Histoire de la « Servo-ventilation »

Pr Bjorn Jonson

Service de Physiologie,
Hôpital de Lund, Suède

Histoire de la “Servo-Ventilation” or History of “Servo-Ventilation”

Björn Jonson
Department of Clinical Physiology
Lund University Hospital
Lund Sweden

Kindly invited to present the “History of ServoVentilation” I was charmed by the opportunity to tell the story and to do so in a personal manner. Accordingly, this story should in no way be regarded as a review, but rather as a personal testimony of a break through that continues in small evolutionary steps.

How it all came about

The ServoVentilator story commenced in 1964, upon return from the USA of a 24 year old student, Björn Jonson. He had been trained during 3 months in the field of respiratory physiology by professor Arend Bouhuys in Atlanta. BJ met in Lund Sven Ingelstedt, an associate professor of Ear, Nose and Throat diseases. Sven was an odd fellow, a marvellous, wonderful character, and I would say a genius. On his bookshelf I found a folder with the following words on its back:

GOD and I know!

Upon my question if we might be three Sven replied: “As time goes by”. Within short, Sven put the folder on my desk and I read. The message on page after page stayed between GOD and Sven because I did not understand. However, on one page I read:
Ventilators are pressure or volume controlled. That is silly! They should be flow controlled! WHY? Because then, one could do anything!
P.S. It is not possible to control flow!

That was not so difficult to understand, so I asked Sven: “If I learn how to control flow, could we then make a flow controlled ventilator?” “Oh, Yes do it!”

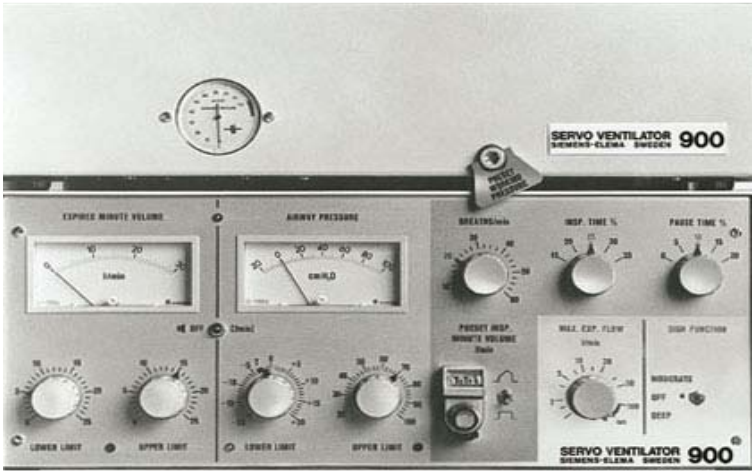
I tried hard. Every attempt to make a god flow regulating valve failed because of friction or leakage. One day complaining over the difficulties, I nervously sat with a glass syringe in my hands. I twirled the piston in the cylinder. It spun without noticeable friction and it was very tight. EUREKA! I started to do mechanical work. Immediately, I was in need for help.
I needed a turning-lathe. After some rather dangerous events in the mechanical workshop of the Medical Faculty I was thrown out from there. My head, Professor Håkan Westling, sacrificing rules and regulations, bought me a lathe. I also needed to drill in glass, cut in glass and grind the glass. The glassblower of the University, Alf Lundberg, taught me and offered me his tools. Soon enough I made a mechanical flow regulator. I wrote a patent application and my first patent was granted. I needed some electronic stuff to open and close the inspiratory and expiratory flow regulators. An engineer at Elema-

Schönander, later Siemens-Elema, Sven-Gunnar Olsson, had sold ECG apparatus to our department and also shown that he was skilled in construction work fulfilling our specific requirements. He was offered partnership in the realisation of a ventilator. After a patent bureau failed in patenting the ventilator I wrote a new application that passed. With a 3 kg ventilator and a patent in my hands I met three wise men at Elema-Schönander, already then owned by Siemens. The ventilator could do everything a 300 kg Engström ventilator could do and much more. It was silent and it was patient triggered. The wise men understood the message and a contract was singed in 1967. Under the electronic revolution that just had started, the limitations of mechanical flow regulation and the desire to use electronic flow control became an obsession. The highest technical expertise told us, it was impossible to servocontrol flow rate at some milliseconds and still more so to do so using clinical, simple to clean and to use electronics. Many efforts failed. Serendipity came once more to our help, when someone was pinching a silicone tube with a haemostatic forceps. The flow regulating pinched tube was born.

