Upsala J Med Sci 95: 53–61

Airway Pressures during Positive-pressure Ventilation with Superimposed Oscillations

Anders Jonzon, José A. Tantalean, Torgny Norsted and Gunnar Sedin

Department of Pediatrics, University Hospital and Department of Physiology and Medical Biophysics, Uppsala University, Uppsala, Sweden

ABSTRACT

This study was undertaken to determine whether oscillations superimposed on a regular ventilatory pattern influence the arterial blood gases and pH and the airway pressures at adequate alveolar ventilation at the onset of inhibition of inspiratory activity. The peak, mean and end-expiratory airway pressures were therefore measured at inhibition of this activity with and without superimposition of oscillations on the ventilatory pattern. It was found that superimposed oscillations lowered the airway pressure only at a low ventilatory frequency, whereas inhibition occurred at almost equal arterial PCO2 and pH values with and without superimposed oscillations on the ventilatory pattern.

INTRODUCTION

In a study by Jonzon et al (8) it was found that with high-frequency positive-pressure ventilation at frequencies of 60 to 120 insufflations per minute, adequate alveolar ventilation could be achieved with tidal volumes smaller than the anatomical dead space. This was in conflict with existing rules for ventilation, which stated that the tidal volume should be greater than the dead space volume (13). The high inspiratory gas flow rate used in a low-compressible ventilator system makes adequate gas exchange possible (5) which means that this technique is beneficial in ventilation of newborn infants (2,3,14,15,16).

In 1972 Lunkenheimer et al (9) introduced a modification of "diffusion respiration" by applying transtracheal pressure oscillations at frequencies of up to 40 Hz and found that the CO2 elimination was related to the frequency and to the

amplitude of endothoracic vibrations. In the first clinical trials of high-frequency oscillations, oscillatory "tidal" volumes of 1.5-3 ml/kg body weight were used (4) at a frequency of 15 Hz. Other frequencies (8-20 Hz) were applied in clinical treatment of the respiratory distress syndrome in newborn infants (6,11). The low pressure swings that were needed to achieve adequate alveolar ventilation seemed to be one way of further reducing the barotrauma to the lungs. One of the first fields in which oscillatory ventilation seemed to be of great advantage was in the clinical treatment of newborn infants with interstitial emphysema (7).

Whereas oscillatory ventilation in the newborn infant has been difficult to monitor and adjust, superimposition of pressure oscillations on high-frequency positive-pressure ventilation (HFPPV) has been found to improve the CO2 elimination in infants with interstitial emphysema (17). Simultaneously with a reduction of the interstitial emphysema, the ventilatory pressures and the oxygen concentration in the inspired air could be lowered in most infants. The question is whether this is achieved through enhanced diffusion by oscillation of the gas in conducting airways, or through oscillation of the gas in the interstitium. In both cases the gas mixing could be improved and the absorption of gas promoted by rapid oscillatory pressure variations. In addition, it has been debated how oscillatory ventilation influences the pressure gradient in the airways and lungs.

The aim of this study was to determine whether pressure oscillations superimposed on intermittent positive-pressure ventilation alter the arterial blood gases and pH and the airway pressures at the onset of inhibition of inspiratory activity, i.e. at adequate alveolar ventilation, in cats with normal lungs.

METHODS

Subjects and preparation - Ten cats, weighing 2.7 - 4.9 kg (mean 3.5 kg, SD 0.7) were studied. Anaesthesia was induced with chloroform and maintained with intermittent injections of chloralose (Merck, AG, GFR). Catheters were introduced through the femoral vein and artery into the inferior vena cava and aorta. The left phrenic nerve was exposed through a frontal,

medial incision in the neck. An endotracheal tube was inserted just below the larynx and a ligature was placed so that no air could leak between the endotracheal tube and the tracheal wall. Two equally long catheters had previously been attached to the endotracheal tube so that one had its tip 1 cm above the carina and the other had its tip 5 cm further down the airway. An equally long catheter was attached to the distal connector of the endotracheal tube. Another catheter was inserted through the rib cage, without letting any air into the pleural space.

