

Anders LARSSON

Gentofte University Hospital
Copenhagen University
Hellerup, Denmark

Saturday May 31, 2003

Euroanaesthesia 2003 - Glasgow

Lung mechanics deal with the forces which are needed to move air in and out of the lungs. However, these forces are also due to the condition of the chest wall and I will therefore discuss both the mechanics of the lungs and the chest wall, i.e., the respiratory mechanics.

The forces are needed to counteract 1) the resistance to airflow in the airways and 2) the elastance of the lungs and of the chest wall. To be able to describe these forces it is necessary to measure 1) airflow — lung volume change, 2) pressure in the airways and, 3) an estimate of the pleural pressure, usually from esophageal pressure

Respiratory mechanics can be measured either under “no flow — static” or dynamic conditions.

“STATIC” MEASUREMENTS

Static measurements are usually made by slow or interrupted insufflations of air into the lungs under simultaneous registration of airway (and sometimes esophageal) pressure and volume changes. From these registrations a static pressure-volume (PV) curve or loop is constructed (figure 1).

The purpose of measuring under “static” conditions is to decrease the influence of resistance (which is present during airflow) as much as possible, in order to examine the elastic forces of the respiratory system. However, true static conditions are not possible to achieve because of the visco-elasticity of the lungs and the effect of gas exchange.

A PV-loop can be divided in an inspiratory and an expiratory part. The inspiratory part can, at least under pathological conditions, be divided into three parts: 1) a part at low pressures with low compliance (volume change/pressure change), 2) a part at higher pressures with high compliance, and 3) a part with low compliance at the highest airway pressures. The “knee” between 1) and 2) is commonly called “lower inflexion point (LIP)” or “Pflex”, and the “knee” between 2) and 3) the “upper inflexion point (UIP)”. The expiratory part may be divided in two parts: 1) a part with low compliance at the high airway pressures, and 2) a part with high compliance at somewhat lower airway pressures. The “knee” between these two parts is named “collapse pressure (CP)”.

FIGURE 1

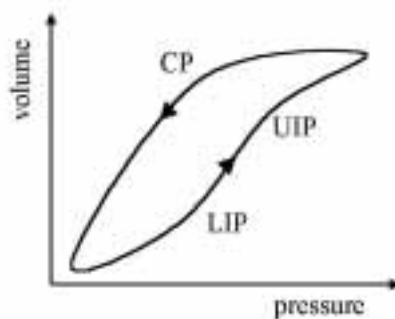


Figure 1. A static pressure volume loop obtained during slow insufflation and exsufflation of the lungs. LIP = lower inflexion point, UIP = upper inflexion point, CP = collapse pressure

MYTH

Lower inflexion point represents the pressure at which all lung regions are open and is therefore useful when setting PEEP.

REALITY

Probably not.

Originally, it was thought that LIP represented the end of lung recruitment process (all lung regions are open), the part of the curve between LIP and UIP distension of the lungs and the part at higher pressures than UIP overdistension. However, LIP does not represent the end of lung recruitment process, but instead the start of the lung recruitment process [1,2]. The part of the curve between LIP and UIP represents both lung recruitment (which gives a high compliance) and distension of already open lung regions [1,2,3,4,5]. Furthermore, LIP is dependent on the compliance of the chest wall and particularly on the compliance of the abdominal-diaphragmic part of the chest wall [6]. This is in its turn dependent on the intra-abdominal pressure. In fact, in patients with abdominal compartment syndrome, LIP may only indicate the level of intra-abdominal pressure [Manu Malbrain, personal communication]. Also, if the patient has an obstructive lung disease or is intubated with an endotracheal tube with a small inner diameter, and not enough time is given for expiration before an inspiratory PV-curve is registered, LIP might only show the level of auto-PEEP [7]. Moreover, if the flow rate during insufflation is too high, the pressure at LIP only represents the pressure needed to overcome the flow resistance in the endotracheal tube and in the airways and not the start of lung recruitment.

