

Revisiting signs, strengths and weaknesses of Standard Chest Radiography in patients of Acute Dyspnea in the Emergency Department

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ABSTRACT

Dyspnoea, defined as an uncomfortable awareness of breathing, together with thoracic pain are two of the most frequent symptoms of presentation of thoracic diseases in the Emergency Department (ED). Causes of dyspnoea are various and involve not only cardiovascular and respiratory systems. In the emergency setting, thoracic imaging by standard chest X-ray (CXR) plays a crucial role in the diagnostic process, because it is of fast execution and relatively not expensive. Although radiologists are responsible for the final reading of chest radiographs, very often the clinicians, and in particular the emergency physicians, are alone in the emergency room facing this task. In literature many studies have demonstrated how important and essential is an accurate direct interpretation by the clinician without the need of an immediate reading by the radiologist. Moreover, the sensitivity of CXR is much impaired when the study is performed at bedside by portable machines, particularly in the diagnosis of some important causes of acute dyspnoea, such as pulmonary embolism, pneumothorax, and pulmonary edema. In these cases, a high inter-observer variability of bedside CXR reading limits the diagnostic usefulness of the methodology and complicates the differential diagnosis. The aim of this review is to analyze the radiologic signs and the correct use of CXR in the most important conditions that cause cardiac and pulmonary dyspnoea, as acute exacerbation of chronic obstructive pulmonary disease, acute pulmonary oedema, acute pulmonary thromboembolism, pneumothorax and pleural effusion, and to focus indications and limitations of this diagnostic tool.

KEY WORDS

Dyspnoea; chest X-ray; pulmonary oedema; heart failure; pleural effusion

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Introduction

Dyspnoea and thoracic pain are the most frequent symptoms of presentation of thoracic diseases in the Emergency Department (ED). In the emergency setting, thoracic imaging and, first of all, standard chest X-ray (CXR) play a crucial role in the diagnostic process. According to one prospective observational study, the most common diagnoses among elderly patients presenting to

an ED with a complaint of acute shortness of breath or dyspnoea are decompensated heart failure, pneumonia, chronic obstructive pulmonary disease, pulmonary embolism, and asthma (1).

A CXR is frequently helpful in evaluating patients with dyspnoea. Characteristic roentgenographic findings occur in patients with congestive heart failure and pneumonia, and pulmonary fibrosis. The chest radiograph may also be abnormal in patients with obstructive pulmonary disease, but the chest film (particularly the bedside chest film) have low sensitivity above all for the detection of airflow obstruction or pulmonary embolism (2).

Dyspnoea is defined as an uncomfortable awareness of breathing. NYHA classified dyspnoea in four classes, according to the functional decrease performance status of patients: in the I class dyspnoea appears after moderate physical effort, in the II class dyspnoea appears during normal activities, in the III class dyspnoea appears for lower physical efforts, in the IV class dyspnoea is always present (3). Causes of dyspnoea are various and can involve mainly cardiovascular and respiratory apparatus.

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Figure 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.

The aim of this script is to analyze the correct use of CXR in the most important conditions causing cardiac and pulmonary dyspnoea, and to focus indications and limitations of this diagnostic tool.

Acute exacerbation of chronic obstructive pulmonary disease

Chronic obstructive pulmonary disease (COPD) is a syndrome characterized by a progressive limitation to the air flow, poorly reversible and associated with an inflammatory response of airway epithelium. Within this definition we can find both chronic bronchitis and emphysema. Pathophysiological tests can demonstrate a persistent reduction of FEV1 and FEV1/FVC. Presentation of COPD is characterized by persistent exertional dyspnoea, that can worsen during infective exacerbations. During exacerbations it is possible to observe hypoxemia and hypercapnia, while the sputum become abundant and purulent.

Patients with COPD usually have one or two exacerbations per year, often needing hospitalization, with an overall mortality of 3-4%. Incidence of death is higher in the intensive care unit (24%) (4).

Most exacerbations are due to infections of the upper airways (4). In the most severe cases, it is common to observe co-morbidity with congestive heart failure, extra-pulmonary infections or pulmonary embolism.

Role, principal aspects and limitations of chest X-ray

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. Very often COPD exacerbation with involvement of large and/or small airways is not associated with radiographic signs. CXR demonstrates abnormal images only in 16% of cases, mainly limited to signs of inflammatory infiltrates or pulmonary congestion (5-7) (Figure 1).

For these reasons CXR is not recommended as a routine exam, but only in cases of suspected pneumonia, or to rule-out other causes of dyspnoea, such as massive pleural effusion, atelectasis, pneumothorax, pulmonary edema.

Other limitations of CXR in the diagnostic procedure of exacerbation of COPD are high inter- and intraobserver variability, but also low rates of agreement among radiologists regarding the interpretation of pneumonia signs. Rates of agreement for the diagnosis of pneumonia are even lower among trainees or non-radiologist practitioners (8,9).

