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## Lung imaging in the adult respiratory distress syndrome: current practice and new insights

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### Introduction

The lungs respond pathologically in a stereotypical manner to a variety of insults [1]. In 1967, Ashbaugh and colleagues documented their clinical experience of 12 adult patients, all of whom, despite differing precipitating events, developed sudden and progressive dyspnoea, refractory hypoxemia, decreased lung compliance and diffuse alveolar infiltrates on chest radiographs [2]. Their descriptions gave birth to the term acute respiratory distress syndrome (ARDS). Much of the subsequent interest in the syndrome has focused on its pathophysiology, complications, management and prognosis. This review considers the role of chest radiography and computed tomography (CT) in ARDS. The pathology relevant to the imaging and the recent insights into the pathophysiology of the syndrome provided by computed tomography (CT) are also discussed.

### Radiographic considerations:

Chest radiography is performed daily in the majority of patients with ARDS in intensive care units (ICUs) and is used to detect supervening complications and monitor progress. Clinically unsuspected abnormalities or progression of previously documented changes have been reported in 15–65% of ICU patients [3–5], such a wide range between published studies probably reflecting differing concepts of “significant abnormality”. However,

even with an optimistic view of the utility of chest radiography, the cost effectiveness of performing this investigation with such frequency has so far escaped formal evaluation.

The satisfactory imaging of critically ill patients on the ICU poses special problems. Limited patient mobility and co-operation plus difficulties of access due to monitoring equipment often result in radiographs of non-diagnostic quality. The power output of portable units is limited so that longer exposure times are required, resulting in movement artefact. The shorter film-to-focus distance, together with the usual antero-posterior projection necessitated by the supine posture of most ICU patients, causes magnification effects which hamper accurate interpretation of cardiac size. Consistency of film exposure from day-to-day is hard to achieve using standard portable units, making valid comparisons between serial radiographs difficult and sometimes impossible.

The advent of digital systems, particularly those using storage phosphor technology, has addressed many of the limitations of conventional film radiography. A review of the basic principles of digital storage phosphor radiography [6] is useful in considering the advantages and disadvantages of this relatively new technology.

Following transmission of X-rays through the patient, incident energy is trapped in the phosphor plate as a latent image; the plate itself being housed in the same way as conventional film in a cassette. The plate is scanned by a laser light source whereupon the trapped energy is released in the form of light (the phenomenon of photostimulable luminescence). The light emitted has an intensity proportional to the X-ray energy previously absorbed and is detected by a rapid scanning photomultiplier tube which follows in the wake of the laser beam. The detected signal is transformed by an analogue-to-digital converter to digital data. The final digital image may be presented in a number of different formats, including one which resembles conventional film [7–9] (Fig. 1).

**Table 1** Advantages of digital imaging in intensive care

- Consistency of optical density allowing reliable comparison on serial radiographs
- Significant reduction of film “re-takes”, with a resultant decrease in radiation burden
- Ability to post-process images into different formats and enhance difficult image areas
- Potential for rapid transmission and recall of digital images

In the ICU storage phosphor radiography offers some clear advantages over conventional film-screen combinations (Table 1). The phosphor plate has a linear (X-ray) dose-response curve with a large dynamic range [6], allowing satisfactory images to be obtained over a large range of exposures. The need for re-take films is dramatically reduced. A further feature is the ability to post-process the digital image [10]. In practical terms, perhaps the most important advantage of digitally acquired chest radiographs is the uniformity of their overall optical density over a wide exposure range; the result is that serial radiographs are truly comparable, and any detectable change in lung density is likely to be significant rather than due to over- or under-exposure.

The clinical validity of digital systems compared to conventional radiography has been tested in many studies [8, 9, 11–13]. Although there are some discrepancies in published reports, studies comparing the digital to conventional formats have, in general, yielded favourable results for the detection of interstitial abnormalities, the siting of cardiopulmonary monitoring devices and the general perception of image quality [7, 9, 11, 13, 14]. Indeed, the use of a digital image network in one department was associated with improved clinical efficiency, as judged by a significant decrease in the time interval from the completion of the chest radiograph to a specific intervention being performed on the basis of radiographic findings [15]. Although the implementation of digital systems into a department is inevitably associated with an initial financial burden [16, 17], potential cost-savings have been envisaged in the medium term [18].

