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Pulmonary computed tomography and adult respiratory distress syndrome

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Summary

Computed tomography has completely changed the views and interpretation of ARDS, opening a new era in our understanding of the physiological, pathological and clinical aspects of this syndrome. In this brief review we will emphasize the most relevant new knowledge achieved using CT scanning and we will briefly discuss its clinical use in ARDS patients.

Key words: adult respiratory distress syndrome; CT scan

Introduction

For many years, since the original description of the adult respiratory distress syndrome (ARDS) by Asbaugh et al. [1], the ARDS lung was considered homogenously altered by the disease process with a widespread inflammatory oedema. In fact the traditional chest X ray usually shows a diffuse involvement of the lung parenchyma. To our knowledge, the first report of computed tomography (CT) scans in ARDS was by Rommelsheim et al. but due to language problems, this paper had little impact on intensive care management [2]. In 1986, two independent reports, one from the U.S.A and one from our group, described the ARDS findings in CT scans. Maunder et al. [3] reported that the densities were not homogeneously distributed through the parenchyma, being prevalent in the gravity dependent regions of the lung. We observed the same findings [4] and, in addition, we reported the morphological effects of positive end expiratory pressure, which differed dramatically between patients. The use of CT scan refuted the common belief that the pathology of the ARDS lung was a homogenous entity.

CT scan technology

In order to interpret the CT scan it is essential to understand the basic principles of this technology. The CT scan produces a digital image that consists of a square matrix of picture elements, the pixel. Each pixel in the matrix represents a voxel (volume element of the tissue). The CT scan measures the linear attenuation coefficient (μ), which represents the reduction of radiation intensity upon passage through matter, reflecting all types of interactions. The attenuation number depends on several factors, such as the density, the atomic number of the material scanned and the X ray energy. However, using appropriate technical arrangements the X-ray matter interactions can be minimized, so that μ primarily represents the density of the tissue, i.e. the ratio of its mass to its volume. The μ is then converted to a CT number, using the μ of water as reference. Indeed for a given voxel, its CT number is expressed as CT = 1000 × (μ–μ water)/ μ water. The conventional unit of the CT number is the Hounsfield unit (H).

It is evident from the above formula that the CT number of water is zero H. The factor scale of 1000 is used to magnify the small differences of the attenuation coefficient in different tissues. Conventionally the CT number assigned to bone is +1000 H and the CT number assigned to gas is –1000 H. Due to the relationship between CT number and density (see eq. 1) for any given lung region of interest (assuming its average CT number and volume, i.e. the sum of the voxels) it is possible to compute the volume of tissue:

Eq. 1 CT/–1000 = volume of gas / (volume of gas + volume of tissue), from which

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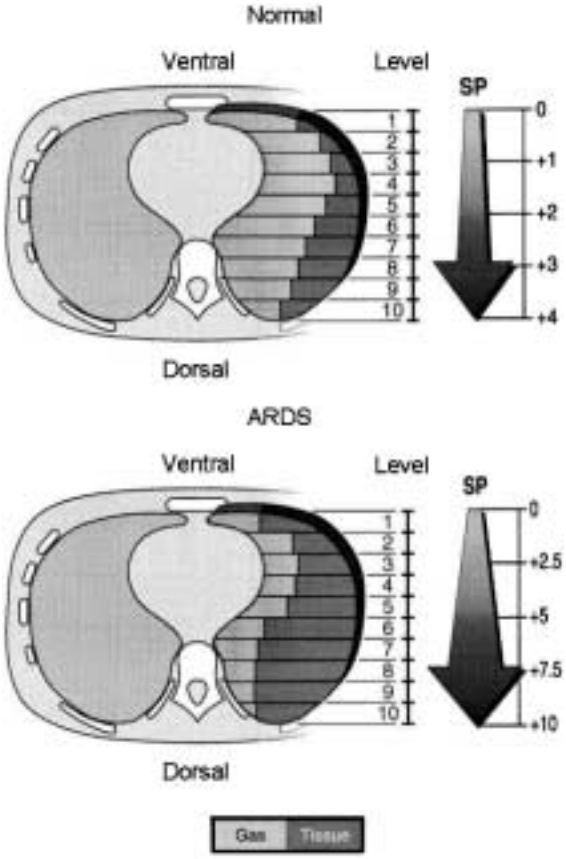

Figure 1

A schematic drawings of the relationship between CT number and lung density.