Acute pulmonary oedema

Acute pulmonary oedema (APE) is a condition of increased fluid content of the 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 or 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 easy, even if the history recording with description of symptoms, clinical findings at examination, time course during hospital stay and treatment response, are all of great help. Nevertheless, a correct differential diagnosis cannot always be clarified, particularly in the critically ill patients.

Role, Main findings and limitations of standard chest X-ray

CXR represents the first line imaging exam in a patients presenting to the ED complaining of acute dyspnoea. The possibility of correct diagnosis at CXR is directly proportional to the severity and the duration of pulmonary congestion. The role of CXR is not only the first diagnosis of APE, but also the differentiation between cardiogenic and non-cardiogenic causes (10) and guiding treatment.

To these purposes, the radiologic signs and findings to be studied are: the perfusion pattern and the spatial distribution of oedema, the size of the vascular peduncle and the cardiac volume. Moreover, it is highly important the recognition of some specific signs, like lung

Table 1. Stage of congestive heart failure. PCWP = pulmonary capillary wedge pressure.

Stage I	-Redistribution of pulmonary vessels
Redistribution	-Cardiomegaly
PCWP 13-18 mmHg	-Broad Vascular Pedicle (non acute CHF)
Stage 2	-Kerley lines
Interstitial Edema	-Peribronchial cuffing
PCWP 18-25 mmHg	-Hazy contour of vessels
	-Subpleural edema
Stage 3	-Consolidation
Alveolar Edema	-Butterfly appearance
PCWP > 25 mmHg	-Cottonwool appearance
	-Pleural effusion



Figure 2. Posterior-anterior CXR demonstrating enlargement of atrial and left ventricles, with redistribution of lung circulation from bases to apex suggestive to pulmonary congestion in a patient with acute decompensated heart failure.

interstitial oedema, pleural effusion and air bronchogram.

In cardiogenic pulmonary edema, CXR may show cardiomegaly, pulmonary venous hypertension, and pleural effusions. Radiologic signs of cardiogenic APE are related to the severity of the condition, and may be divided into 3 stages (Table 1) (11,12). In stage I, an upright examination demonstrates redistribution of blood flow to the nondependent portions of the lungs and the upper lobes (Figure 2). In stage II, there is evidence of interstitial edema with ill-defined vessels and peribronchial cuffing, as well as interlobular septal thickening (Figure 3). In stage III, perihilar and lower-lobe airspace filling is evident, with features typical of consolidation (e.g., confluent opacities, and the inability to



Figure 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 interstitium, the modest pleural effusion and the B Kerley's lines.

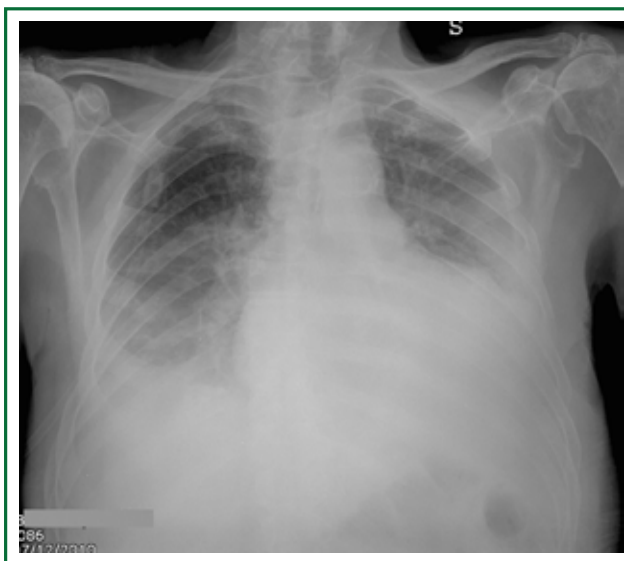


Figure 4. Supine radiogram in a patient with cardiogenic alveolar edema. Note that the vascular perihilar structures are not defined because of the presence of pathology or confluent consolidation shadows, with large pleural effusion. Cardiomegaly is also present.

see pulmonary vessels in the area of abnormality) (Figure 4). The airspace edema tends to spare the periphery in the mid and upper lung. The distribution of the alveolar edema can be influenced by:

- Gravity: supine or erect position and right or left decubitus position;
- Obstructive lung disease, i.e. fluid leakage into the less severe diseased areas of the lung.

Table 2. Radiographics features of pulmonary edema. Modified from Milne *et al*.