#### Radiological-pathological correlations in ARDS:

The pathological changes of acute lung injury generally follow a stereotypical course of varying severity regardless of the inciting insult [1, 19]. Although the appearances of ARDS on chest radiography are generally non-specific, three pathological stages have been described [1, 19–21] which, in the absence of pre-existing lung disease or concurrent infection and prior to the institution of mechanical ventilation, broadly correlate with radiographic findings [20, 22–24].

#### Stage I (0–24 h)

This stage is an initial acute exudative phase in which sloughing of the alveolar epithelium and capillary endothelium occurs. A mild interstitial inflammatory response causes widening of the interstitium, but fluid leakage into the interstitium is minimal at this stage. There are few, if any, radiographic correlates of the early phase, which has accordingly been termed the “radiologically-latent” stage [20, 22, 23, 25], although very occasionally there may be signs of interstitial oedema in the form of septal lines [23].

#### Stage II (24–36 h)

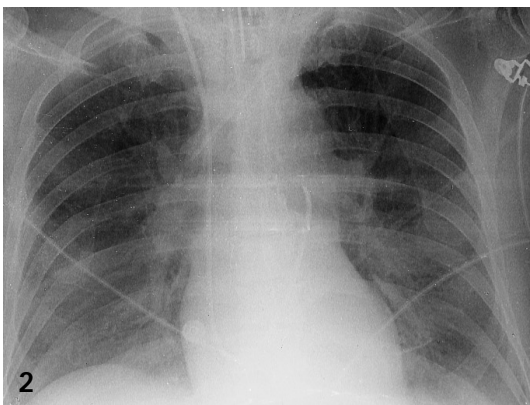
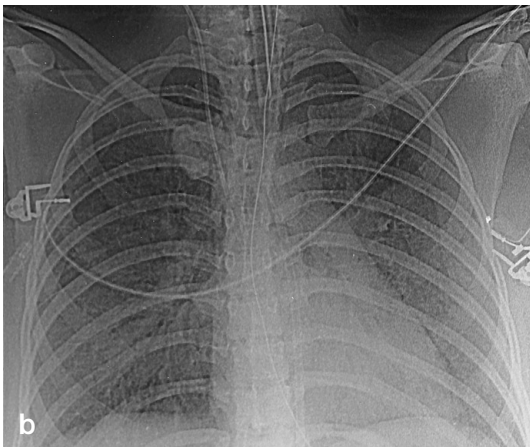
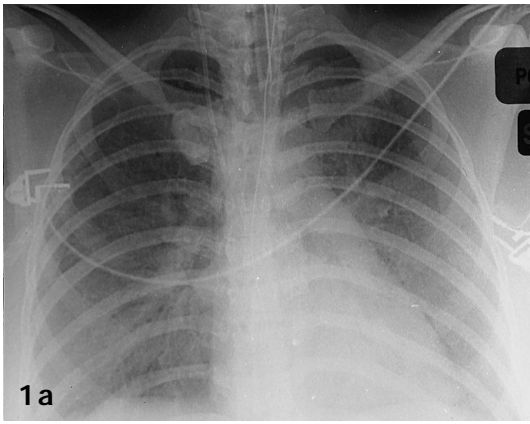
Fluid leakage into the interstitium and alveolar spaces becomes more prominent, as does alveolar collapse. Macroscopically this results in the appearance of dense, consolidated lungs at histological examination. The formation of hyaline membranes within respiratory bronchioles and alveolar ducts is also seen. At the outset of this stage the chest radiograph may show an ill-defined hazy or “ground-glass” opacification reflecting a more pronounced leak of fluid into the interstitium [22, 24] (Fig. 2). As alveolar filling proceeds however, the radiographic picture changes; initially involvement may be patchy but is usually followed by diffuse and apparently homogeneous air-space opacification [20, 22, 24, 25]. Air bronchograms may become a prominent feature; a sign which has been reported to be a useful differentiating feature between the increased permeability oedema of ARDS and that due to cardiac failure [25, 26] (Fig. 3). Following the catastrophic events of the first 36 h and for a variable time thereafter, the radiographic appearances generally remain static. Any significant change in regional opacification during this stage may therefore indicate supervening infection.