In the mean time, Lars Nordström, joined the group. Lars was a fabulous doctor in anaesthesia – a real doctor if there ever was one. He became my clinical teacher and I his scientific mentor. His Ph.D. thesis included the paper describing the ServoVentilator[1].

Fig. 1. Sven Ingelstedt, Björn Jonson and Lars Nordström on the day of Björn's Ph.D. dissertation in 1970. The dissertation was on lung mechanics studied at controlled flow rate, but not in conjunction with mechanical ventilation.

The electronic prototype of the ServoVentilator was up and running in 1970. In 1971 it was on the market that was concurred in months – to begin with in Scandinavia and within very short on continental Europe. In focus, already from the start, was treatment of the most gravely ill patients, those with IRDS or ARDS. We were much impressed and stimulated by the paper on ARDS by Ashbaug, Petty et al. in Lancet 1967[2] and by many others who understood basal physiology related to surfactant deficiency[3-5]. Therefore, the ServoVentilator was equipped with a PEEP valve, with which gross success was made in large numbers of patients. The valve contributed importantly to the success of the ventilator, Fig. 2.



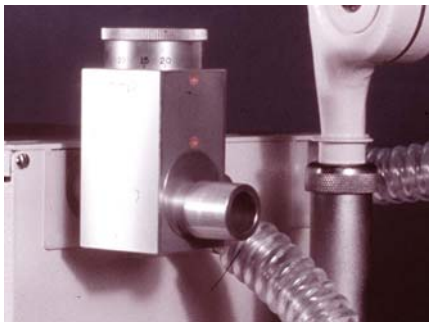


Fig. 2. ServoVentilator 900 and its PEEP valve.

How was the success story possible? Figure 3 illustrates how. In the Faculty of Medicine there was hardly any organisation, and absolutely no steering and controlling bodies. Instead there was freedom and a lot of spontaneous contacts between people. Would it be possible to repeat a similar development today? Probably not! Within the "professional organisation" of our modern faculty resources are rationed and steered to fashionable fields in which everyone competes. There is very little opportunity for spontaneity.

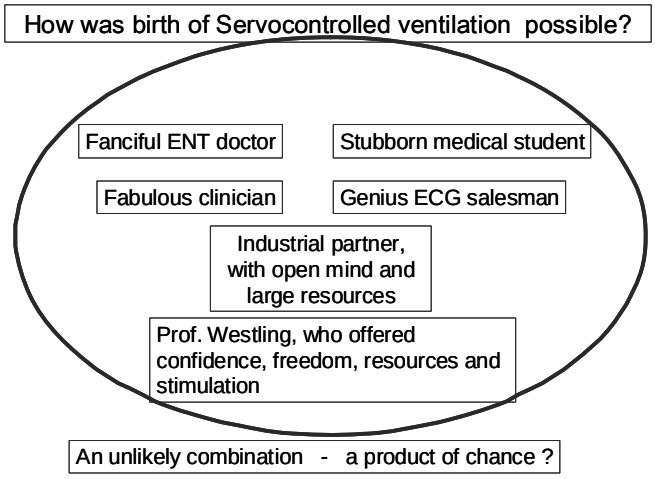


Fig. 3. ServoVentilation history was conceived in a milieu characterised by freedom and opportunity for personal contacts. In such an environment, what is hardly possible has a chance to happen.
In a modern controlled organisation only what is set up as goal can happen – often it doesn't.