	Cardiac	ARDS
Heart size	Enlarged	Normal
Vascular pedicle	Normal or enlarged	Normal or reduced
Pulmonary blood flow distribution	Inverted	Balanced
Peribronchial cuffs	Common	Not common
Regional distribution lung edema	Even	Peripheral/patchy
Air bronchogram	Not common	Very common
Pleural effusion	Very common	Not common

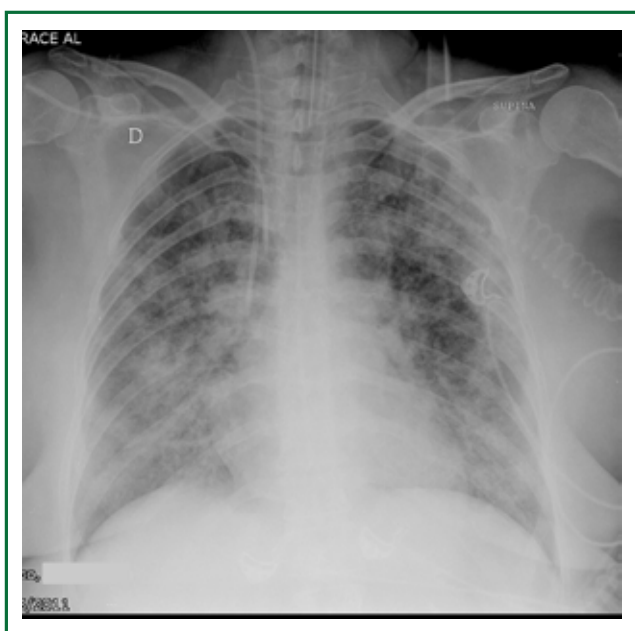


Figure 5. ARDS in H1N1 virus pneumonia. Supine CXR showing bilateral, predominantly peripheral, asymmetrical patchy consolidation with air bronchograms. Septal lines and pleural effusions, are absent.

In non-cardiogenic causes, cardiomegaly and pleural effusions are usually absent. The edema may be interstitial but is more often consolidative. The cephalization of blood flow is missing, though there may be shift of blood flow to less affected areas. The edema is diffuse and does not spare the periphery of the mid or upper lungs (Table 2) (Figure 5).

In cases of large, acute myocardial infarction (MI) and infarction of the mitral valve, support apparatus may produce atypical patterns of pulmonary edema that may mimic noncardiogenic edema or in some cases even a pneumonia.

CXR is moderately specific (specificity 76%, 83%), but not very sensitive (67-68%) for the diagnosis of heart failure (13). Therefore, CXR does not have a direct role in the pathway for the positive diagnosis of heart failure, where the key investigation is echocardiography. The main reason of this limitation is that CXR

is not sensitive enough to rule out heart failure in the presence of a normal radiologic pattern or specific enough to rule - in the condition in the presence of an abnormal pattern. However, CXR is helpful to rule-out other conditions that may enter the differential diagnosis.

Acute pulmonary thrombo-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, in most cases due to embolization of thrombi originated from deep veins, right cardiac chambers or, rarely, from the same pulmonary circulation.

This pathologic condition is quite frequent and sometimes constitutes a hemodynamic and respiratory emergency, leading to death in 30% of untreated cases (14,15). To date, APT is considered the third leading cause of death in western countries and the most misdiagnosed pathologic condition, being correctly diagnosed only in 20% of cases (16). Physical signs as well as routine diagnostic tests are not enough accurate for a safe diagnosis of the condition. Indeed, history, physical examination and blood d-dimer are useful to hypothesize APT in the emergency setting and determine the pre-test probability according to the criteria published by Wells and co-authors (17).

Hemodynamic and clinic consequences of APT are directly related to the extension and stability of the occlusion, as well as the number of obstructed vessels serving the eighteen identifiable bilateral lung segments. From the anatomic point of view, it is common to differentiate three degrees of severity: slight (reduction inferior to 40% of flow), severe (40-60% of flow obstruction) and massive (over 60% obstruction). This classification does not necessarily coincide with the clinical definition of massive APT, that relies exclusively on hemodynamic criteria. When vascular embolic obstruction is superior to 80%, electromechanical dissociation and sudden death usually follow (16). Obviously, hemodynamic and respiratory consequences are widely variable, depending on co-morbidities and pre-existing health status. Clinics of APT may widely vary



Figure 6. Pulmonary thromboembolic disease. In this patient we can find enlargement of the right pulmonary artery to associated sub-segmental atelectasis and elevation of the hemidiaphragm.



Figure 7. Pulmonary thromboembolic disease. In this patient we can find one radiographic findings with high specificity that is decreased vascularity in the left superior lobe. This sign is more easy to recognize in chronic thromboembolism.

from complete lack of symptoms, usually in small segmental or sub-segmental embolism, to severe manifestations as acute respiratory failure, hemodynamic shock and cardiac arrest (18).

Role, main findings and limitations of standard chest X-ray

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, because it is burdened by a low sensivity and specificity.

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 and classified by the clinician (19,20). SCT has a higher sensitivity (87% vs. 33%) and specificity (95% vs. 59%) over CXR, and indubitable advantages due to its fast execution, broad view and objective interpretation, as well as its ability to allow for other diagnoses when the initial clinical suspicion is excluded (21).