MEASUREMENTS

Airway pressures were measured at the proximal end of the endotracheal tube ("tubing pressure"), at the tip of the endotracheal tube, 1 cm above the carina ("tip pressure") and 5 cm below the tip of the endotracheal tube ("distal pressure"). Pleural pressure was measured through a catheter inserted into the pleural space. Arterial blood pressure was measured through a catheter placed in the lower aorta. All catheters were connected to identical transducers (Druck AG, GFR) and amplifiers (Hellige AG, GFR). All signals were amplified with an 8-channel medical amplifier system (Hellige AG, GFR) and fed to a recorder (Recorder 330-P, Hellige AG, GFR). Measurements of arterial blood gases and pH were made with an automatic acid-base analyzer (Radiometer, Denmark).

The phrenic nerve activity was recorded by placing the left phrenic nerve on bipolar hook electrodes. The nerve and electrodes were immersed in mineral oil. For amplification a Neurolog system (Digitimer Ltd, Welwyn Garden City, U.K.; preamplifier NL 103, AC-amplifier NL 105, filters NL 115, spike trigger NL 200) was used.

Ventilators - A Siemens Elema servo ventilator (SE 900C) was used in the experiments. A positive end-expiratory pressure (PEEP) of 0.5 kPa was used. The set PEEP was not changed when oscillatory ventilation was superimposed. Oscillations of the ventilation gas were accomplished by attaching metal bellows to the tubings between the ventilator and the endotracheal tube. The bellows were controlled by a motor on which the stroke volume and number of strokes per minute could be set independently. The stroke volume of the bellows was 19 ml in these experiments, the number of strokes was 577-600/minute.

Experimental procedure - Before measurements a check was made to see that all airway pressures were zero at expiratory rest during spontaneous breathing and also that the acid-base status was normal and that base excess was above -5 mmol/l.

The experiments were performed during ventilation at frequencies of 15, 60 or 100 breaths per minute (b.p.m.) with the volume controlled ventilator (SV 900C). The inspiratory time was always 33% of the ventilatory cycle and a plateau of 10 % was used.

First the cat was ventilated at 15, 60 or 100 b.p.m. to the minute ventilation at which the phrenic nerve activity was inhibited. An arterial blood sample was then immediately drawn for determination of arterial blood gases and pH, and recordings of peak, minimum and mean airway and pleural pressures were made.

Oscillations were then superimposed on that minute ventilation and the same measurements and recordings were repeated.

Subsequently the tidal volume of the volume-controlled ventilator was reduced until phrenic nerve activity reappeared. The minute ventilation was then slowly increased until the phrenic nerve activity was again inhibited and the measurements and recordings were repeated.

RESULTS

The mean airway pressures were found to be lower with than without oscillations superimposed on intermittent positive-pressure ventilation (IPPV). When oscillations were superimposed on an IPPV of 15 b.p.m., there was a marked decrease in mean airway pressure ($p<0.5$). These pressures were also reduced to some extent ($p<0.5$) when oscillations were superimposed on an IPPV of 60 b.p.m. No decrease in mean airway pressures was noted when oscillations were superimposed on IPPV at 100 b.p.m. (Fig. 1). The peak airway pressure was unchanged by superimposition of oscillations on IPPV, regardless of the ventilatory rate. The end-expiratory pressure was significantly decreased when oscillations were superimposed on IPPV at 15 and 60 b.p.m. ($p<0.5$; Fig. 1).