ServoVentilation and physiology

The ServoVentilator was not only a ventilator. Being born within the framework of Clinical Physiology it was natural that it also was a tool for physiological monitoring and diagnostics. From the beginning the signals for pressure and flow could be recorded or displayed on the very simple monitors of these days, which were not much more than oscilloscopes. In a recording cable compliance and resistance were calculated and could together with the pressure and flow signals be read from multi-channel paper recordings.

The lung mechanics unit calculated and displayed inspiratory and inspiratory resistance as well as compliance[6]. End Expiratory Lung Pressure, EELP, years later denoted intrinsic PEEP or auto-PEEP. Compliance was corrected for EELP. In 1975 I worked as visiting researcher at Thoraxcentrum in Rotterdam. There I met Omar Prakash, a great character and a fabulous anaesthesiologist. With a prototype of the CO_2 Analyzer 930 we studied physiological effects of deep hypothermia and launched the pH regime based upon the α-stat of histidine[7]. It implies that pH is allowed to shift at low body temperatures as is now a standard concept. We also proved the feasibility of extubation on the day of heart surgery on the basis of thorough physiological analyis[8]. Omar was of great importance for the evolution of ServoVentilation including physiological monitoring. He was a marvellous friend to be remembered for ever.

Gas exchange

The CO_2 Analyzer 930 was based upon a mainstream transducer yielding nearly instant measurement[9]. Very high fidelity measurement of flow and CO_2 is prerequisite for calculation of dead space and even more so of CO_2 elimination per breath, $VtCO_2$, or per minute, $V'CO_2$. $V'CO_2$ is a monitoring parameter of paramount importance. In steady state $V'CO_2$ reflects metabolic rate, and is for that purpose nearly equivalent to oxygen consumption. Alveolar ventilation must be strictly proportional to metabolic rate to yield a particular $PaCO_2$. Information of $V'CO_2$ gives valuable information that may indicate anxiety, stress, pain and undue work of breathing. A high value of $V'CO_2$ should lead to action against such factors, particularly as a part of a lung protective low tidal volume strategy.

CO_2 elimination reflects confluence of alveolar perfusion and ventilation. Any change of ventilation/perfusion efficiency will immediately be reflected in transient changes of $V'CO_2$. Hundreds of times I have been watching the immediate effects of a change in ventilator setting. Modest changes in PEEP, respiratory rate, post-inspiratory pause etc may often lead to distinct instant changes in $V'CO_2$, to the better or to the worse. After a few minutes of testing with different combinations of ventilator settings it is nearly always possible to find a setting that leads to improved CO_2 elimination, lower tidal volumes or lower pressures, depending upon what is most desirable in the clinical situation. Positive effects of resetting may even be dramatic as can be witnessed by colleagues who have asked for my advice. Very regrettable, I have not been able to design scientific protocols to prove such strategies or to stimulate my colleagues in intensive care to undertake such studies. However, some steps have been taken[10,11].

The CO_2 Analyzer 930 displays not only end-tidal CO_2, but first and foremost $V'CO_2$. In a series of studies together with Roger Fletcher, the single breath test for CO_2 was developed for detailed analysis of dead space separating airway from alveolar dead space[12-17]. It is hard to accept that an instrument with similar features is not longer available. More recent instruments are based on slower CO_2 transducers; filters are often used which attenuate the instant information one obtains when the ventilator is reset. I hope that further research will stimulate the industry to produce more optimal monitoring systems and to be a partner of such research.

Lung protective ventilation

Stimulated by the analysis of Mead et al.[4,5] and by observations of Reynolds[3], I studied the effects of various modes of ventilation, often in partnership with Burkhard Lachmann[18-20]. On the basis of these studies, in 1982 I stated: "A respiratory pattern should open up closed units and maintain aeration and stability throughout the respiratory cycle."[21]. The underlying observations were from own and from others' studies based upon the use reversed I/E ratio, high frequency jet ventilation and high frequency oscillation. It stood soon clear that although such extraordinary modes of ventilation were efficient with respect to lung protection they are complex to set and to monitor and differ fundamentally with respect to traditional physiological concepts. A more rational was to address lung protective ventilation would rather be to refine traditional modes of ventilation by tailoring ventilator setting to the individual pathophysiology of the patient, at all times. Therefore, the following development was focused on diagnostics and improved understanding of elastic P/V curves, factors of importance for dead space and other physiological aspects.