Limitations of CXR are related to the difficulty to recognize specific signs. Some radiologic findings have been corroborated in many years of experience. They have been argued by the careful observation of CXR studies in patients with confirmed APT, but rarely such signs are found altogether even in case of clear clinic presentations (22). Nevertheless, many authors suggest that a careful observation of CXR images can show some non specific abnormalities in at least 90% of the cases (23-25). The possible findings of standard CXR in APT are the following (16,26):

(I) **Pulmonary infiltrates**, due to haemorrhagic or oedematous infiltration of secondary lobules, often multiple and

presenting as round foci of alveolar consolidations or irregular jeopardized opacities, without a segmental arrangement, more often located to the right base, sometimes associated with signs of atelectasis or pleural effusion.

(II) **Atelectasis**, often sub-segmental, appearing as curved lines reaching the pleura, secondary to alveolar collapse (line of Fleishner), caused by bronchial obstruction due to mucosa congestion, or alveolar collapse secondary to surfactant reduction, or hypoventilation due to reduced diaphragmatic excursion (Figure 6).

(III) **Diaphragm elevation** secondary not only to reduction of pulmonary volume due to the reduction in surfactant, but mainly to the dysventilation due to reduced respiration movement during pleural pain (Figure 6).

(IV) **Pleural effusion**, mainly serous, bilateral and of slight entity, often in association with basal atelectasis.

(V) **Westermark sign**, uncommon but highly specific, corresponds to a region of impaired vascularisation in the lung region distally to the site of the embolism (Figure 7) (27). Sometimes it is associated with deletion and dilation of the affected pulmonary branch (more often the right pulmonary artery). For a safe interpretation of this sign when present, the film should be compared with an old radiogram where it was absent. Another limitation of this sign is linked to the difficult visualization when CXR is performed in the supine patient.

(VI) **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 hilar regions and other signs previously described. As for the Westermark sign, visualization of these signs should always be

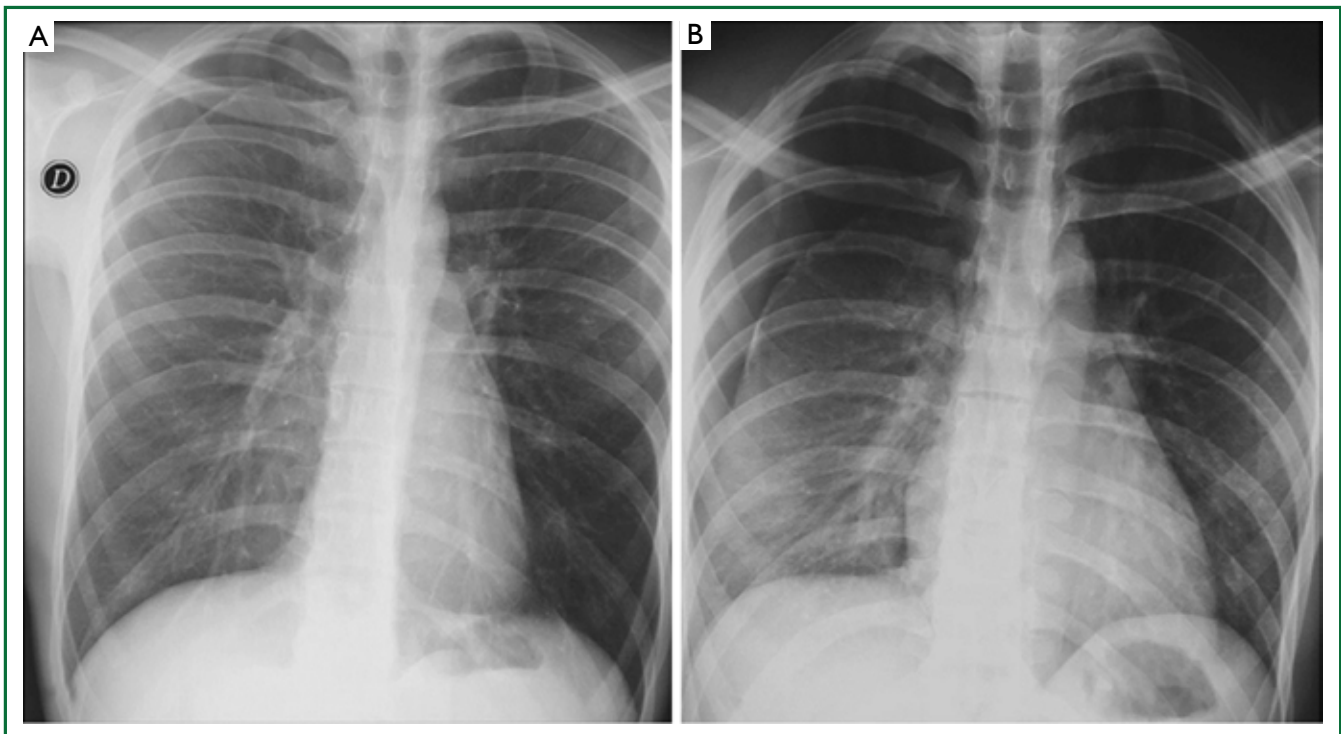


Figure 8. Inspiration and expiration CXR in a case of right sided spontaneous pneumothorax. Note that the extension of pneumothorax is larger during expiration than inspiration and the expansion of the affected hemi-lung is more evident in the affected side.

compared with previous images and they are unreliable when examination is performed in the recumbent position.