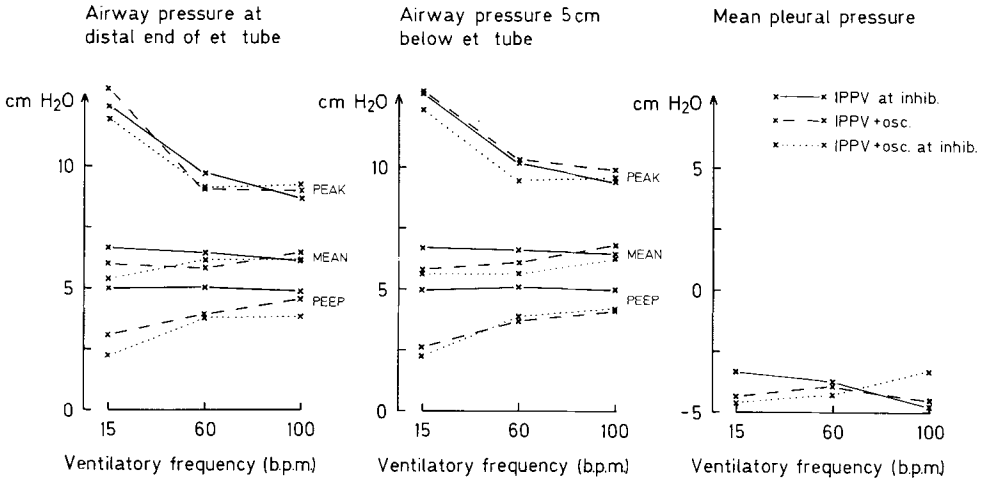

Fig. 1.
Peak, mean and end-expiratory airway pressures during ventilation at 15, 60 and 100 b.p.m. The pressures were measured at the distal end of the endotracheal (et) tube, 5 cm below the et tube and in the pleural space. Whole lines represent ventilation at inhibition of inspiratory activity without superimposed oscillations, broken lines the same minute ventilation with superimposed oscillations and dotted lines the pressures when the minute ventilation had been adjusted to inhibition of inspiratory activity.

When oscillations were superimposed on the IPPV that caused inhibition of inspiratory activity, without the minute ventilation being readjusted to the lowest minute ventilation that could cause inhibition of inspiratory activity, the end-expiratory pressure increased ($p<0.1$) at IPPV rates of 15 and 60 b.p.m. (Fig. 1).

The arterial PO_2, PCO_2, pH and BE were the same at the onset of inhibition of inspiratory activity during IPPV with and without superimposed oscillations (Fig.2).

This study confirmed the previous observations that without superimposed oscillations, peak airway pressures were higher during IPPV at 15 than at 60 b.p.m. ($p<0.1$), and that peak airway pressures at 60 b.p.m. were higher than during IPPV at 100 b.p.m. ($p<0.1$; Fig. 1) (12).

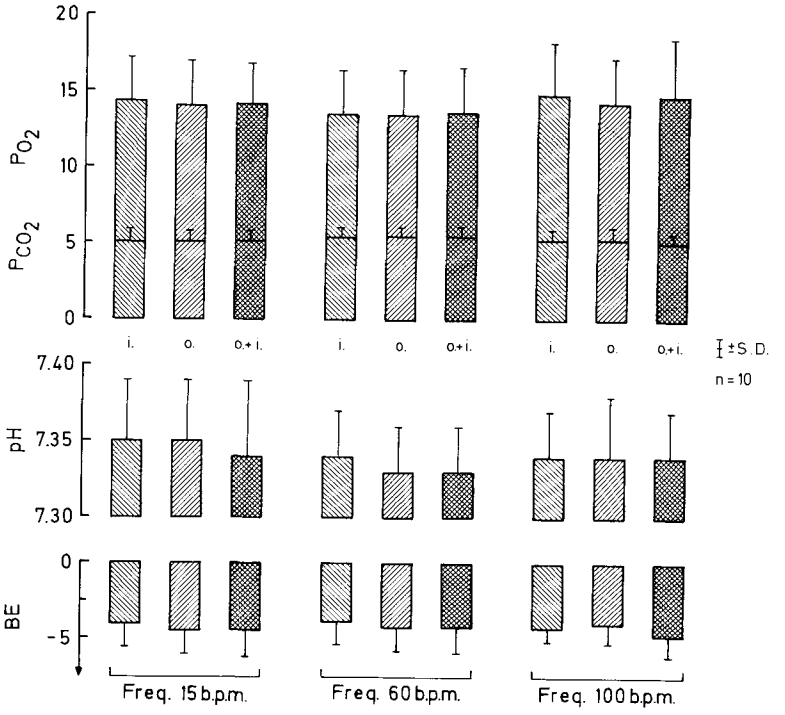

Fig. 2.
Arterial PO_2, PCO_2, pH and base excess during ventilation at 15, 60 and 100 b.p.m. The bars marked i. represent experiments with ventilation to inhibition of inspiratory activity without superimposed oscillations, the bars marked o.i. represent experiments with the same minute ventilation with superimposed oscillations, and the bars marked o.+i. represent experiments when the minute ventilation had been adjusted to inhibition of inspiratory activity.