Eq. 2 Volume of tissue = [1–(average CT/–1000)] × total volume, the total volume being the sum of the tissue volume and the gas volume

Assuming the specific weight of the tissue to be equal to 1, it is then possible to compute the lung weight in any region of interest (figure 1).

Lung compartments

The quantitative analysis of CT scans allows the definition of different lung compartments. To do so it is necessary to know the CT number frequency distribution either in the whole lung or in the region of interest. The ribs and the mediastinum form the borders of the lung parenchyma. Each voxel included in a defined region is characterized by a given CT number. It is then possible to compute the number of voxels included in a predefined CT number interval. For example, in a normal lung 70–80% of the voxels have a CT number ranging from –500 H (i.e. 50% gas and 50% tissue) to –900 H (i.e. 90% gas and 10% tissue). In our first quantitative analysis of CT scans [5], using the CT frequency distribution of the normal lung as a reference, we defined the following compartments: the non inflated compartment which is gasless (CT number between –100 to +100 H), the poorly inflated (CT number between –100 to –500 H), the normally inflated (CT number –500 to –900 H) and the overinflated (CT number between –900 to –1000 H) compartment. Subsequently these cut off points have been used by some but not all authors [6]. Dambrosio et al., for example, used the voxels with CT number between –800 to –1000 H to define the over inflated compartment [7]. When comparing apparently contradictory results it is therefore important to understand the definitions used by the different authors.

Moreover, some confusion in the literature arises from the use of different methods to quantify the compartments. In fact, one method refers to the volume of the compartment i.e. the number of voxels multiplied by the volume of the voxels [8], whereas another method (the one we prefer) refers to the weight of tissue included in that compartment, i.e. volume of compartment × [1–(average CT of the compartment/–1000)] [9]. These two methods of quantification (volume vs. tissue) result in largely differing estimates of the size of the compartments. As an example, in a study assessing the overinflation induced by positive end expiratory pressure (PEEP) in ALI (ALI = acute lung injury)/ARDS, the volume of the over-inflated compartment (i.e. tissue + gas) at total lung capacity in the healthy volunteers was about 30% [10]. However the tissue of the overinflated compartment i.e. the tissue actually exposed to the hyperinflation was only approximately 9% of the total lung tissue.

We believe that referring to tissue, instead of volume, offers advantages, as it is the tissue and not the volume, which is strained and stressed by mechanical ventilation [11].

Anatomical equivalents

It is important to consider what a single voxel includes anatomically. In most ARDS studies an axial thickness of 10 mm was used (matrix size 256 × 256) which corresponds to a voxel volume of 22.5 mm^3 (i.e., 1.5 × 1.5 × 10 mm). This is equivalent to the volume of the acinus at functional residual capacity (16–22 mm^3), which includes about 2,000 alveoli. Approximately 10% of this volume is "tissue" [12]. Thus if the acinus is gasless, its representative volume is about 2 mm^3 and one standard voxel comprises about 10 acini (20,000 alveoli). If the acinus is inflated a voxel will include less alveoli, as an example at total lung capacity one voxel will include about half of the acinus, i.e. 1,000 alveoli.

With the new 16 slice CT scanner it is possible to obtain slices of 0.4 to 0.7 mm thickness. In this case the voxel is almost cubic with a volume of 0.216–0.343 mm^3. At functional residual capacity one voxel will include one percent of an acinus, i.e. 20 alveoli.

Thus, when interpreting the literature, the technical conditions of CT scan acquisition as well as the phase of mechanical ventilation (end expiration or end inspiration) must be kept in mind as they may markedly affect the morphological pattern seen on CT.