#### Stage III (approximately 72 h onwards)

This stage is characterized histologically by fibrotic repair of the lung parenchyma and hyperplasia of type II alveolar pneumocytes. Chest radiography reveals slow resolution of air-space consolidation, but there may be evidence of residual interstitial and intra-alveolar fibrosis in the form of reticular and persistent ground-glass shadowing [20, 27].

#### The utility of chest radiography in the management of ARDS

Superimposed on the radiographic pattern of “uncomplicated” ARDS there may be signs, sometimes subtle, of co-existing pathology whose recognition has a consid-



erable impact on patient management. Clinical questions which commonly arise in critically ill patients include the distinction between causes of pulmonary oedema and the detection of complications (in particular, those of superimposed infections and barotrauma). Serial chest radiographs are often the cornerstone in providing this information.

### 1. Differentiating hydrostatic (cardiogenic) from increased permeability oedema:

Determining the cause of pulmonary oedema by clinical examination alone is problematic. Although the differentiation of cardiogenic from non-cardiogenic oedema is aided by the estimation of pulmonary arterial occlusion pressure, this apparently objective method is also prone to errors [28].

Plain chest radiographs may be more sensitive than clinical examination in the early detection of pulmonary oedema [29]. Certainly, radiographic scores of extravascular lung fluid have been shown to correlate well with physiological measurements of lung water [30] and depression of arterial oxygen tension [31]. Radiographs taken both before and after a period of prolonged decubitus positioning may be a useful, if cumbersome, adjunct in the differential diagnosis of pulmonary infiltrates on chest radiography [32]. Nevertheless, opinion remains divided on the ability of plain chest radiographs to differentiate consistently between causes of pulmonary oedema [26, 30, 33–36]. The radiographic signs considered most discriminatory in one series included the pattern of blood flow, the distribution of oedema and the width of the vascular pedicle [26]. In 50% of cases with cardiogenic oedema, blood flow appeared preferentially diverted to the upper lobe vessels (termed an “inverted” pattern) whereas in the remaining 50%, a “normal” (lower zones > upper zones) or “balanced” (lower zone = upper zone) pattern was evident. In the group of patients with ARDS, only 10% showed an inverted pattern; normal or balanced flow were more commonly seen. A peripheral distribution of oedema was strikingly absent in patients with cardiogenic oedema but, by contrast, it was the commonest pattern

◀ **Fig. 1 a, b** Digital CXR pair in a patient with early ARDS. **a** has been processed to resemble a conventional radiograph whereas **b** has an edge-enhanced effect due to its high spatial frequency enhancement and wide latitude

**Fig. 2** Digital chest radiograph in a patient with chronic myeloid leukaemia who developed ARDS secondary to systemic sepsis. Note the bilateral ground-glass shadowing causing obscuration of the underlying pulmonary vessels. No air bronchograms are seen

**Fig. 3** Radiograph in a patient with acute lung injury following surgery. Consolidation with air bronchograms is a dominant feature, reflecting outpouring of fluid into the air spaces

seen in ARDS. Based on these findings and some ancillary features, the authors claimed an overall accuracy for chest radiography of 86–89%. Their findings are, however, at variance with other studies using similar scoring systems to differentiate between causes of oedema [33, 35]. Though a high specificity (87%) for the finding of patchy or peripherally distributed oedema in high permeability oedema has been shown, the sensitivity is below 50% [33]. Similarly, the discriminatory value of signs of interstitial fluid accumulation (peribronchial/peri-vascular cuffing and septal lines) and pleural effusions have been questioned [33, 35, 37, 38].

Clearly, although some general radiographic patterns sometimes allow a distinction to be made (Table 2), the inconsistency of these radiographic signs suggests that the differentiation between the various forms of pulmonary oedema, on radiographic features alone, is unreliable.

