Chapter objectives

After studying this chapter you should be able to:

1. Define compliance and hysteresis.
2. Describe why compliance changes in restrictive lung disease.
3. Explain why lung compliance is increased by filling the lungs with water.
4. Understand the relationship between diameter and pressure in bubbles.
5. Explain the problems that might arise because of different sizes of alveoli in the lungs and the development of atelectasis.
6. Explain the significance of the properties of the liquid lining of the lungs and their disturbance in prematurity.
7. Describe how the interaction between the lungs and chest wall produces negative intrapleural pressure.
8. Describe how compliance is measured.
Introduction

One of the properties of the respiratory system most often changed by disease is the ease with which it can be expanded and contracted in breathing.

The lungs and the chest wall that surrounds them are elastic structures, that is, they return to their original shape if a force that is distorting them is removed. The lungs have no muscles capable of changing their shape. The chest, on the other hand, is well supplied with internal and external intercostal muscles which can change its shape, and is separated from the abdomen by the most important inspiratory muscle, the diaphragm. Despite being closely pressed against it the lungs are not attached to the chest wall: there is a space of a few millimetres filled with a slippery plasma-like fluid. Because of this separation we can conveniently deal with the properties of the lungs and chest wall separately, but bearing in mind that in life they work together.

We can use a simple model to describe how changes in the volume of the elastic lungs are brought about by the changes in pressure around them.

The most commonly used model of lung inflation is a toy balloon. Many of the principles that follow can be demonstrated by this model. For example, if you inflate a balloon and prevent the air escaping by blocking the neck with your finger (Fig. 3.1A), the elastic recoil of the balloon will be proportional to its elastance (1/compliance, see below) and will produce a recoil pressure. The pressure inside the balloon will be the same throughout if no flow is taking place into or out of the balloon. These observations demonstrate important principles of lung function.

An even more physiological model of the respiratory system can be made by suspending a balloon in a jar with a piston at its base, like a large syringe (Fig. 3.1B). In this case the balloon represents the lungs, the jar represents the chest wall and the piston represents the diaphragm. Lowering the piston reduces the pressure round the balloon (intrapleural pressure) and causes it to inhale.

Intrapleural pressure ($P_{pl}$)

For an object to be stretched or in some other way distorted it must be subjected to a force. In the case of a three-dimensional object this force may be pressure. In our simple model of breathing (Fig. 3.1A), inspiration would be inflation of the balloon and expiration deflation. The pressure that brings about inflation in Figure 3.1A would be applied to the inside. There is a pressure gradient from inside the balloon to outside. The other, more complicated, way for us to inflate the balloon would be to reduce the pressure outside it using the jar and plunger (Fig. 3.1B): again there is a pressure gradient from inside to outside the balloon, and this is the way we inflate our lungs.

Even when we are completely relaxed, at the end of an expiration with no contraction of the respiratory muscles there is a tension in the thorax between the lungs, whose elasticity is causing them to collapse, and the chest wall whose elasticity is causing it to spring outward. These two structures are ‘locked together’ by the intrapleural fluid in the intrapleural space. Because it is a fluid and therefore not compressible or expandable, and the intrapleural space is airtight, the lungs are as firmly pressed to the chest wall as a suction cup attached to a window.

The tension between the lungs trying to collapse and the chest wall trying to spring out is most clearly seen in surgery, when the sternum is split to allow the surgeon to get at the heart, for example: the lungs collapse and the ribs spring out.

Another way of visualizing what is happening in the space between the lungs and chest wall is to imagine a syringe with two plungers being pulled in opposite directions (Fig. 3.2).

You can see from such a model that intrapleural pressure is negative with respect to atmospheric pressure. What is not immediately obvious is that intrapleural pressure is also negative with respect to air pressure within the alveoli, because the alveoli are connected to the atmosphere by a system of open tubes, the bronchial tree (Fig. 3.3).