(VII) **Hampton's hump** is a triangular opacity with the apex pointing to 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. It is more frequent when APT overlaps with some pre-existing conditions, like venous pulmonary hypertension, cardiac illnesses with left heart failure and COPD. Very often this sign is associated to pleural effusion. 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 (16).

Pneumothorax

Pneumothorax is defined as the presence of air in the pleural cavity, with secondary lung collapse (28). It is usually classified into spontaneous, when it occurs without a preceding event; traumatic, due to direct or indirect trauma; and iatrogenic, categorized by some investigators as a subdivision of traumatic

pneumothorax (29). Spontaneous pneumothorax is the largest group and is classified into primary spontaneous pneumothorax (PSP) and secondary spontaneous pneumothorax (SSP). PSP occurs in young patients without obvious underlying lung disease, and is usually caused by the rupture of a sub-pleural bleb. SSP occurs as a complication of an underlying lung disease, most often COPD or pulmonary tuberculosis (29,30).

Pneumothorax can be complete, with totally collapsed lung, or small with little or no consequences. Continuous introduction of air after every breath without possibility of release, because of a valve mechanism, determines a life-threatening situation that is indicated as tension pneumothorax. Clinical consequences of pneumothorax are strictly connected with the timing of interventions and pre-existing condition of the patient.

Role, principal aspects and limits of chest X-ray

Standard CXR, acquired in orthostatic position, is the elective exam for the diagnosis. Signs used are better visible by acquisition of a forced-expiration imaging (Figure 8). When air is collected between the two pleura layers, the visceral pleura becomes visible as a thin diaphanous line, with no bronco-vascular texture beyond it. Although highly specific, the detection of this sign has a low sensitivity particularly when CXR is performed in the supine position at bedside. A large number of pneumothoraces (probably more than 30%) are not

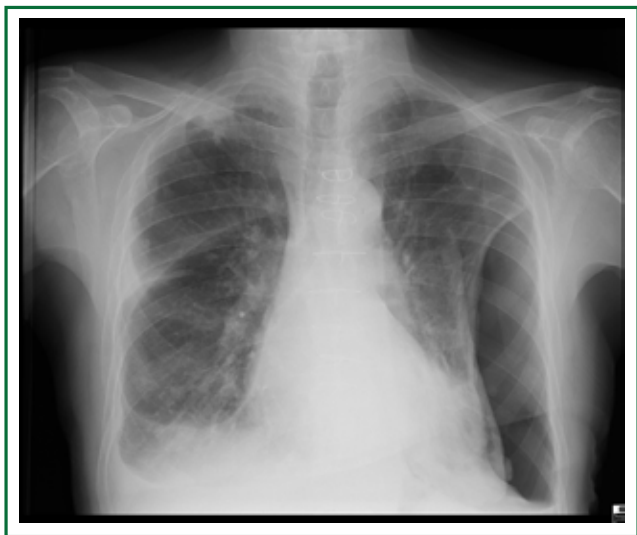


Figure 9. CXR of a patient affected by fibrothorax consequence of tuberculosis. Note a limited layer of pneumothorax visible in the left posterior base.

diagnosed by conventional CXR, particularly when expiration and orthostatic radiograms cannot be obtained for clinical reasons (31). When an anterior/posterior view obtained from a supine patient is evaluated, diagnosis is more difficult because there is the possibility to misdiagnose even large pneumothoraces, because air move up and medially between the lung and the heart. Only after having filled these spaces, free air can gather the usual apical-lateral position (4).

When a CXR is not acquired in an orthostatic posterior-anterior view, there are some other indirect signs that can be important for diagnosing pneumothorax. These are the emphasized radiolucency of the paracardiac region, the deep sulcus sign (32), the appearance of sharp edges of mediastinum, heart and subcutaneous tissues, or the visibility of the anterior-inferior edge of the lung (33). 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 (21,34). In these cases, it is sometimes possible to demonstrate even the smaller layer of pneumothorax.

Free air can also collect in a fissure or behind the triangular ligament, or it can distribute around an atelectasis or a consolidated 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 9).

In these cases the differential diagnosis between pneumothorax, pneumo-pericardium and pneumo-mediastinum at CXR can be very difficult.

Diagnosis of tension pneumothorax is generally based mainly on the first clinical evaluation because it gives usually clear

physical signs that may evolve rapidly to hemodynamic shock and cardiac arrest. When the clinical conditions are not rapidly evolving, CXR may be helpful in the early diagnosis allowing the emergency physician a greater confidence in deciding aggressive life-saving decompression treatment. The main radiologic signs of tension pneumothorax 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 pneumothorax and even in the detection of radio-occult pneumothorax, being far more accurate than CXR and equivalent to CT scan (35). Its advantages include the fact that it can easily and quickly be performed at the bedside by a wide range of operators, such as trauma, emergency, and critical care physicians (36).