## 2. Detection of complications during ARDS:

Chest radiography plays an important role in the detection of both iatrogenic and nosocomial complications during ARDS. A review of iatrogenic complications has been comprehensively dealt with elsewhere [39, 40].

### a) Infection

Pulmonary infection in patients with ARDS is common and may pose a more serious threat to survival than the underlying respiratory failure [41–44]. Although Fowler and co-workers [45] considered respiratory insufficiency to be a major determinant of mortality, deaths directly related to irreversible respiratory failure may account for less than a quarter of all fatalities [41]; a figure which contrasts with a mortality exceeding 70% in patients developing infection during ARDS [41, 42]. Pulmonary and abdominal foci are the most frequent sources of sepsis following acute lung injury [41, 42] and in one autopsy series in ARDS, a clinically unsuspected, but potentially treatable source of pulmonary or abdominal sepsis was found in 40% of cases [43].

The diagnosis of nosocomial lung infection based on clinical or radiological signs is not straightforward [44, 46, 47]. Acknowledged clinical indicators of pneumonia (pyrexia, purulent secretions, cough and a leucocytosis) are unreliable in critically ill, ventilated patients [44, 46, 48, 49]. The diagnosis of pneumonia based on the evolution of signs on chest X-ray is similarly hampered by the lack of specificity of radiographic changes [50]. Though an appreciation of the sequence of radiographic changes seen in uncomplicated ARDS is theoretically useful in predicting superimposed pneumonic consolidation, in practice the diagnostic accuracy of chest radiography in these circumstances is low [51].

### b) Barotrauma

The introduction of mechanical ventilation with positive end-expiratory pressure (PEEP) in the management of patients with ARDS was a significant advance in the care of patients with refractory hypoxia [52–54]. The increasing use of such therapy however, resulted in a concomitant rise in the incidence of pulmonary barotrauma [55]. The incidence of ventilator-related pulmonary injury in ARDS may be as high as 50% [56] and is largely related to the type of ventilation used, the use of high tidal volumes and inflationary pressures, the magnitude of peak airway pressure, the age of the patient and the presence of underlying lung disease [44, 55, 57–59].

Leakage of air into extra-alveolar spaces with subsequent tracking into the mediastinum, pleural cavity or subcutaneous tissues may be explained by a knowledge of the pathophysiology of alveolar rupture, plus the anatomy of the intra-thoracic fascial planes and peri-vascular interstitium [60, 61]. Nevertheless, it is often impossible to infer from the chest radiography the mechanism by which air gains access to the various extra-pulmonary spaces. Extra-alveolar air may be identified on chest radiographs in many sites; pulmonary interstitial emphysema, subcutaneous emphysema, subpleural cysts, pneumothorax, pneumomediastinum, pneumoperitoneum and pneumoretroperitoneum are all reported complications of ventilatory therapy and apparently readily visible [55, 62–64].

The radiological detection of pulmonary interstitial emphysema (PIE) during mechanical ventilation is important. The tendency for interstitial air to migrate along known anatomical planes makes PIE an important precursor of pneumomediastinum and potentially life-threatening pneumothoraces [63]. In a retrospective study of 15 ventilated patients, PIE occurred in 13 cases (88%). Pneumothorax developed in 10/13 cases and in none of these cases did a pneumothorax predate the signs of PIE [57]. Unfortunately, although the radiographic signs of PIE have been well documented [62, 65, 66], in practice PIE is often difficult, if not impossible, to detect [65, 66].

The diagnosis of a pneumothorax in the erect patient usually poses no problems. Most commonly, identification of a visceral pleural edge in the upper zones confirms the diagnosis [67]. In the supine position however, the detection of pneumothorax may not be easy; the chest radiographic appearance being governed by the anatomical localisation of air within the pleural space. In one review of 88 critically ill patients, the antero-medial pleural space was the most common site of localization (51 of 112 pneumothoraces) [68]. Air was present in the subpulmonic space in just over a quarter of cases. Less common sites of localization were the apico-lateral and postero-medial pleural spaces and air collections within fissures.