Expiratory flushing of airways and Aspiration of dead space – ASPIDS

Since 1986 I have spent about 5 years in Paris, starting in Hôptital Beaujon where I together with Michel Aubier, Milic-Emili and others studied the effect of expiratory flushing of airways, EFLA[22], later denoted tracheal gas injection[23]. Realising that EFLA was associated with problems related to jet insufflation of dry gas into trachea and had limited effects in the presence of expiratory flow continuing to end-expiration, EFLA was abandoned. I developed aspiration of dead space, ASPIDS, that was studied in co-operation with Edoardo De Robertis[24-26]. ASPIDS implies that during late expiration, gas is aspirated from an extra channel at the tip of the tracheal tube. The Aspirated gas is replaced by fresh gas supplied through the main lumen of the tracheal tub. ASPIDS has none of the drawbacks of EFLA and has proven efficient in drastically reducing tidal volume in ARDS[27]. It is noteworthy that a reduction in dead space implies that the optimal respiratory rate is importantly reduced, so as to pave the way for very low tidal volume ventilation, a field that is currently being explored.

Mode of inspiratory gas insufflation – Mean Distribution Time

Gas transport within the lung periphery is by diffusion rather than by bulk flow. Diffusion takes time. Accordingly, during delivery of tidal volume gas transport by diffusion to and from the gas/blood interface commences when the fresh gas interface has reached the respiratory zone and continues until expiration starts, when the interface rapidly moves away from the respiratory zone. Uttman et al. presented the concept mean distribution time, MDT, that represents the average time tidal volume partitions is available for gas diffusion into and from the respiratory zone[11,27-29]. In animal and human studies a logarithmic relationship exists between MDT and CO_2 exchange. This implies that at an increased respiratory rate, the short MDT may lead to dramatic impairment of gas exchange. To prevent that, delivery of inspiratory flow must maintain an adequate MDT. Particularly, a relatively long post-inspiratory pause is beneficial in balancing the influence on MDT by a high rate. I have been kept responsible and even accused for green numbers on the ServoVentilator such as inspiratory time of 25 % and a pause time

of 10 %. I don't remember if I was responsible but the mistake may have been mine. In many situations a rational inspiratory time including pause is around 50 % rather than 65 and particularly so at high respiratory rates. Some auto-PEEP may develop because of the shorter expiration time, but that can efficiently be balanced by lowering of set PEEP.

Mechanics and P/V curves

In Hôpital H. Mondor, where I still am active in the marvellous team of François Lemaire and Laurent Brochard, we extensively studied lung recruitment in relation to mechanics, with emphasis on the elastic P/V curve[30-40]. In short, the unique feature of the ServoVentilator 900C that it can be controlled by external electronic means, e.g. by a computer, was taken advantage of[30]. The relationships between tidal volume and PEEP as means for lung recruitment could efficiently be explored on the basis of automatically recorded P/V curves[31-33]. By automatic recording of multiple inspiratory/expiratory P/V loops it is possible at bedside to elucidate the distribution of opening and closing pressure at bedside, Fig. 4 [41,42].

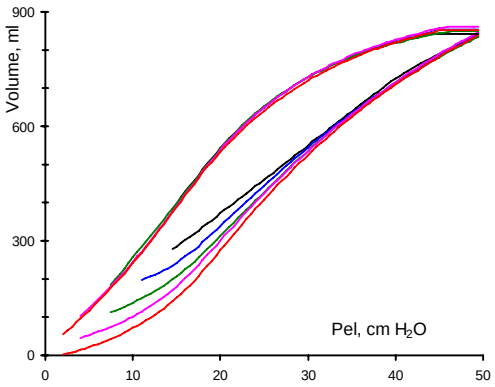


Fig. 4. In an ARDS patient elastic pressure/voume loops, were automatically recorded (Aboab, Jonson, Brochard et al. in preparation). The inspiratory limbs started at 12.5, 10, 7.5, 5, 2.5 and 0 cm H_20 of PEEP. For each level of lower PEEP a certain volume was lost, illustrating the wide range of closing pressure in this patient. During re-insufflation the lost volume was regained as shown by the converging inspiratory curves Recruitment was complete at 30-35 cm H_2O.