Hence, LIP does not theoretically indicate the pressure for optimal PEEP [8]. However, it might incidentally show the same pressure as at the collapse pressure (CP). CP is the pressure where the lungs start to lose volume that in collapse-prone lungs is caused by de-recruitment. Ideally, if 'the open lung approach' is considered, PEEP should be set by using the pressure at CP [9].

MYTH

Upper inflexion point indicates the pressure at which overdistension occurs.

REALITY

Overdistension occurs also at lower pressures.

Upper inflexion point (UIP) shows the pressure where compliance starts to decrease and was therefore thought to indicate the pressure at which overdistension starts. However, overdistension of a lung unit is due to a high local trans-pulmonary pressure. Because there is a gradient of increasing trans-pulmonary pressures from dependent to non-dependent lung regions, the local trans-pulmonary pressures in the non-dependent regions could be quite high causing overdistension already at airway pressures much lower than UIP [10].

MYTH

Upper inflexion point shows the end of the lung recruitment process.

REALITY

To recruit the most severely collapsed lung regions, higher airway pressures are needed.

Upper inflexion point (UIP) is usually located at airway-pressures of 25-30 cmH₂O. Rothen and coworkers have shown that an airway pressure above 40 cmH₂O is needed in cardio-pulmonary healthy patients to open up anesthesia-induced atelectasis [11]. In patients with ARDS even a higher pressure is necessary to achieve adequate lung recruitment [12] and sometimes a pressure as high as 60 cmH₂O is required [13]. Thus, UIP does not indicate the pressure at which recruitment ends.

MYTH

Esophageal pressure is equal to the intra-pleural pressure.

REALITY

The intra-pleural pressure is different in different parts of the pleura, and the esophageal pressure might at best represent the intra-pleural pressure at the point of measurement.

Esophageal pressure, measured by use of a balloon in the lower esophagus, has been considered as an adequate estimate of the pleural pressure. There are several problems with esophageal measurements. First, to get a correct value, the balloon has to be filled with a very small volume of air and if the balloon is over-inflated, the pressure is overestimated. Second, the balloon has to be located just above the diaphragm. Due to the technique to evaluate the position of the balloon, this might be difficult to achieve when the patient is artificially ventilated. Third, even if the above procedures are made correctly, the measurement might not reflect the pleural pressure. When the patient is positioned supine, the mediastinal structures will compress the esophagus and the pressure in the esophagus will be positive at zero end-expiratory airway pressures [14,15]. If this pressure would be similar to the local pleural pressure, the local trans-pulmonary would be negative indicating atelectasis formation. However, in normal cardio-pulmonary healthy spontaneously breathing subjects atelectasis is not formed just by the supine position. Although the absolute values of esophageal pressures might not correctly represent the absolute pressures in the pleural space in the supine position, there are strong indications that the changes in esophageal pressure with ventilation or pressure-volume maneuvers adequately reflect the changes in pleural pressures [4].

MYTH

Infants have lower lung compliance than adults.

REALITY

This is true only if the absolute volume is considered, but not if compliance is related to total lung capacity or functional residual capacity (specific compliance).

The lower the lung volume, the lower is the lung compliance. However, this indicates only that the increase in pressure for a specific volume is more in a small lung than in a large lung and not anything about the elastic structures of the lung. An infant has lower lung compliance, but if this is related to the size of the lungs, an infant has higher lung compliance than an adult suggesting that an infant's lung is less elastic [16].

MYTH

A low chest wall compliance indicates that a low compliance of the respiratory system is due to extra-pulmonary conditions.

REALITY

Even if the primary problem is a stiff chest wall, a low compliance of the respiratory system is usually due to both the chest wall and the lungs, particularly when a low chest wall compliance is caused by a high intra-abdominal pressure, compressing the diaphragm.

Chest wall restriction will reduce the size of the lungs and cause atelectasis. For example, when the intra-abdominal pressure increases, the diaphragm is pushed up in the thorax at end-expiration, producing atelectasis in dependent and basal lung regions. The atelectasis formation decreases the size of the lungs and thus also the compliance of the lungs. Hence, the decreased compliance of the lungs will contribute to the reduced compliance of the respiratory system under these conditions.