This means a hole made between either the atmosphere or the alveoli and the intrapleural space will
allow the pressure surrounding the lung to rise and the lung to collapse: this dangerous condition is called a \textbf{pneumothorax}.

Because the lungs are to some extent suspended from the trachea and rest on the diaphragm, they behave like a child’s ‘slinky’ (a very soft spring), held at one end and supported from underneath. Gravity causes the spring or lungs to slump under their own weight (Fig. 3.4).

Because of this effect the chest behaves as if it is filled with a liquid of the average density of the lungs. As you descend below the surface of any liquid gravity causes pressure to increase at a rate dependent on the density of the liquid. The intrapleural pressure therefore increases (in fact becomes less negative) as you move from the apex to the base of the lung. At the end of expiration the pressure is about $-0.8$ kPa at the top of the lungs and $-0.2$ kPa at the base. That this gradient of pressure depends on the effect of gravity on the contents of the thorax is clearly demonstrated by the fact that it reverses if the subject in which it is being measured stands on their head.

At any instant the negative pressures that surround the lung expand it to a given volume. If those pressures did not change lung volume would not change and we would not breathe. We cause our lungs to breathe by changing the negative pressure around them by making the diaphragm contract and, by acting like the plunger of a syringe, draw air into the chest.

Intrapleural pressure can be measured by inserting a hollow needle between the ribs into the intrapleural space. It is not easy to obtain volunteers to undergo this procedure, and as we are usually more interested in changes in intrapleural pressure than their absolute values, we frequently measure changes in pressure in the oesophagus, which forms a very flexible tube running through the thorax. Because the oesophagus is so flexible the changes of pressure within it closely follow changes in intrapleural pressure.

**Static lung compliance ($C_L$)**

We have seen that the lungs are elastic structures, i.e. they return to their original shape and size when distorting forces are removed. These distorting forces are usually those of intrapleural pressure, which becomes more negative to bring about inspiration and then becomes less negative, and the elasticity of the lungs leads to quiet expiration. This elasticity is a measure of how easily the lungs can be stretched and is
conventionally expressed as **compliance**, the reciprocal of elastance. This ‘stretchiness’ of the lungs can be measured under static conditions, i.e. by measuring pressure and volume when there are no breathing movements taking place, or as **dynamic compliance** during breathing (see below).

### The effect of disease

The compliance of the lungs is changed by most lung diseases. Such changes have a detrimental effect on lung function and increase the work of breathing. It seems that healthy lungs are at optimal compliance, and an increase or decrease from this norm is for the worse. For example, in lung fibrosis the lungs are stiffened by the laying down of collagen and fibrin bundles, so that compliance is reduced. In emphysema the parenchyma of the lungs is destroyed, there is less elastic recoil, and compliance is therefore increased. In infant respiratory distress syndrome it is the nature of the liquid lining the lungs that is at fault (see below), and this also reduces lung compliance. The origin of these changes becomes clear if we consider the two physical systems that contribute to the elasticity of the lungs and hence their compliance. One originates in the elasticity of lung tissues, the other depends on the nature of the liquid lining of the alveoli.

### The physical basis of lung compliance

The elastic properties of the lungs, and hence their compliance, depends almost equally on the elastic properties of their tissues and on the elastic properties of their liquid lining.

### Elastic properties of the respiratory system Box 3

**Respiratory distress syndrome of the newborn**

Mrs Aldridge had been expecting her first child in about 12 weeks’ time. Nevertheless, she had gone into labour and had given birth to a baby boy. The paediatrician examined Mrs Aldridge’s son after he was born. It soon became apparent that the baby was breathing very rapidly and appeared to be having some difficulty: his chest was indrawing with each breath and he was making a grunting sound. The paediatrician made the diagnosis of respiratory distress syndrome (RDS) of the newborn.

In this chapter we will consider:

1. The causes of respiratory distress syndrome of the newborn.
2. Treatment and prevention of respiratory distress syndrome of the newborn.

