 ml (figure 9 A,B).
**Fig.**: Fig-9. Posterior-anterior (A) and lateral (B) views at CXR of a patient with massive left pleural effusion. Note the typical Damoiseau-Ellis line.

**References:** E. M. Laugelli; Dipartimento di Scienze Cliniche e Biologiche, University of Torino, Orbassano (TO), ITALY
Fig.: Fig-9. Posterior-anterior (A) and lateral (B) views at CXR of a patient with massive left pleural effusion. Note the typical Damoiseau-Ellis line.

References: E. M. Laugelli; Dipartimento di Scienze Cliniche e Biologiche, University of Torino, Orbassano (TO), ITALY

Anyway, severity, lung and chest wall compliance, capillarity of the pleural layers and the physical features of the fluid, condition the distribution in the pleural cavity.

Classical radiologic signs are consistent with basal opacity and horizontal air-fluid interface, with flattening of the diaphragmatic dome. In case of massive effusion, all the hemi-thorax can be filled and mediastinum can be shifted contralaterally.

If CXR is acquired at bedside in the anterior-posterior view, it is extremely easy to underestimate the real amount of the free effusion [15]. Moreover, from 10% to 25% of the milder forms of effusion can be completely misdiagnosed by bedside CXR [3].

Some radiologic signs allows diagnosis of pleural effusion at CXR, even if the classical visualization of the basal opacity is lacking. They are the thickening of fissures and
of pleural line at the apex, the blurring of the diaphragmatic profile and the haze of costophrenic angle, the complete but slight haze of the hemi-thorax with still visible vascular tree.

In a supine patient, one of the more declivous part of the thorax are the apical posterior zones, so in this place can accumulates large amount of pleural effusion for gravity. These signs are useful when comparison between the two hemi-thorax is possible, while in case of massive effusion equally distributed on both sides, they are extremely difficult to be recognized.

A negative supine bedside CXR cannot accurately rule-out even large amount of effusion. In these cases a lateral view with 20° of Trendelemburg inclination (the Hessen view) can obviate to lack of accuracy [34, 36]. This manoeuvre may visualize even small amount of effusion, normally located in infrapulmonary regions, because fluid move to the pleural space near the costal plane of the superior chest, were concavity is more accentuated. The presence of a short pulmonary ligament allows the accumulation of huge amount of pleural effusion (> 500 ml) below the lung, thus mimicking a lifting of the hemi-diaphragm (figure 10).

**Fig.**: Fig-10. Pleural sub-pulmonary right effusion mimicking the lifting of diaphragm. Observe that performing the Hessen's technique the pleural effusion becomes clearly visible.

**References:** E. M. Laugelli; Dipartimento di Scienze Cliniche e Biologiche, University of Torino, Orbassano (TO), ITALY

Of course, thoracic ultrasound has higher accuracy in the detection of pleural effusion, and can be extremely helpful [37]. Another limitation of the CXR technique is the inability to quantify the fluid collection and to diagnose the type of effusion (figure 11).
**Fig.**: Fig-11. CXR in a supine patient. An empiematous limited effusion can be observed on the left.

**References:** E. M. Laugelli; Dipartimento di Scienze Cliniche e Biologiche, University of Torino, Orbassano (TO), ITALY

Conversely, thoracic ultrasound may be helpful to these purposes (figure 12-13).
Fig.: Fig-12. US image of a sepimented massive pleural efusion.

References: E. M. Laugelli; Dipartimento di Scienze Cliniche e Biologiche, University of Torino, Orbassano (TO), ITALY
Fig.: Fig-13. US image of a essudative pleural effusion without seppimentation.

References: E. M. Laugelli; Dipartimento di Scienze Cliniche e Biologiche, University of Torino, Orbassano (TO), ITALY

Conclusion

The sensitivity of CXR is rather low in the diagnosis of some important causes of dyspnoea, such as pneumothorax, pleural effusion and pulmonary edema, particularly in bedside-acquired images. It has been shown a high inter-observer variability of reading that limits the diagnostic usefulness of bedside CXR and complicates the differential diagnosis.

Nevertheless thoracic imaging by standard chest X-ray (CXR) plays a crucial role in the diagnostic process in ED, because of its fast and cheap execution, as for the wide number of critical findings that help identify patients who need subsequent emergency care. It is also very important to indicate the usefulness of other diagnostic tools, as US, or II level exams, as CT multislice.
In conclusion, it is essential to understand role, main findings and limits of standard chest x-ray in these situations.

Personal Information

References