**Table 2** Differentiating features on plain chest radiographs

	Cardiogenic oedema	ARDS
Heart size	Increased	Normal
Blood flow distribution	Inverted	Normal or balanced
Septal lines	+	Rare
Bronchovascular cuffing	+	Rare
Pleural effusions	+	Rare
Air bronchograms	Rare	+
Distribution of oedema	Central and peripheral	Patchy, peripheral

**Table 3** Indications for CT in ARDS

- Detection of complications (e.g. cryptic pneumothoraces, abscesses) not demonstrated by chest radiography in patients who are deteriorating or not responding to conventional therapy
- As an aid to quantify the extent of lung involvement in ARDS patients with equivocal radiographic and/or physiological signs
- Demonstration of significant areas of dependent atelectasis – potentially identifying patients in whom PEEP and/or prone nursing may be beneficial
- In the follow-up survivors to identify the morphological consequences of ARDS and its treatment

A medially sited pneumothorax [69] and more particularly a shallow anterior collection may be overlooked on standard chest radiographs. In this context, a lateral horizontal beam film may be useful to confirm the suspicion of an antero-medial collection [69–71]. Subpulmonic pneumothoraces may also present as an area of increased lucency over the lower zone. Helpful ancillary signs of subpulmonic localisation include the appearance of an unusually deep anterior costophrenic sulcus, increased clarity of the adjacent mediastinal/cardiac borders and a rounded mass-like appearance to the pericardial fat pads [67, 71, 72].

Recurrent pneumothoraces are common in ventilated patients [73]: recurrence was documented in 16 out of 47 (34%) pneumothoraces, despite the presence of an ipsilateral chest drain, in a recent study [73]. Patients at risk of recurrent pneumothorax were identified by analysis of the orientation of chest drains on chest radiographs. Twelve out of 14 (86%) recurrent pneumothoraces in ARDS patients were associated with a “horizontally” positioned drain, presumably reflecting the siting of a drain in the interlobar fissures or posteriorly in the hemithorax. A horizontally oriented drain had a positive predictive value of 86% for recurrence in patients with ARDS.

Pneumothoraces are frequently under tension in ventilated patients and may rapidly lead to life-threatening cardiovascular compromise [55, 64]. Diagnosis of a tension pneumothorax is usually clinical and relies on a

high index of suspicion. Chest radiographs, if performed, reveal contralateral mediastinal shift and ipsilateral diaphragmatic depression [67]. Occasionally, however, the signs of a tension pneumothorax may be more subtle [74].