DYNAMIC MEASUREMENTS**MYTH**

Dynamic ventilator loops are useful for evaluation of lung function.

REALITY

The loops reflect the dynamic pressure-volume (PV) relations of the ventilator tubing and the endotracheal tube, and only in a lesser extent of the lungs.

The pressure shown on ventilator monitor is commonly measured in the ventilator tubing. During pressure controlled ventilation, the pressure in the tubing increases to the preset inspiratory pressure before flow starts and the volume increases in the lungs, and during expiration the pressure decreases rapidly to the PEEP-level before the expiratory flow starts and the volume decreases. Thus, the PV-loop seen on the ventilator monitor is square-like and does not reflect anything about the conditions in the lungs [17]. During volume controlled ventilation, the pressure in the tubing rapidly increases to the pressure needed to overcome the resistance in the tubing and the airway before the flow starts. The ascending part of the P-V loop reflects the compliance of the lungs and of the ventilator tubing. However, if an active humidifier is used, the compliance of the tubing-system may be substantial (up to 4 cmH₂O/ml). The expiratory part of the curve is similar to the one obtained during volume controlled ventilation.

MYTH

Quasi-static breath –by-breath compliance reflects lung compliance.

REALITY

It might do so, but is highly influenced by many other factors which should be considered.

First, the chest wall compliance, which can be quite low in ARDS, is included in the calculation. Second, usually the end-expiratory pressure is not obtained after a short pause, disregarding auto-PEEP. Third, breath-by –breath compliance is highly affected by lung recruitment, the PEEP-level as well as the tidal volume.

MYTH

PEEP recruits collapsed lung regions.

REALITY

PEEP does not recruit, but prevents de-recruitment of collapse-prone lung regions.

Recruitment is an inspiratory phenomenon (i.e. dependent on the inspiratory pressures and not on PEEP). On the other hand, de-recruitment is an expiratory phenomenon which might be prevented by a PEEP set above the pressure at which de-recruitment occurs.

Recruitment of collapsed lung regions depends on the regional trans-pulmonary pressure, i.e. the alveolar (airway) pressure minus the local pleural pressure. In acute respiratory distress syndrome (ARDS), to recruit collapsed lung this pressure (opening pressure) has theoretically to be between 14 and 20 cmH₂O. In ARDS the pleural pressure increases markedly from non-dependent to dependent lung regions due to the compressing forces of the weight of the edematous lung, the mediastinum as well as the abdominal pressure. The airway pressure needed to produce adequate trans-pulmonary opening pressures is therefore higher in the dependent parts of the lung and is usually above 45 cmH₂O in adult patients with ARDS. Hence, in order to recruit basal lung collapse, inspiratory airway pressures has to be above this level [12,13]. However, the trans-pulmonary pressure at which the lungs start to collapse again (collapse pressure, see above) is lower than the opening pressure [3,9] and the positive end-end expiratory airway pressure needed to prevent de-recruitment (of dependent lung regions) is therefore only 10-20 cmH₂O. Without an adequate PEEP, the lungs would immediately collapse after recruitment [17] and PEEP is for that reason an important part of the lung recruitment process.

An explanation to the belief that PEEP causes lung recruitment might be the common procedure to set PEEP. When setting PEEP during pressure controlled ventilation, the pressure above PEEP is not usually adjusted downwards and during volume controlled ventilation the tidal volumes are not reduced. This will generate a higher inspiratory pressure and thus increased trans-pulmonary pressures which will recruit denser lung collapse and the higher PEEP will then prevent de-recruitment of these newly recruited lung regions.

CONCLUSION.

There are many myths in medicine including respiratory mechanics. However, what is considered as a myth today might be a reality tomorrow and vice versa.