### Elastic properties of the respiratory system Box 2

**Causes of RDS of the newborn**

The principal cause of RDS of the newborn is a deficiency of lung surfactant related to prematurity, although the disease is also related to the general immaturity of a premature baby’s respiratory system. The more premature an infant, the more likely it is to develop RDS.

The type II pneumocytes that produce surfactant develop at about 24 weeks’ gestation, although most fetuses do not start producing large amounts of surfactant until about 34 weeks (babies are born, on average, at 40 weeks). Premature babies also have smaller lungs and alveoli than full-term babies. Remember the law of Laplace, which states that:

\[ P = \frac{2T}{r} \]

where \( P \) is the pressure inside a bubble, \( T \) is the surface tension and \( r \) is the radius. In the alveoli of premature infants \( T \) is greater than normal because of the lack of surfactant, and \( r \) is less than normal because the premature infant has smaller alveoli. For both these reasons, a high pressure \( (P) \) is needed to keep the alveoli open. This means that the lungs tend to collapse during expiration, and the effort of inspiration is very much increased. Furthermore, the lack of surfactant means that fluid tends to be drawn from the blood into the alveoli, which therefore become oedematous. All these things mean that the dynamic compliance of the lungs is very much decreased.

Because it has not developed fully the compliance of a premature infant’s chest wall is high, and this means
The elasticity of lung tissue

It might be reasonably assumed that the elastic properties of the lungs are due to the yellow elastin fibres of the lung parenchyma, the fibre type that gives most other organs their elasticity. In fact, only about half the elastic recoil of the lungs comes from the elastin fibres in the alveolar walls, bronchioles and capillaries. Also present in the lungs are collagen fibres, which are less easily stretched and limit overexpansion of the lung. The elastin fibres act in a rather complicated way to provide elasticity. The fibres are kinked and bent round each other, and during inspiration unfold and rearrange in a manner that has been likened to the straightening of the fibres of a nylon stocking when it is put on (Fig. 3.6).

In the lung disease emphysema the elastin fibres are degraded and therefore do not recoil so strongly. The lung is easier to inflate; it is more compliant.
In the disease lung fibrosis there is an increase in the elastin and collagen of the interstitial tissues and the lung recoils more strongly, i.e. it is less compliant.

**Treatment and prevention of the newborn**

Baby Aldridge was initially given oxygen by the paediatrician. An endotracheal tube was then inserted into his trachea, he was placed on a mechanical ventilator and his lungs were ventilated artificially. Artificial surfactant was administered down the endotracheal tube in order to treat the deficiency of natural surfactant. Ventilation was continued for 1 week, during which time Baby Aldridge continued to improve. After this time, the endotracheal tube was removed and he was able to breathe for himself.

Mechanical ventilation is usually required in infants with RDS. Ventilation needs to be carried out very carefully, however. This is because of the low compliance of the infant's lungs, which means that relatively high airway pressures are needed to achieve an adequate tidal and hence minute volume. High airway pressure can cause trauma to immature lungs and airways and can cause a pneumothorax. Furthermore, high inspired oxygen concentrations (greater than 50% oxygen) can also cause lung damage. The damage is done by oxygen free radicals, which are toxic molecules, including the hydroxide ion and the superoxide ion, each of which has an unpaired electron, making it very reactive. High oxygen concentrations favour the production of these molecules. The lung damage that can be caused by high airway pressure and high oxygen concentrations can lead to a chronic lung condition called bronchopulmonary dysplasia.

In addition to artificial ventilation of the lungs, RDS is treated with surfactant; indeed, it is common practice to administer surfactant to premature babies in order to try and prevent RDS from occurring. Both natural surfactant obtained from animal lungs and artificial surfactant containing dipalmitoyl phosphatidylcholine can be used. The administered surfactant acts in the same way as endogenous surfactant and reduces surface tension in the alveoli, stabilizing them and increasing lung compliance.