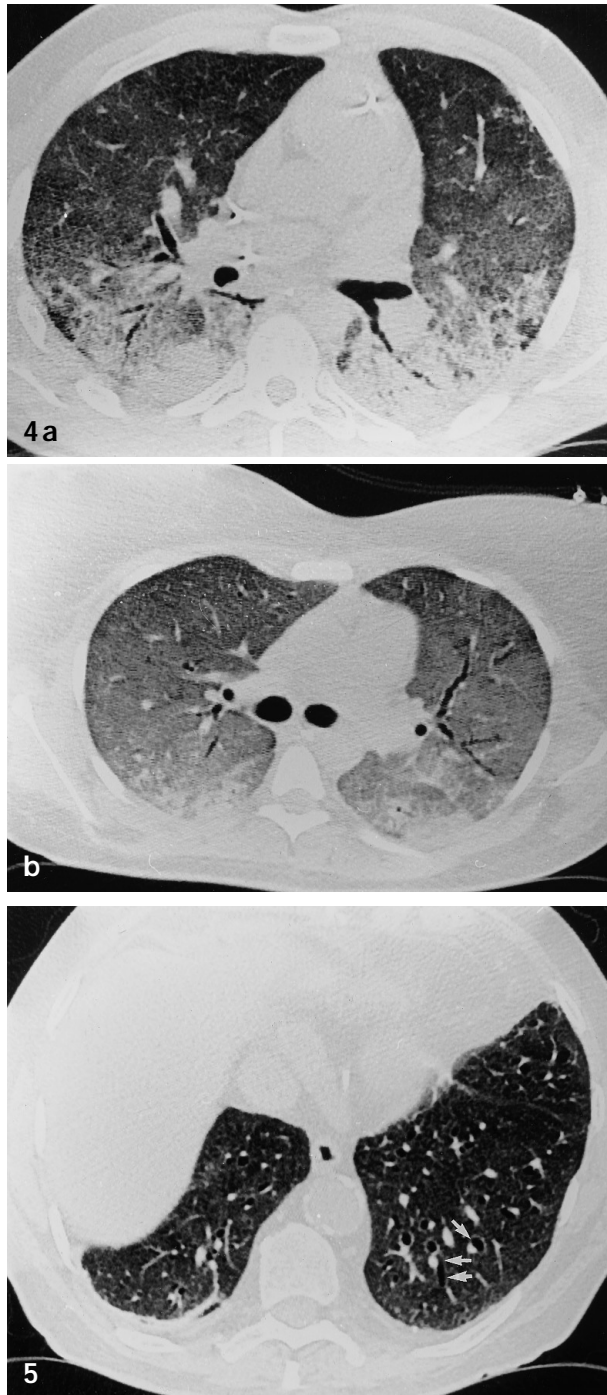
### Computed tomography in ARDS

Computed tomography (CT) is increasingly used as a routine investigation in critically-ill patients in centres with access to a fast, sub-one second scanner (Table 3). In addition to its contributions to understanding the pathophysiology of ARDS, CT scanning has proved valuable in the detection of complications not identified on chest radiography such as unsuspected abscesses, emphysemas and mediastinal disease [75]. The transfer of ventilated patients to scanning suites has generally not been associated with increased morbidity [76, 77], although in the planning of a new ICU, an ideal situation would be to have a CT scanner at the point of entry to the unit.

#### I. CT patterns of parenchymal involvement in ARDS:

CT has shown that, contrary to what has traditionally been described on plain chest radiography, homogeneous parenchymal involvement in ARDS is unusual [78, 79]. In a small study comparing the chest radiographic and CT features in 13 cases, only 3 had homogeneous opacification on both plain film and CT images [80]. The majority of CT examinations (9/13) revealed patchy parenchymal involvement, in contrast to chest radiographs which showed generalised involvement in 11/13 (85%). Areas of increased density were interspersed with lung of relatively normal appearance. The most striking finding was the tendency for parenchymal opacification to be sited in the dependent lung regions, a feature confirmed by subsequent investigators [76, 81–83]. Remote from areas of dense opacification, CT may show ground-glass opacification [84] (Fig. 4). It is important to understand that the latter is a non-specific finding that cannot be used to differentiate between the various possible causes of ground-glass opacification in ARDS (i.e. fine fibrosis versus other interstitial infiltrates).

High-resolution CT (HRCT) has shown abnormalities in regions thought to be uninvolved on conventional thick section scans [22, 85]. In one study of ARDS patients, areas of ground-glass opacification and evidence of parenchymal distortion were seen in all eight patients with ARDS in whom HRCT was performed [85]. Ground-glass shadowing was present on the follow-up examination in four out of eight cases. Evidence of interstitial fibrosis, as judged by persistent reticular shadowing in association with a tractional dilatation of



**Fig. 4 a, b** HRCT in two patients with acute ARDS. **a** Areas of dense opacification in the dependent regions of both lungs, indicate regions of “compression atelectasis”. Remote from these areas there is widespread but patchy ground-glass opacification. **b** An earlier stage of acute lung injury showing ground-glass opacification as the dominant pattern. Minimal dependent atelectasis is seen

**Fig. 5** HRCT in a survivor, 7 months after acute lung injury. Dilated subsegmental bronchi in the left lower lobe (*arrows*) combined with a background ground-glass opacification are evidence of a fine established interstitial fibrosis, below the limits of resolution of HRCT

subsegmental bronchi, was also noted (Fig. 5). Dilatation of segmental and subsegmental bronchi within regions of ground-glass opacification is a reliable indicator of fine interstitial fibrosis below the limits of resolution on HRCT in chronic infiltrative lung disease [86, 87], but whether this rule applies in ARDS has not yet been determined.