Pleural effusion

Pleural effusion is defined as the presence of fluid 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 laparotomy or in post-partum (37-39).

Several conditions can cause pleural effusion, as cardiovascular diseases, hyper-expansion of body fluids due to renal and hepatic failure, infections, autoimmune disorders, cancer and traumas (40).

Role, principal aspects and limitations of chest X-ray

CXR is always been considered the first line diagnostic tool to be used in the diagnosis and 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. Anyway, severity of the disorder, lung and chest wall compliance, capillarity of the pleural layers and the physical features of the fluid, influence the spatial distribution in the pleural cavity (41).

Classical radiologic signs are consistent with a dependent opacity with lateral upward sloping of a meniscus-shaped contour. The diaphragmatic contour is partially or completely obliterated, depending on the amount of collected fluid (silhouette sign) (Figure 10 A,B). In case of massive effusion, all the hemi-thorax can be filled and mediastinum can be shifted

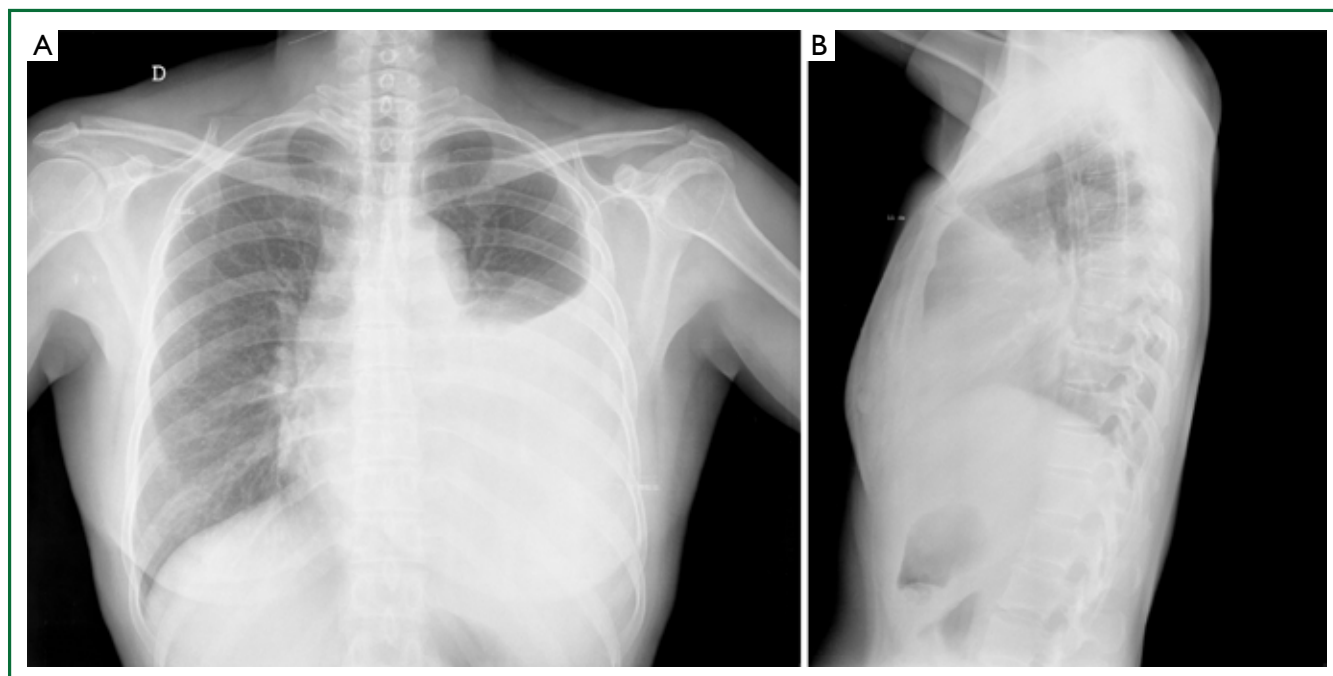


Figure 10. Posterior-anterior (A) and lateral (B) views at CXR of a patient with massive left pleural effusion. Note the typical Damoiseau-Ellis line.



Figure 11. In Hessen's view we can recognize a little amount of pleural effusion, not visible in the standard projection (courtesy of Prof. Cesare Fava).

contra laterally.

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 (4). Some radiologic signs allow 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 accumulate large amount of pleural effusion for gravity. These signs are useful only when a comparison between the two hemi-thorax can be performed, while in case of massive effusion equally distributed on both sides, they are extremely difficult to be recognized.

When bedside CXR is correctly interpreted, the reader can detect large pleural effusions 92% of the time and can exclude large effusions with high confidence (42).