It is also possible to try and prevent RDS where a birth is expected to be premature, for example in the case of a fetus that is not growing adequately in the uterus and whose birth is to be induced. Administration of steroids to the mother increases the rate of maturation of the fetal lungs and reduces the likelihood of RDS occurring in the newborn infant.

With modern treatment survival rates in babies affected by RDS are good, particularly in older premature babies.

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The liquid lining of the lungs

So, about half the elastic recoil of the lungs comes from the elastic properties of their tissues, just as there is recoil in an inflated rubber balloon. The remaining half of the elastic recoil of the lungs comes from their unique structure of millions of tiny bubble-like alveoli, lined with liquid and connected to the atmosphere by a series of tubes (the bronchial tree). The importance of this structure was demonstrated by von Neergaard, in 1929, when he showed that an isolated lung completely filled with water is about twice as easy to inflate as one filled with air. The cause of this change in ease of inflation lies in the removal of the air–liquid surface that lines the millions of spherical bubbles surrounded by lung tissue by filling the air space with water to form one single small air–liquid interface somewhere in the trachea (Fig. 3.7).

![Air–liquid interface](image)

**Fig. 3.7** Change in the area of the air–lung interface produced by filling the lungs with water.

To gain approximation of the change imagine an enormous bottle with a very narrow neck. When filled with air it has a large internal surface area exposed to
the air; when filled with water right up to its neck this surface area is greatly reduced. To complicate matters, the internal surface of the alveoli is curved, which has a significance we will deal with later.

Not only does filling a lung with fluid make it easier to inflate, it also abolishes the hysteresis seen in the normal lung. Hysteresis (Greek, hysteron, to lag behind) means that inflation of the lung follows a different pressure/volume relationship from deflation (Fig. 3.8). It requires a greater pressure to reach a particular lung volume when you are inflating it than to hold it at that volume when you are deflating it. Something is ‘propping the lung alveoli open’. These peculiar changes are due to the nature of bubbles and the properties of the liquid that makes up the liquid lining of the lung. We need to consider the nature of the surface of liquids, the nature of bubbles and the very special properties of the liquid lining of the alveoli to understand static and dynamic compliance of the lungs.

The surface of liquids

That liquids form a clear boundary between themselves and the air above them could almost be a definition of a liquid. That this boundary is under tension or stress is clearly seen in the liquids we come across in everyday life: the surface of a cup of coffee, if touched lightly with a spoon, seems to leap up to the spoon. This is the effect of surface tension (Fig. 3.9).

The ‘skin’ or surface of a liquid exists because at the surface there is an imbalance of the forces acting on the molecules at the surface, as explained in the Appendix (p. 173).

Just as mechanical and chemical systems move to a state of minimal energy (maximum entropy), so surfaces seek minimal energy and hence minimal area (this is why water droplets are spheres, the shape that has minimal surface for a given mass). The tendency to reduce in surface area produces tension in a liquid surface which can be measured by a surface balance (Fig. 3.10). In this, a bar of metal dips into the liquid and is exposed to the same forces as act on molecules at the surface of liquids, or the coffee spoon. These forces can be measured by a sensitive transducer. The total force will depend on the surface tension and the length of the bar, and the units of surface tension are therefore N m⁻¹. The surface balance was modified to produce the Wilhelmi Balance by the ingenious addition of a movable barrier that can compress the surface of the liquid in the balance trough. Under these circumstances the depth of the liquid alters as the barrier is moved, but this doesn’t matter as it is the air–liquid interface that is important. If pure water is placed in a Wilhelmi Balance, moving the barrier to and fro, expanding and contracting the surface, has no effect on the measured surface tension. If, however, a phospholipid of the type found lining the alveoli of the lungs is placed on the surface of the liquid it spreads out to form a layer between water and air, and the
surface tension changes as the barrier advances, forcing the phospholipid molecules closer together, or retreats allowing them to separate. The tension reaches a minimum when all the phospholipid molecules are neatly packed as a single layer on the surface. When the surface area is reduced further the molecules pile up on each other and the tension begins to rise again. It is possible to measure the size of the molecules of the phospholipid from the area of the surface at which the surface tension is minimal.