CT technique and X ray exposure

In most of the CT scan studies, we and others used 1–3 slices as a representative sample of the entire lung [5, 7, 9, 13]. This approach has been challenged on several occasions by the Rouby group, who used whole lung reconstructions [14, 15].

There is no question that entire lung reconstruction is a more accurate method with a more precise quantification of anatomical regional recruitment [16], however the 1–3 slices approach provides the same basic information and it is hard to find substantial new information in the reports in which entire lung reconstruction was used.

Moreover the X ray exposure must be carefully evaluated. In fact, we must always remember that all lung CT scans imply an X ray dose that is approximately fifteen times higher than a simple slice approach. Indeed we prefer to use less slices in several different pressure, inflation and position situations in order to acquire more functional information than is possible with the whole lung technique, which is usually employed to explore the lung in maximally two such situations.

However the problem of X ray exposure may be attenuated by the introduction of new technologies, such as the high resolution CT scan, in which the radiation exposure can be significantly reduced [17, 18].

Hydrostatic pressure throughout the lung: the superimposed pressure

This analysis assumes that the hydrostatic pressure is transmitted throughout the lung parenchyma, as in a fluid (fluid-like model). It follows that at any given lung level along the vertical axis the pressure is defined as $P = \rho gh$ where ρ is the density, g the gravitational constant and h is the height at which the pressure is estimated. It is then easy to estimate the hydrostatic pressure as the CT number reflects the lung density and the height of the level can easily be measured.

As the lung density in moderate to severe ARDS is two to three times greater than the normal density, it follows that the most dependent regions of the lung undergo a "compressive pressure" which is two to three times greater than normal [19]. Moreover the higher the vertical axes of the lung (ventral to dorsal in supine position), the greater will be the compressive force for the same lung density. Indeed, in ARDS patients the hydrostatic pressure in the most dependent regions of the lung is in the order of 10–15 cm H_2O instead of the 2–4 cm H_2O estimated in a normal lung. The higher hydrostatic superimposed pressure accounts for the tendency of the ARDS lung to collapse in the most dependent regions.

This has been shown both in humans and in experimental animals. In humans we found that the compressive forces are responsible for the inflection point on the regional expiratory pressure volume curve [20], whilst in experimental animals the superimposed pressure was found to correlate highly with the pleural pressure measured directly in the pleural cavity [21]. It is important to note that this model (i.e. heavy lung which collapses under its own weight at the end of expiration) accounts both for the mechanism of PEEP, which represents an end expiratory counterforce against the compressive forces [20], and for the redistribution of density we observed when changing body position from supine to prone [22].

Physiological insight: the baby lung and the sponge lung

ALI and ARDS usually present with near normal inflation in the non dependent regions, ground glass opacification (i.e. a hazy increase in lung attenuation with preservation of bronchial and vascular margins) [23] in the middle regions and consolidation (i.e. increase in lung attenuation that obscures bronchovascular margins in which an air bronchogram may be present) [23] in the most dependent regions. It has also been suggested that ARDS may present as "lobar" ARDS, usually involving the lower left lobe, primarily in patients after major cardiovascular surgery [24]. We have some doubts as to the classification of this kind of pathology as ARDS. In fact ARDS, by definition, should demonstrate a generalised inflammatory oedema of the pulmonary parenchyma.

However, independent of the morphology, most ARDS patients have a variable amount of normally aerated tissue (200–400 g vs 1000–1300 g in a normal man). This has led to the concept of the "baby lung" as this amount of normally aerated tissue found in a normal 4–5 year old boy. Interestingly, the dimension of the baby lung is highly correlated with the compliance of the respiratory system [9]. As a practical rule a compliance of 50 ml/cm H_2O indicates that about 50% of the original lung is normally aerated, while a compliance of 20 ml/cm H_2O indicates that 20% of the original lung is normally aerated and so on. Indeed the respiratory system compliance is not a measure of the non aerated tissue, i.e. that tissue which is not reached by the gas, but it is a measure of the ventilatable tissue. In others words, in ARDS the lung is not "stiff" but "small".