In selected cases a lateral view with 20° of Trendelenburg inclination (the Hessen view) can obviate to lack of accuracy (37,43) (Figure 11A,B,C). This manoeuvre may visualize even small amount of effusion, normally located in intrapulmonary

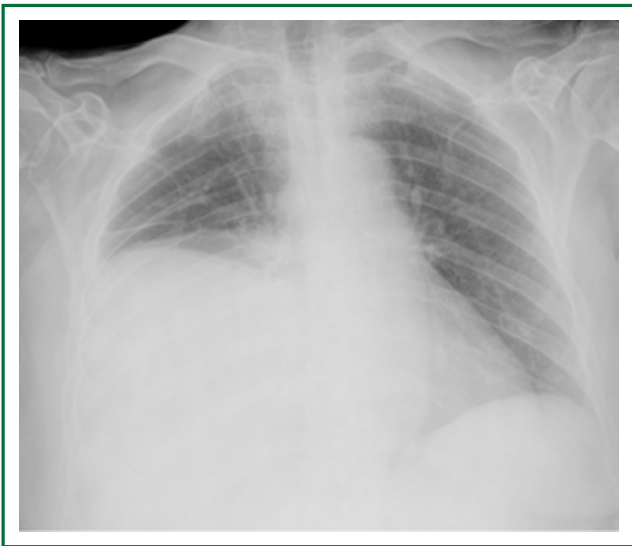


Figure 12. Pleural sub-pulmonary right effusion mimicking the lifting of diaphragm.

regions, because fluid move to the pleural space near the costal plane of the superior chest, where 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 12). This approach is now replaced by lung ultrasound.

Of course, thoracic ultrasound has higher accuracy in the detection of pleural effusion, and can be extremely helpful (35). Another limitation of the CXR technique is the inability to quantify the fluid collection and to diagnose the type of effusion (44). Conversely, thoracic ultrasound may be helpful to these purposes.

Conclusion

In conclusion, CXR has a great potential in the first diagnosis of many lung disorders causing acute dyspnoea and chest pain, pending the knowledge and correct interpretation of several signs. However, the physicians should be aware that the sensitivity of CXR is rather low in the diagnosis of 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. For these reasons it is very important that it should be interpreted by a radiologist experienced in thoracic radiology. Nevertheless thoracic imaging by CXR plays a crucial role in the diagnostic process in ED, because it allows a panoramic view, being at the same time cost-safe and relatively time-saving.

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References

1. Ray P, Birolleau S, Lefort Y, et al. Acute respiratory failure in the elderly: etiology, emergency diagnosis and prognosis. *Crit Care* 2006;10:R82.
2. Fox JC, Irwin Z. Emergency and critical care imaging. *Emerg Med Clin North Am* 2008;26:787-812.
3. Russell SD, Saval MA, Robbins JL, et al. New York Heart Association functional class predicts exercise parameters in the current era. *Am Heart J* 2009;158:S24-30.
4. Barozzi L, Valentino M. La diagnostica per immagini in pronto soccorso Cap 4: 110-111. CG Ed Medico Scientifiche, Torino; 2008.
5. Tsai TW, Gallagher E, Lombardi G, et al. Guidelines for the selective ordering of admission chest radiography in adult obstructive airway disease. *Ann Emerg Med* 1993;22:1854-8.
6. Emerman CL, Cydulka R. Evaluation of high-yield criteria for chest radiography in acute exacerbation of chronic obstructive pulmonary disease. *Ann Emerg Med* 1993;22:680-4.
7. Sherman S, Skoney J, Ravikrishnan K. Routine chest radiographs in exacerbations of chronic obstructive pulmonary disease. *Arch Intern Med* 1989;149:2493-6.
8. Campbell SG, Murray DD, Hawass A, et al. Agreement between emergency physician diagnosis and radiologist reports in patients discharged from an emergency department with community acquired pneumonia. *Emerg Radiol* 2005;11:242-6.
9. Eng J, Mysko WK, Weller GE, et al. Interpretation of emergency department radiographs: a comparison of emergency medicine physicians with radiologists, residents with faculty, and film with digital display. *AJR Am J Roentgenol* 2000;175:1233-8.
10. Milne ENC, Pistolesi M, Miniati M. The radiologic distinction of cardiogenic and noncardiogenic edema. *AJR Am J Roentgenol* 1985;144:879-94.
11. Meszaros WT. Lung changes in left heart failure. *Circulation* 1973;47:859-71.
12. Ravin CE. Radiographic analysis of vascular distribution: a review. *Bull N Y Acad Med* 1983;59:728-43.
13. Mant J, Doust J, Roalfe A. Systematic review and individual patient data meta-analysis of diagnosis of heart failure, with modelling of implications of different diagnostic strategies in primary care. *Health Technol Assess* 2009;13:1-207,iii.
14. Chiesa A, Olivetti L. Diagnostica per immagini in medicina clinica. CG Ed Medico Scientifiche, Torino; 2003.
15. MacMahon H. Portable chest radiology. *Respir Care* 1999;44:1018-32.
16. Fraser RG, Parè JAP. Diagnosis of diseases of the chest. Vol 1. Ed W B Saunders Co, Philadelphia; 1979.
17. Collins SP, Lindsell CJ, Storrow AB, et al. Prevalence of negative chest radiography results in the emergency department patient with decompensated heart failure. *Ann Emerg Med* 2006;47:13-8.
18. Volpicelli G, Caramello V, Cardinale L, et al. Bedside ultrasound of the lung for the monitoring of acute decompensated heart failure. *Am J Emerg Med*