The nature of bubbles

Anyone who has indulged in the congenial occupation of blowing soap bubbles using a child’s pipe or wire loop will have demonstrated most of the basic physical principles underlying elastic recoil of the lung due to its liquid lining.

They will have noticed that once a complete sphere has formed the bubble is stable. While there is a hole in the bubble, however, through the pipe or wire loop, if you stop blowing the bubble immediately collapses, returning to a flat layer of soap stretched across the pipe or loop. The molecular basis of this effect is explained in the Appendix (p. 170).

The same forces that produce this collapse are at work in the liquid lining the alveoli of the lung. The fact that the outer surface of the ‘bubble’ is in contact with lung tissue does not alter the tendency of this bubble to collapse, driving air out through the ‘hole’ which is its connection with the bronchial tree.

The stable, completely spherical, bubble you blew with your wire loop was held inflated by an excess of internal pressure. The relationship between this excess pressure, the surface tension of the liquid of the bubble and the radius of the bubble is described by the Laplace Relationship

\[ P = \frac{4T}{R} \]

where \( P \) is the excess pressure (Pa), \( T \) is the surface tension (N m\(^{-1}\)) of the liquid making up the bubble, and \( R \) is the radius (m) of the bubble. The constant 4 appears in this equation because a bubble has two surfaces exposed to the air. For alveoli whose outer surface is in contact with the lung tissue it becomes:

\[ P = \frac{2T}{R}. \]

Human alveoli are about 0.1 mm in diameter; if they were lined with interstitial fluid the pressure required to hold them open (the excess pressure inside them above the surrounding intrapleural pressure; see Chapter 3) would be 3 kPa. This is more than twice the pressure found in normal individuals. The liquid lining of the lungs must therefore be very different from interstitial fluid, with a significantly lower surface tension.

The nature of the liquid lining of alveoli

From the Laplace Relationship we can see, somewhat surprisingly, that the pressure in a small bubble can be expected to be greater than the pressure in a large one. This might lead us to anticipate problems in the lungs, as there exists a whole variety of sizes of alveoli, those at the top being larger than those at the bottom (see Chapter 00). Under these circumstances an unstable situation might be expected to arise, with small alveoli (containing higher pressure) emptying into large ones (containing lower pressure) (Fig. 3.11A). The nature of the liquid lining of the lungs provides an ingenious solution to this problem.

The liquid lining of the lungs can be extracted by washing them out with saline (bronchial lavage). The washed-out liquid can then be investigated in a Wilhelmi Balance, where it shows some interesting and useful properties. Adding the extract to water in the balance reduces the surface tension from 70 to \( 40 \times 10^{-3} \) N m\(^{-1}\). Surface tension falls even further when the surface is compressed, reaching a minimum below \( 10 \times 10^{-2} \) N m\(^{-1}\). This effect is due to surfactant secreted by type II cells of the alveolar epithelium.

The surfactant spreads over the inner surface of the alveoli and into the bronchioles. It is made up of dipalmitoyl phosphatidylcholine: the structure of this and the way it arranges itself at an air–liquid interface is shown in Figure 3.13.
The straight structure of these molecules enables them to pack more closely during expiration than would other shapes. This packing and unpacking during inspiration and expiration causes surface tension to decrease and increase in a manner that produces the characteristic hysteresis of the lungs. The change in surface tension also provides the solution to the problem posed earlier of the alveoli of different sizes containing different pressures, and the tendency for the small to empty into the large (Fig. 3.11A). If the surface tension in the large alveolus is sufficiently greater than the tension in the small the effect of the larger radius (R) in the Laplace Relationship

\[ P = \frac{2T}{R} \]

will be matched or even overpowered by changes in tension (T).