Initially we thought that the baby lung was anatomically located in the non dependent regions. We discarded this interpretation on finding the redistribution of the lung densities in the prone position [22]. In this case the "baby lung" was located in the dorsal regions, not dependent on the prone position. The regional analysis and the computation of the superimposed pressure led to a different model: the "sponge" lung [25]. According to this model the oedema is evenly distributed throughout the lung parenchyma in both dependent and non dependent lung regions. The gravitational forces squeeze the gas from the non dependent regions leading to the appearance of the non dependent baby lung. Indeed the baby lung is more a functional than anatomical entity. Its dimensions

are estimated by lung compliance, which directly measures the normally inflated tissue. The total gas exchange of the patient must take place in this small area of lung. It is important to underline this point. The small baby lung must maintain the gas exchange of an adult patient. Using this model it is quite easy to understand that even a normal tidal volume may be dangerous in ALI/ARDS patients. A normal adult tidal volume distributed to a very small lung leads to unphysiological stresses and strains in the lung regions available for ventilation.

Recruitment

The different appearance on CT scan at the end of inspiration and at the end of expiration provides strong insights into the recruitment and derecruitment phenomena. It is convenient to refer to the acinus as a pulmonary unit [12]. Recruitment may be defined as the increasing number of pulmonary units involved in a new inflation state and/or function. With the CT scan it is possible to measure accurately the recruitment of pulmonary units to a new state of inflation, for example, from a gasless state to a poor normal inflation state.

This technique has allowed us to understand that recruitment is a continuous phenomenon from the end of expiration to full inspiration [6]. This has been documented both in man [26, 27] and in experimental animals [21]. In both cases it has been shown that in the ALI/ARDS lung there is a Gaussian distribution of the opening pressure, which usually peaks between 20–25 cm H_2O [26]. However opening pressures as low as 5 cm H_2O or as high as 40 cm H_2O are also present. Indeed recruitment is an inspiratory phenomena occurring along the entire volume-pressure curve of the lung. The distribution of the opening pressure implies that during a normal tidal volume with a plateau pressure between 20–30 cm H_2O most (but not all) pulmonary units open.

The so called "recruitment manoeuvre" is a technique that allows the opening of pulmonary units in the higher pressure range (30 to 45 cm H_2O). One important concept, not well recognized in the literature, is the "potential for recruitment". This may be defined as the amount of the original lung that may actually be opened at 40–45 cm H_2O of pressure. It is a consistent finding that experimental models such as oleic acid or lung lavage are characterized by huge "potential for recruitment" (i.e. most of the lung may achieve a normal inflation at 40–45 cm H_2O of pressure) [13, 22, 28]. However, in ALI/ARDS of pulmonary origin the case is different. While the "rules" for recruitment are identical to those observed in experimental models, the "potential for recruitment" may be as low as 5% of the original lung. On the other hand, there is some evidence that in ARDS originating from extrapulmonary foci the "potential for recruitment" is greater.

Unfortunately we lack an easy bedside measure of the potential for recruitment. We believe that this issue is clinical relevant. In fact the use of recruitment manoeuvres and high PEEP (see below) may be indicated in patients with high potential for recruitment (figure 2) while their use may be useless or even dangerous in patients with a low potential for recruitment (figure 3).