- 2008;26:585-91.
19. Ruskin JA, Gurney JW, Thorsen MK, et al. Detection of pleural effusions on supine chest radiographs. *AJR Am J Roentgenol* 1987;148:681-3.
 20. Chen JY, Chao TH, Guo YL, et al. A simplified clinical model to predict pulmonary embolism in patients with acute dyspnea. *Int Heart J* 2006;47:259-71.
 21. Harrison A, Amudson S. Evaluation and management of the acutely dyspnoic patient: the role of biomarkers. *Am J Emerg Med* 2005;23:371-8.
 22. Pedicelli G, Boni S, Concorsi P, et al. La tromboembolia polmonare. *Radiol Med (Torino)* 1992;84:242-6.
 23. Wells PS, Anderson DR, Rodger M, et al. Derivation of a simple clinical model to categorize patients probability of pulmonary embolism: increasing the models utility with the simplified d-dimer. *Thromb Haemost* 2000;83:416-20.
 24. Shiber JR, Santana J. Dyspnea. *Med Clin North Am* 2006;90:453-79.
 25. Fox JC, Irwin Z. Emergency and critical care imaging. *Emerg Med Clin North Am* 2008;26:787-812.
 26. Westcott J. Criteria of appropriateness for dyspnea. *Radiology* 2000;215:641-643.
 27. Fleischner FG. Observation of the radiologic changes in pulmonary embolism - In: Sasahara AA (Ed) "Pulmonary Embolic Disease", New York; 1965.
 28. Heitzman ER. The lung: radiologic pathologic correlation. CV Mosby Company. St Louis; 1984.
 29. Tocino IM, Miller MH, Fairfax WR. Distribution of pneumothorax in the supine and semirecumbent critically ill adult. *AJR Am J Roentgenol* 1985;144:901-5.
 30. Weissberg D, Refaely Y. Pneumothorax: experience with 1,199 patients. *Chest* 2000;117:1279-85.
 31. Kelley JM, Elliott PL. The radiologic evaluation of patient with suspected pulmonary thromboembolic disease. *Med Clin North Am* 1975;59:3-36.
 32. Moses DC, Silver TM, Bookstein JJ. The complementary roles of chest radiography, lung scanning and selective pulmonary angiography in the diagnosis of pulmonary embolism. *Circulation* 1974;49:179-88.
 33. Worsley DF, Alavi A, Aronchic JM, et al. Chest radiographic findings in patients with acute pulmonary embolism: observations from the PIOPED study. *Radiology* 1993;189:133-6.
 34. Westermark N. On the Roentgen diagnosis of lung embolism. *Acta Radiol* 1938;19:357-72.
 35. Volpicelli G, Elbarbary M, Blaivas M, et al. International evidence-based recommendations for point-of-care lung ultrasound. *Intensive Care Med* 2012;38:577-91.
 36. Volpicelli G. Sonographic diagnosis of pneumothorax. *Intensive Care Med* 2011;37:224-32.
 37. Trupka A, Waydhas C, Hallfeldt KK, et al. Value of thoracic computed tomography in the first assessment of severely injured patients with blunt chest trauma: results of a prospective study. *J Trauma* 1997;43:405-11.
 38. Khan AN, Al-Jahdali H, Al-Ghanem S, et al. Reading chest radiographs in the critically ill (part I): normal chest radiographic appearance, instrumentations and complication from instrumentation. *Ann Thorac Med* 2009;4:75-87.
 39. Light RW, George RB. Incidence and significance of pleural effusion after abdominal surgery. *Chest* 1976;69:621-5.
 40. Muller R, Lofstedt S. The reacting of the pleura in primary tuberculosis of the lungs. *Acta Med Scand* 1945;122:105-33.
 41. Hessen I. Roentgen examination of pleural fluid: a study of the localization of free effusion, the potencialities of diagnosing minimal quantities of fluid and its existence under physiological conditions. *Acta Radiol* 1951;86:1-80.
 42. Kitazono MT, Lau CT, Parada AN, et al. Differentiation of pleural effusions from parenchymal opacities: accuracy of bedside chest radiography. *AJR Am J Roentgenol* 2010;194:407-12.
 43. Garofalo G, Busso M, Perotto F, et al. Ultrasound diagnosis of pneumothorax. *Radiol Med (Torino)* 2006;111:516-25.
 44. Eibenberger KL, Dock WI, Ammann ME, et al. Quantification of pleural effusions: sonography versus radiography. *Radiology* 1994;191:681-4.



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