And this is what happens in the lung. The surface tension changes to match the radius, so that all alveoli contain about the same air pressure and the small do not tend to empty into the large (Fig. 3.11B).

The air pressure inside an alveolus not only resists the effects of surface tension causing the alveolus to collapse, but also resists exudation of fluid from pulmonary capillaries into the alveolar space. If surface tension is reduced at any particular air pressure within an alveolus more of that air pressure will be available to resist exudation and prevent pulmonary oedema.

The presence of surfactant is clearly important to normal lung function. It:

- reduces surface tension and therefore elastic recoil, making breathing easier
- reduces the tendency to pulmonary oedema
equalizes pressure in large and small alveoli
produces hysteresis, which ‘props’ alveoli open.

This important substance appears rather late in human embryological development, at about 30 weeks, and fetuses above that age which are not producing surfactant are stimulated to do so by the administration of corticosteroids.

### The opening and closing of alveoli

Even in an isolated lung the relationship between pressure and volume is complicated by the interaction of the surface-active forces already dealt with and the elastic tissue of the lung. One of the most important aspects of this is the tendency for alveoli to close at low lung volumes despite the assistance from surfactant to stay open.

Range 1 of Figure 3.8 is the result of alveoli staying shut despite increased inflation pressure.

Range 2 begins at about 1 kPa, when alveoli begin to pop open and the lung inflates with very little increase in pressure.

Range 3 is where tissue elasticity, particularly from collagen fibres, stiffens the lung.

Range 4 is deflation, where hysteresis of surfactant ‘props open’ the alveoli.

Because of the pressure of overlying tissue the alveoli in the lower parts of the lung are always smaller than those at the top (except at total lung capacity), and therefore show a greater tendency to collapse. The lung volume at which the airways in the lower part of the lung begin to close (Range 1) is known as closing volume. This has considerable significance in life, as regions of the lung closed off from the atmosphere are functionally useless. In young people closing volume is less than functional residual capacity (FRC) and all is well, but by the average age of 66 closing volume equals FRC, with the unfortunate consequence of increased airway closure and reduced ventilation of the lower lung.

### Static compliance

In respiratory physiology compliance is defined as the change in volume produced by a change in pressure across the wall of the structure being investigated: the lungs alone, the lungs and chest wall, or the chest wall alone (which is very rarely measured).

\[
\text{Compliance} = \frac{\text{Change in volume}}{\text{Change in pressure}}
\]

For the lungs alone, in life at least, the appropriate pressure measurement is from the alveoli to the intrapleural space. Change in volume can easily be measured using a spirometer, and as we are interested in measuring change in pressure we can use the change in pressure in the oesophagus within the chest as an indication of the change in pressure of the intrapleural space, which is difficult to measure in subjects or patients. Intraoesophageal pressure is usually measured by introducing a small air-filled balloon attached to a pressure transducer by a catheter into the oesophagus via the nose. Because intrapleural pressure varies with the position in the chest it is usual to standardize the position of measurement by pushing the balloon in 30–35 cm from the tip of the nose.

Measurement of total compliance (lungs + chest wall) is much easier because in this case the appropriate pressure measurement is from alveoli (or mouth pressure under static conditions) to atmosphere.

Neither pressure nor volume has the dimensions of time, and therefore these measurements can be made under static conditions where the subject breathes in slightly, relaxes his respiratory muscles while measuring mouth pressure with a transducer; he then breathes in a known volume and again relaxes against the transducer. The slope of the pressure–volume graph so formed is his total static compliance (Fig. 3.15).

The slope of the line that makes up the loop in Figure 3.15 is the compliance of the lungs, which in this case have been taken out of the body. This loop
describes the condition of the lungs from total collapse to maximum volume.

In normal breathing there are about 3.0 L of air left in the lungs at the end of expiration (FRC), and we seldom inhale to total lung capacity (TLC). Most quiet breathing takes place within the shaded area of Figure 3.15, which shows less hysteresis than the total collapse to total volume manoeuvre, but still shows that compliance reduces due to airway collapse when residual volume (RV) is approached.