## II. Patho-physiological insights from CT in ARDS:

Based on the CT observations in lungs of anaesthetized individuals, it has been suggested that the areas of dense postero-basal opacification in ARDS are actually regions of compression atelectasis [88–90]. Following the induction of anaesthesia, these areas of opacification can appear very rapidly, move to more dependent regions of lung with a change in posture and may be reversed by the application of PEEP [88, 89]. More significantly, correlations between areas of atelectasis on CT and the degree of intra-pulmonary shunting and poor oxygenation in both human subjects and animals have been shown [90, 91].

The findings in anaesthetized patients have stimulated similar investigations in patients with ARDS. A shift from the supine to prone position results in a redistribution of previously postero-basal opacities to more dependent lung regions and may be accompanied by improvements in arterial oxygenation in some cases [81, 92]. In 8/13 (61 %) patients with ARDS, there were significant improvements of arterial oxygen tension, from baseline levels, after 30 min of prone positioning ( $p < 0.01$ ) [81]. Furthermore, improvements in oxygenation remained above baseline values ( $p < 0.01$ ) even after returning patients to the supine position. Based on their results, the authors have recommended a trial of prone positioning in all ARDS patients, to identify likely “responders” and “non-responders”.

CT has shown that the superimposed pressure on the most dependent lung in ARDS is significantly higher than in normal volunteers ( $p < 0.01$ ) [92]. It therefore seems likely that the lung behaves as a “sponge”, with compression atelectasis of dependent lung units [92, 93]. The relative speed with which redistribution of densities occurs on changing position is in keeping with this hypothesis and there is further support in that increasing the level of PEEP above the estimated superimposed hydrostatic pressure prevents this compression phenomenon [94].

The use of PEEP in patients with ARDS also results in a quantitative decrease in areas of increased density with a concomitant rise in arterial oxygenation [83]. In a study of 22 consecutive patients with severe ARDS, the impairment of arterial oxygenation, the degree of right-to-left shunting and the proportion of dead space ventilation were all correlated with the fraction of

non-inflated tissue, as determined by CT [95]. In the same study it was shown, by analysing the frequency distribution of CT numbers, that increases in PEEP were associated with a concurrent increase in the number of pixels with CT numbers close to air density, the logical conclusion being that PEEP recruits previously collapsed alveoli, thereby increasing the proportion of normally inflated lung tissue. Clearly, the inherent trade-off with PEEP is the potential for overinflation of lung that is normally aerated [94, 96]. The latter phenomenon, combined with its effect on local pulmonary vascular resistance, may in part explain the deleterious response to PEEP in some patients [97]. The ability of CT to quantify alveolar recruitment has also been exploited in recent studies evaluating modern therapies in ARDS. In a study on 21 patients with ARDS, the beneficial effects of inhaled nitric oxide on arterial oxygenation were shown in those cases in whom there was CT evidence of PEEP-induced alveolar recruitment [98]. In a similar way, the quantification of alveolar recruitment on CT has been useful in comparing the efficacy of different forms of mechanical ventilation [99].

Intriguing correlations between indices of lung compliance and the frequency distribution of CT numbers have been made. In 20 patients with acute respiratory failure, compliance varied as a function of the proportion of the normally aerated lung, as judged by CT density measurements [100]. No correlation between the volume of poorly or non-aerated tissue and lung compliance (determined by the pressure-volume curve of lung) was demonstrated. The results of this study indicated that compliance as quantitated by the pressure-volume curve only relates to healthy lung units. Furthermore, it would seem that normally aerated parenchyma on CT represents lung units with normal compliance and has led the authors to challenge the central concept of the "stiff lung" in ARDS [101].

In summary, chest radiography is a relatively insensitive and non-specific guide to monitoring pathological changes in the lungs of the critically ill patient. In practice, however, the daily chest radiograph remains an important tool in the management of cases on the ICU. The advent of CT and particularly fast CT scanners has facilitated the study of patients with ARDS and provided insights into the pathophysiology of the syndrome.

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