Goal oriented ventilation

Lung physiology, comprising mechanics, ventilation/perfusion relationships and gas exchange is complex. A modern ventilator, among the first one was the ServoVentilator, also offers many degrees of freedom with respect to mode of operation and setting. Lung and ventilator together, is an overwhelmingly incomprehensive system. As said above, also moderate changes of ventilator settings may lead to drastic effects, for good or bad. If a doctor defines the immediate physiological goals of mechanical ventilation, how should he set ventilator in order to reach these goals? Let us suggests that in ARDS tidal volume should be a low as possible while plateau pressure should not be higher than 30 cm H_2O while $PaCO_2$ should be e.g. 50 mm Hg. Although I do not believe in "holy numbers", let us assume in that these are adequate goals in a particular patient. How to set the ventilator to achieve these goals as efficiently as possible? Even for the most experienced expert it is hardly possible to say and for the less experienced doctor at 3 a.m. on a Sunday the problem is insurmountable. The solution to the issue depends on the detailed actual physiology of the patient.

Goal oriented ventilation stands for that the physician defines the immediate goals to be achieved by mechanical ventilation. The monitoring system, comprising adequate physiological signals, at least pressure, flow and CO_2, defines the actual pathophysiology of the patient. The optimal operational mode of the ventilator is then sought by iterative analysis during which the computer systematically simulates ventilation at different ventilator settings until a setting is found that leads as far as possible towards the goals. Leif Uttman and I are stepwise developing and documenting a system for goal oriented ventilation[27,28,43]. The objective is to apply old and recent knowledge about how different modes and settings of ventilation affect lung mechanics and gas exchange in the search for an optimal setting. The greatest obstacle may be that current dogmas incline to use a recipe valid for wide categories of patient rather than to search for the optimal treatment in the individual patient. Another problem with the actual state of the art is that small advantages of certain modifications of ventilation are ignored. An example is the positive effect on gas exchange by an adequate postinspiratory pause[11,29]. In itself, the effect of a pause may be regarded as trivial, but as the reduction in dead space motivates the use of a higher respiratory rate the total effect may be considerable and at times even dramatic.

Final reflections

The ServoVentilation history has been an adventurous journey over more than 40 years. Still the story is full of opportunities and challenges. True ServoVentilation resides in the future, when monitoring facilities allow characterisation of the physiology of the patient at each stage of disease and when physicians are led by the "physiological mapping" in his/hers decision about the immediate physiological goals of mechanical ventilation. The computer/ventilator may then more or less automatically in a true servo controlled mode accomplish optimal ventilation leading to the goals. This bright future is still at a distance.

References

1. Ingelstedt, S., Jonson, B., Nordstrom, L. & Olsson, S.G. A servo-controlled ventilator measuring expired minute volume, airway flow and pressure. *Acta Anaesthesiol Scand Suppl* **47**, 7-27 (1972).
2. Ashbaugh, D.G., Bigelow, D.B., Petty, T.L. & Levine, B.E. Acute respiratory distress in adults. *Lancet* **2**, 319-23 (1967).
3. Reynolds, E.O. Effect of alterations in mechanical ventilator settings on pulmonary gas exchange in hyaline membrane disease. *Arch Dis Child* **46**, 152-9 (1971).
4. Mead, J., Whittenberger, J.L. & Radford, J., E.P. Surface Tension as a Factor in Pulmonary Volume-Pressure Hysteresis. *J Appl Physiol* **10**, 191-196 (1957).
5. Mead, J., Takishima, T. & Leith, D. Stress distribution in lungs: a model of pulmonary elasticity. *J Appl Physiol* **28**, 596-608 (1970).
6. Jonson, B., Nordstrom, L., Olsson, S.G. & Akerback, D. Monitoring of ventilation and lung mechanics during automatic ventilation. A new device. *Bull Physiopathol Respir (Nancy)* **11**, 729-43 (1975).