Derecruitment and positive end expiratory pressure

While recruitment is an inspiratory phenomenon, derecruitment and PEEP are related to the deflation and to the end expiration. It is well established, although not universally accepted, that the intratidal recruitment and derecruitment i.e. end expiratory collapse, may be associated with an in-

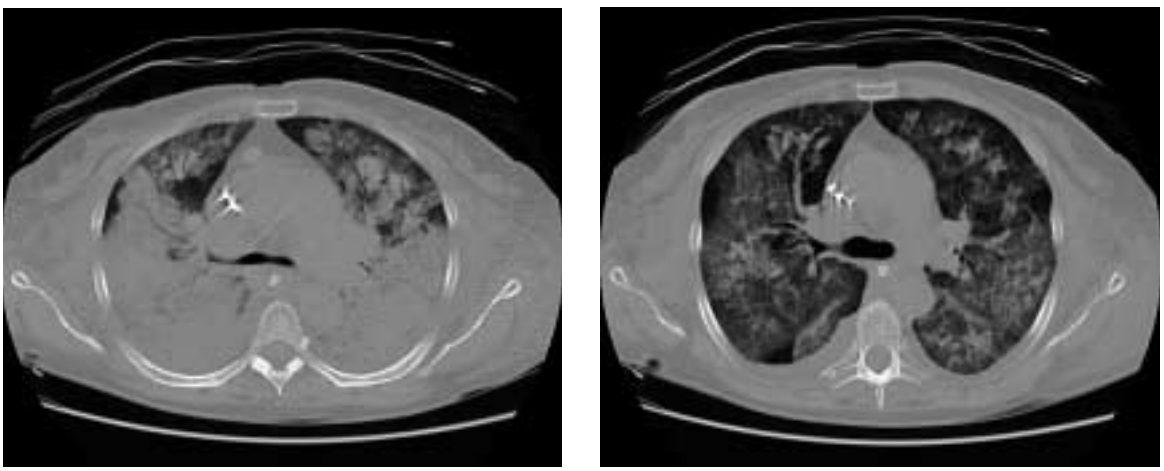

Figure 2

An example of an ARDS patient with high potential for recruitment. a: PEEP of 5 cm H_2O; b: end inspiratory pressure of 45 cm H_2O.

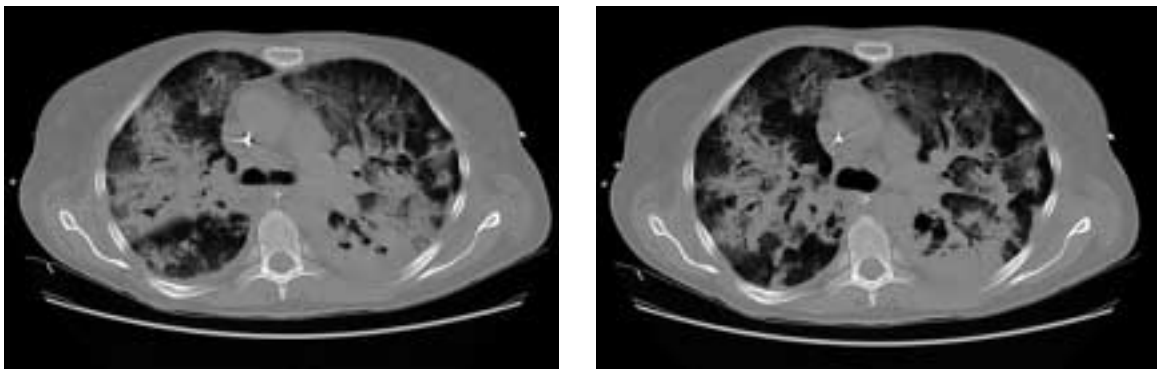

Figure 3

An example of an ARDS patient with low potential for recruitment. a: PEEP of 5 cm H_2O; b: end inspiratory pressure of 45 cm H_2O.

flammatory response of the lung cells and with ventilator induced lung injury (VILI) [29, 30]. Indeed after recruitment, derecruitment should be avoided in order to prevent VILI, as laid out in the open lung approach [31, 32].

We may speculate why some lung regions, recruited during inspiration undergo collapse (derecruitment) at the end of expiration. There are three possible reasons, not mutually exclusive: 1) low ventilation/perfusion ratio of some pulmonary units leads to reabsorption atelectasis [33] 2) surfactant deficit [34] and 3) hydrostatic superimposed pressure is higher than the closing pressure [20, 26].