The lung compliance of an average healthy young male is about 2 L kPa⁻¹. That of a female is slightly greater, even taking into account the fact that lung compliance depends on lung size, and therefore body size. This effect is taken into account by measuring specific lung compliance (sCL), which is compliance divided by maximal lung volume.

The nature of specific compliance can be visualized if you consider the inflation of one or more balloons by a pump that provides as much air as you like, but only to a maximum pressure of 1 kPa above atmospheric.

If you connect a single balloon to the pump and it inflates 2 L the balloon has a compliance (volume increase/pressure) of 2 L kPa⁻¹ (Fig. 3.16a).

If two balloons are connected to the pump they will both be subjected to a pressure of 1 kPa and increase in volume by 2 L each (4 litres in all), which gives the system a compliance (volume increase/pressure) of 4 L kPa⁻¹ (Fig. 3.16b).

**Fig. 3.15**
The pressure–volume relationship of an excised lung (large loop) compared with that of quiet breathing in the intact situation (shaded loop). In the intact situation the lungs start from a partially inflated state at FRC and there is little hysteresis. These curves were obtained under static conditions.

**Fig. 3.16**
The nature of specific compliance.
The pump with an unlimited supply of air but delivering a fixed pressure will inflate (a) one or (b) any number of identical balloons, each to the same volume. The compliance (change in volume/change in pressure) of these two systems is very different.

**Dynamic compliance**
The ‘static conditions’ referred to above and in Figure 3.14 consist of the lung neither inhaling or exhaling while pressure is being measured. These no-flow conditions only occur at two points during normal breathing: at the peak of inspiration and at the trough of expiration (Fig. 3.15).

This fact can be used to measure compliance while the subject is breathing – **dynamic compliance** – by measuring intrapleural pressure and lung volume at the same time (Fig. 3.17a). Alternatively, these two variables can be displayed as a loop (Fig. 3.17b), and in this case the angle of the long axis of the loop (volume/pressure) represents dynamic compliance. In this case the area of the loop represents the **work of breathing**.

In healthy lungs the ratio between dynamic compliance and static compliance is fairly constant at all frequencies of breathing. In obstructive lung diseases such as asthma the obstructed areas of the lung do not
have sufficient time to fill or empty, a condition which worsens at high frequencies of breathing, resulting in a fall in the ratio as breathing frequency increases.

**The thoracic cage**

The term chest wall compliance ($C_W$) is frequently used to describe what should be called thoracic cage compliance, because the diaphragm and the abdominal contents pressing on it represent an important element of this component of respiratory mechanics which, unlike the lungs, contains no element of surface tension.

At the end of expiration the lungs do not collapse totally because the thoracic cage is holding them out in a slightly expanded condition. This means that the thorax is slightly pulled in. Because of this the elasticity of the thorax initially helps inspiration. The thorax reaches its neutral position at about two-thirds vital capacity, and after that the direction of its elastic forces is in favour of expiration (Fig. 3.18).

Perhaps surprisingly, considering their very different structures, the compliance of the thoracic cage is about the same as that of the lungs (2 litres kPa$^{-1}$). This truly elastic measurement is very difficult to make as it can only be properly measured when the respiratory muscles are totally relaxed – a very difficult thing for the conscious subject to do and best obtained in the anaesthetized patient.

Just like in the lungs, thoracic cage compliance is influenced by disease, and perhaps even more than the lungs by posture and position. Ossification of the costal cartilages, and scars resulting from burns of the chest, reduce compliance. The diaphragm passively transmits intra-abdominal pressure from obesity, venous congestion and pregnancy, and by this mechanism can reduce the static compliance of the total respiratory system by up to 60% as a result of changes in posture and changes from the supine to the prone position.