7. Prakash, O. et al. Cardiorespiratory and metabolic effects of profound hypothermia. *Crit Care Med* **6**, 340-6 (1978).
8. Prakash, O. et al. Criteria for early extubation after intracardiac surgery in adults. *Anesth Analg* **56**, 703-8 (1977).
9. Olsson, S.G., Fletcher, R., Jonson, B., Nordstom, L. & Prakash, O. Clinical studies of gas exchange during ventilatory support--a method using the Siemens-Elema CO2 analyzer. *Br J Anaesth* **52**, 491-9 (1980).
10. Aboab, J. et al. CO2 elimination at varying inspiratory pause in acute lung injury. *Clin Physiol Funct Imaging* (2006).
11. Uttman, L. & Jonson, B. A prolonged postinspiratory pause enhances CO2 elimination by reducing airway dead space. *Clin Physiol Funct Imaging* **23**, 252-6 (2003).
12. Fletcher, R. & Jonson, B. A new method for calculation of ventilatory deadspace. *Br J Anaesth* **63**, 639-40 (1989).
13. Fletcher, R., Malmkvist, G., Niklason, L. & Jonson, B. On-line measurement of gas-exchange during cardiac surgery. *Acta Anaesthesiol Scand* **30**, 295-9 (1986).
14. Fletcher, R. & Jonson, B. Deadspace and the single breath test for carbon dioxide during anaesthesia and artificial ventilation. Effects of tidal volume and frequency of respiration. *Br J Anaesth* **56**, 109-19 (1984).
15. Fletcher, R., Werner, O., Nordstrom, L. & Jonson, B. Sources of error and their correction in the measurement of carbon dioxide elimination using the Siemens-Elema CO2 Analyzer. *Br J Anaesth* **55**, 177-85 (1983).
16. Fletcher, R. & Jonson, B. Prediction of the physiological dead space/tidal volume ratio during anaesthesia/IPPV from simple pre-operative tests. *Acta Anaesthesiol Scand* **25**, 58-62 (1981).
17. Fletcher, R., Jonson, B., Cumming, G. & Brew, J. The concept of deadspace with special reference to the single breath test for carbon dioxide. *Br J Anaesth* **53**, 77-88 (1981).
18. Jonson, B. & Lachmann, B. Setting and monitoring of high-frequency jet ventilation in severe respiratory distress syndrome. *Crit Care Med* **17**, 1020-4 (1989).
19. Lachmann, B., Schairer, W., Hafner, M., Armbruster, S. & Jonson, B. Volume-controlled ventilation with superimposed high frequency ventilation during expiration in healthy and surfactant-depleted pig lungs. *Acta Anaesthesiol Scand Suppl* **90**, 117-9 (1989).
20. Jonson, B., Lachmann, B. & Fletcher, R. Monitoring of physiological parameters during high frequency ventilation (HFV). *Acta Anaesthesiol Scand Suppl* **90**, 165-9 (1989).
21. Jonson, B. *Positive airway pressure. Some physical and biological effects.*, (Martinus Nijhoff, The Hague, 1982).
22. Jonson, B., Similowski, T., Levy, P., Viires, N. & Pariente, R. Expiratory flushing of airways: a method to reduce deadspace ventilation. *Eur Respir J* **3**, 1202-5 (1990).
23. Marini, J.J. Tracheal gas insufflation: a useful adjunct to ventilation? *Thorax* **49**, 735-7 (1994).