REFERENCES

1. Hickling KG. The pressure-volume curve is greatly modified by recruitment. A mathematical model of ARDS lungs. *Am J Respir Crit Care Med* 1998; 158: 194-202.
2. Jonson B, Svantesson C. Elastic pressure-volume curves: what information do they convey? *Thorax* 1999; 54: 82-87.
3. Crotti S, Mascheroni D, Caironi P, Pelosi P, Ronzoni G, Mondino M, Marini JJ, Gattinoni L. Recruitment and derecruitment during acute respiratory failure: a clinical study. *Am J Respir Crit Care Med* 2001; 164: 131-140.
4. Pelosi P, Goldner M, McKibben A, Adams A, Eccher G, Caironi P, Losappio S, Gattinoni L, Marini JJ. Recruitment and derecruitment during acute respiratory failure: an experimental study. *Am J Respir Crit Care Med* 2001; 164: 122-130.
5. Jonson B, Richard JC, Straus C, Mancebo J, Lemaire F, Brochard L. Pressure-volume curves and compliance in acute lung injury: evidence of recruitment above the lower inflection point. *Am J Respir Crit Care Med* 1999; 159: 1172-1178.
6. Ranieri VM, Brienza N, Santostasi S, Puntillo F, Mascia L, Vitale N, Giuliani R, Memeo V, Bruno F, Fiore T, Brienza A, Slutsky AS. Impairment of lung and chest wall mechanics in patients with acute respiratory distress syndrome: role of abdominal distension. *Am J Respir Crit Care Med* 1997; 156: 1082-1091.
7. Vieillard-Baron A, Prin S, Schmitt J-M, Augarde R, Beauchet A, Jardin F. Pressure-volume curves in acute respiratory distress syndrome. *Am J Respir Crit Care Med* 2002;165:1107-1112.
8. Hickling KG. Best compliance during a decremental, but not incremental, positive end- expiratory pressure trial is related to open-lung positive end- expiratory pressure. A mathematical model of acute respiratory distress syndrome lungs. *Am J Respir Crit Care Med* 2001; 163: 69-78.
9. Rimensberger PC, Cox PN, Frndova H, Bryan AC. The open lung during small tidal volume ventilation: concepts of recruitment and "optimal" positive end-expiratory pressure. *Crit Care Med* 1999;27: 1946-52.
10. Malbouisson LM, Muller JC, Constantin JM, Lu Q, Puybasset L, Rouby JJ and the CT Scan ARDS Study Group. Computed tomography assessment of positive end-expiratory pressure-induced alveolar recruitment in patients with acute respiratory distress syndrome. *Am. J. Respir. Crit. Care Med.* 2001;163: 1444-50.
11. Rothen HU, Sporre B, Engberg G, Wegenius G, Hedenstierna G. Re-expansion of atelectasis during general anaesthesia: a computed tomography study. *Br J Anaesth* 1993, 71:788-95.
12. Lapinsky SE, Aubin M, Mehta S, Boiteau P, Slutsky AS. Safety and efficacy of a sustained inflation for alveolar recruitment in adults with respiratory failure. *Intensive Care Med* 1999; 25: 1297-1301.
13. Medoff BD, Harris RS, Kesselman H, Venegas J, Amato MB, Hess D. Use of recruitment maneuvers and high-positive end-expiratory pressure in a patient with acute respiratory distress syndrome. *Crit Care Med* 2000; 28: 1210-6.
14. Lumb A. Elastic forces and lung volumes. In: Nunn's Applied Respiratory Physiology 5th eds. Oxford: Butterworth-Heinemann, 2000: 37-57.
15. Ingimarsson J, Thorsteinsson A, Larsson A, Werner O: Lung and chest wall mechanics in anesthetized children. Influence of body position. *Am J Respir Crit Care Med* 2000; 162: 412-7.
16. Thorsteinsson A, Larsson A, Jonmarker C, Werner O: Pressure-volume relations of the respiratory system in healthy children. *Am J Respir Crit Care Med* 1994;150:421-30.
17. Karason S, S ndergaard S, Lundin S, Stenqvist O. Continuous on-line measurements of respiratory system, lung and chest wall mechanics during mechanic ventilation. *Intensive Care Med* 2001;27:1328-39.
18. Neumann P, Berglund JE, Mondejar EF, Magnusson A, Hedenstierna G. Dynamics of lung collapse and recruitment during prolonged breathing in porcine lung injury. *J Appl Physiol* 1998, 85: 1533-43.