**Total compliance ($C_{tot}$)**

Because the lungs fit inside the thorax, rather like the inner tube inside a tyre, they must be treated as elements in parallel rather than in series when their properties are added together (Fig 3.19).

We can see that the pressure gradient for both the lungs and chest wall is from the intrapleural space to the atmosphere (or very nearly atmosphere, in the case of the lung alveoli). They are therefore in parallel with each other in terms of pressure gradients. In adding
Elastic properties of the respiratory system

Lung and chest wall compliance to give total compliance we must therefore use the relationship appropriate for parallel structures:

\[ \frac{1}{C_{\text{tot}}} = \frac{1}{C_L} + \frac{1}{C_W} \]

Because the compliance of the lungs and that of the chest wall are about the same (21 kPa\(^{-1}\)), an artificial ventilator needs to apply twice the normal change in intrapleural pressure to the air in the lungs of a paralysed patient to produce the normal volume change.

Factors affecting lung compliance

- **Lung size** The principles of calculating specific compliance (see above) tell us that although a man has a greater compliance than a mouse, this is due to the difference in the amount of lung being inflated.

The lung volume at which compliance is measured is a different effect from that of the amount of lung present, and also exerts an effect.

- **Recent pattern of breathing** Compliance is affected by breath-holding and recent pattern of breathing, probably owing to redistribution of air in the lungs, closing and opening of alveoli, stress relaxation and changes in circulation.

- **Age** Because a large part of lung compliance is due to a surface tension effect that does not age, there is little effect of healthy ageing on compliance.

- **Posture** The changes in lung compliance seen with posture are probably due to the effect of posture on lung volume.

- **Disease** Most diseases of the lung – congestion, fibrosis, consolidation, respiratory distress syndrome – decrease its compliance. Emphysema is unique in that static compliance is increased owing to the loss of lung tissue. Even in emphysema, however, dynamic compliance is decreased because of the disordered distribution of ventilation.

In asthma there is no change in compliance: the pressure–volume relationship of the lungs is moved bodily upward without any change in slope.

Further reading


Haddad DF, Greene SA, Olver RE Core paediatrics and child health. Edinburgh: Churchill Livingstone, 2000


Bangham AD. Lung surfactant: how it does and does not work. Lung 1987;165:17


Self-assessment case study

A 26-year-old man is in an intensive care unit being treated on a mechanical ventilator for acute respiratory distress syndrome (ARDS). This is a different condition from the infant respiratory distress syndrome that we have already looked at in this chapter, but the two conditions share a number of features.

ARDS is characterized by inflammation of both lungs that results in a failure of gas exchange and a reduction in lung compliance. It is a rare complication of a number of disparate conditions, including pneumonia, lung trauma and sepsis.

In order to ventilate this patient’s lungs, his ventilator needs to deliver airway pressures that are much higher than normal. To ventilate the lungs of a healthy patient typically requires airway pressures in the order of 20 cmH\(_2\)O. The ventilator in this case was set to a maximum of 40 cmH\(_2\)O and it frequently occurred that this maximum pressure was reached. The high airway pressure was required because of the low compliance of the patient’s lungs and was causing a great deal of concern to his doctors.

From your knowledge of lung compliance you should be able to attempt the following questions:

1. What do you understand by the term ‘low compliance’?
2. Why is the pressure needed to inflate the patient’s lungs higher than normal?
3. Why do you think there is a maximum pressure set on the ventilator?
What effect do you think a low compliance will have on the tidal volume that a ventilator can deliver?
What effect will this have on gas exchange?

Answers see page 177

Self-assessment questions
1. Describe the origins and nature of intrapleural pressure.
2. Define lung compliance.
3. What is the origin and function of pulmonary surfactant?
4. To what and in what proportion is the elastic recoil of the lungs due?
5. A patient has one of his lungs removed. Assuming both lungs were the same volume and of the same mechanical properties, what would happen to his measured compliance? How does the concept of specific compliance address this problem?

Answers see page 177