One of the primary mechanisms of PEEP is to prevent lung collapse by exerting a pressure greater than the superimposed pressure. This mechanism however, leads to an unavoidable side effect. To prevent an end expiratory collapse in the most dependent regions, where the superimposed pressure is 15 cm H_2O, at least 15 cm H_2O of PEEP must be applied. However in most non dependent regions the superimposed pressure is near to zero and therefore these regions, inflated up to 15 cm H_2O of PEEP, are over inflated [35]. Indeed, while PEEP adequate to prevent end expiratory collapse (usually between 15–20 cm H_2O) is effective in the dependent regions, the price to pay is an overinflation in the non dependent regions.

In summary, the CT scan allows us to understand that in ALI/ARDS the lung is not homogeneously affected, and the increased mass of the lung causes dependent regions to collapse. The ARDS lung is not stiff but small, and respiratory system compliance measures the dimension of "baby" lung. Moreover, we have learned that recruitment is a continuous inspiratory phenomena and the prevention of derecruitment may be achieved with PEEP greater than the compressive forces. This is unavoidably associated with overstretch in non dependent lung regions. We believe that further progress will be obtained when the potential for recruitment in every given patient can be clearly assessed.

Clinical use of the CT scan

Soon after the application of CT scan to ALI/ARDS patients it became evident that the information obtained frequently altered the clinical management [36, 37]. According to the largest study published so far in 66% of the patients the CT scan yielded additional information and had a direct influence on treatment in 22% of the patients [38]. Although the indications for using CT scan in clinical practice are not yet completely clear, in our practice we have two main indications:

1) In patients with early ARDS we use the CT scan to investigate the "potential for recruitment" and the response to different end expiratory pressures. We limit the CT scan to two or three slices (apex, hilum and bases) with different ventilatory settings (inspiration, expiration, different PEEP). By imaging only a few levels we are able to keep the total X ray exposure at an acceptable level while acquiring the relevant information. In fact, using different pressure levels we may distinguish between regions of alveolar collapse and consolidation, providing information on the potential for recruitment and consequent ventilator settings.

2) In early ARDS the CT scan is helpful in assessing co-morbid conditions (lung laceration, abscess, etc). Beyond the initial phase we use CT scan to investigate discrepancies between the chest X ray and various clinical and physiological parameters or in the presence of physiological deterioration without obvious reasons. In these cases, we may find pathological alterations requiring immediate intervention such as occult pneumothorax, pneumomediastinum, pneumatocoele or septic emboli.

The bulk of available data strongly suggests that despite the difficulties of transporting critically ill patients to the CT room, as well as the X ray exposure and the significant cost, the rate of detection of unsuspected thoracic changes fully justifies the use of CT scan in ALI/ARDS patients when the bedside X ray examination does not explain the clinical findings.

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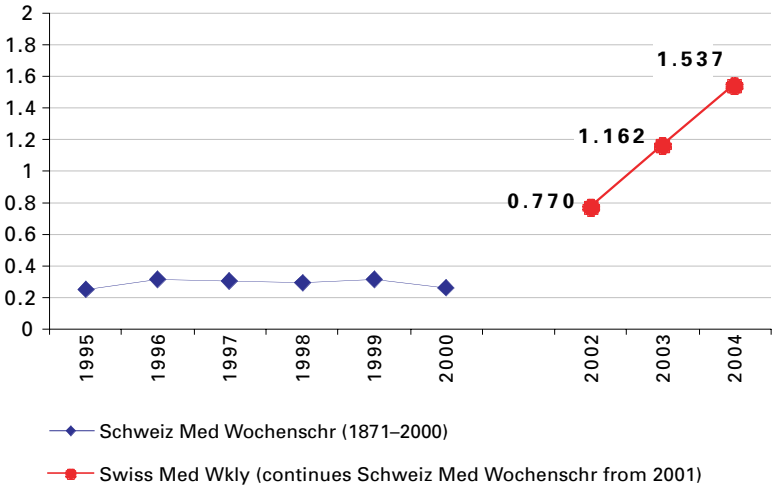

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