24. De Robertis, E., Servillo, G., Tufano, R. & Jonson, B. Aspiration of dead space allows isocapnic low tidal volume ventilation in acute lung injury. Relationships to gas exchange and mechanics. *Intensive Care Med* **27**, 1496-503 (2001).
25. De Robertis, E., Servillo, G., Jonson, B. & Tufano, R. Aspiration of dead space allows normocapnic ventilation at low tidal volumes in man. *Intensive Care Med* **25**, 674-9 (1999).
26. De Robertis, E., Sigurdsson, S.E., Drefeldt, B. & Jonson, B. Aspiration of airway dead space. A new method to enhance CO2 elimination. *Am J Respir Crit Care Med* **159**, 728-32 (1999).
27. Uttman, L., Ogren, H., Niklason, L., Drefeldt, B. & Jonson, B. Computer simulation allows goal-oriented mechanical ventilation in acute respiratory distress syndrome. *Crit Care* **11**, R36 (2007).
28. Uttman, L. & Jonson, B. Computer-aided ventilator resetting is feasible on the basis of a physiological profile. *Acta Anaesthesiol Scand* **46**, 289-96 (2002).
29. Aboab, J. et al. CO2 elimination at varying inspiratory pause in acute lung injury. *Clin Physiol Funct Imaging* **27**, 2-6 (2007).
30. Svantesson, C. et al. A single computer-controlled mechanical insufflation allows determination of the pressure-volume relationship of the respiratory system. *J Clin Monit Comput* **15**, 9-16 (1999).
31. Richard, J.C. et al. Respective effects of end-expiratory and end-inspiratory pressures on alveolar recruitment in acute lung injury. *Crit Care Med* **31**, 89-92 (2003).
32. Maggiore, S.M. et al. Alveolar derecruitment at decremental positive end-expiratory pressure levels in acute lung injury: comparison with the lower inflection point, oxygenation, and compliance. *Am J Respir Crit Care Med* **164**, 795-801 (2001).
33. Richard, J.C. et al. Influence of tidal volume on alveolar recruitment. Respective role of PEEP and a recruitment maneuver. *Am J Respir Crit Care Med* **163**, 1609-13 (2001).
34. Jonson, B. et al. Pressure-volume curves and compliance in acute lung injury: evidence of recruitment above the lower inflection point. *Am J Respir Crit Care Med* **159**, 1172-8 (1999).
35. Maggiore, S.M., Richard, J.C. & Brochard, L. What has been learnt from P/V curves in patients with acute lung injury/acute respiratory distress syndrome. *Eur Respir J Suppl* **42**, 22s-26s (2003).
36. Richard, J.C. et al. Influence of respiratory rate on gas trapping during low volume ventilation of patients with acute lung injury. *Intensive Care Med* **28**, 1078-83 (2002).
37. Servillo, G. et al. The upper inflection point of the pressure-volume curve. Influence of methodology and of different modes of ventilation. *Intensive Care Med* **28**, 842-9 (2002).
38. Decailliot, F. et al. Pressure-volume curves with and without muscle paralysis in acute respiratory distress syndrome. *Intensive Care Med* (2006).
39. Richard, J.C. et al. Effects of vertical positioning on gas exchange and lung volumes in acute respiratory distress syndrome. *Intensive Care Med* (2006).

40. Demoule, A. et al. Relationship between pressure-volume curve and markers for collagen turn-over in early acute respiratory distress syndrome. *Intensive Care Med* **32**, 413-20 (2006).
41. Bitzen, U., Drefeldt, B., Niklason, L. & Jonson, B. Dynamic elastic pressure-volume loops in healthy pigs recorded with inspiratory and expiratory sinusoidal flow modulation. Relationship to static pressure-volume loops. *Intensive Care Med* **30**, 481-8 (2004).
42. Bitzen, U. et al. Multiple pressure-volume loops recorded with sinusoidal low flow in a porcine acute respiratory distress syndrome model. *Clin Physiol Funct Imaging* **26**, 113-9 (2006).
43. Uttman, L., Beydon, L. & Jonson, B. Effects of positive end-expiratory pressure increments can be predicted by computer simulation based on a physiological profile in acute respiratory failure. *Intensive Care Med* **29**, 226-32 (2003).