DISCUSSION

The results of this study show that at the onset of inhibition of inspiratory activity the mean airway pressures are lower with than without oscillations superimposed on IPPV at frequencies of 15 and 60 b.p.m. At 100 b.p.m., the mean airway pressures are the same at inhibition of inspiratory activity with and without superimposed oscillations. Norsted et al (12)

have previously demonstrated that inhibition of inspiratory activity occurs at higher peak airway pressures during IPPV at a low than at a high ventilatory frequency. This observation was confirmed by the present study. The reduction of airway pressure by superimposed oscillations during IPPV at low ventilatory frequencies is not caused by a change in the peak pressure, but is a result of the concomitant reduction in the mean end-expiratory pressure. There were no indications, such as an increased intrapleural pressure, of a peripheral build-up of pressure in the lungs during ventilation with superimposed oscillations.

An important result of this study, with clinical consequences, is that by merely superimposing oscillations on IPPV without assessing whether "inhibition" has occurred, i.e. without lowering the minute ventilation to the lowest value causing inhibition, may give rise to an unnecessarily high pressure in the airways and lungs, and thus increase the risk of pulmonary barotrauma.

Our results also show that inhibition of inspiratory activity occurs at the same arterial PCO2 and pH with and without oscillations superimposed on the regular ventilation. This suggests that normal arterial blood gases and pH are the major factors for inhibition of inspiratory activity and that additional inhibitory input from receptors in the airways and lungs is of lesser importance, at least as long as the lungs remain expanded. Thus, it is likely that during IPPV at low frequencies and with superimposed oscillations the lung is kept in an expanded state throughout the ventilatory cycle with less variation in lung volume than whithout such oscillations. Superimposed oscillations probably promote inhibition of inspiratory activity by producing more continuous activity from slowly adapting pulmonary stretch receptors (cf. 1,10). The design of this study does not allow any conclusions as to whether oscillations transmitted to intercostal muscle spindles influence inhibition or excitation of inspiration as suggested by Sempik & Patrick (18).

ACKNOWLEDGEMENTS

This study was supported by grants from the Swedish Medical Research Council (19M-7544), "Stiftelsen Allmänna BB:s Minnesfond", the Samaritian Foundation and the Sigurd and Elsa Golje Memorial Fund and by the Swedish Institute (fellowship for José A Tantalean). The authors thank Siemens AB, Stockholm for kindly providing the Servoventilator 900C. The authors are indebted to Barbro Kjällström-Östmark, Harry Johansson, Gunno Nilsson and Erik Ekström for technical assistance. We also thank Susanne Thorell for typing the manuscript.

REFERENCES

1. Banzett,R., Lehr,J. & Geffroy,B.: High-frequency ventilation lengthens expiration in the anesthetised dog. J Appl Physiol 55:329-334, 1983.

2. Bland,R.D. & Sedin,G.: High frequency mechanical ventilation in the treatment of neonatal respiratory distress. In: High frequency ventilation (Smith,R.B. and Sjöstrand,U.H. eds) Internat Anaesth Clinics, 125-147, 1983.

3. Bland,R.D., Kim,M.H. Light,M.J. & Woodson,J.L.: High frequency mechanical ventilation in severe hyaline membrane disease. An alternative treatment? Crit Care Med 8:275, 1980.

4. Butler,W.J., Bohn,D.J., Miyasaka,K., Bryan,A.C. & Froese,A.-B.: Ventilation of humans by high-frequency oscillations. Anaestesiology 51:S368, 1979.

5. Eriksson, I., Jonzon,A., Sedin,G. & Sjöstrand, U.: The influence of the ventilatory pattern on ventilation, circulation and oxygen transport during continuous positive pressure ventilation. An experimental study. Acta Anaesth Scand Suppl. 164:149-163, 1977.

6. Frantz,I.D. III, Werthammer,J. & Stark,A.R.: High-frequency ventilation in premature infants with lung disease: Adequate gas exchange at low tracheal pressure. Pediatrics 71:483-488, 1983.

7. Frantz,I.D., Stark,A.R. & Werthammer,J.: Improvement in pulmonary interstitial emphysema with high frequency ventilation. Pediatr Res 15:719, 1983.

8. Jonzon,A., Öberg,P.Å., Sedin,G. & Sjöstrand,U.: High-frequency positive-pressure ventilation by endotracheal insufflation. Acta Anaesth Scand Suppl. 43, 1971.

9. Lunkenheimer,P.P., Rafflenbeul,W., Keller,H., Frank,I., Dickhut,H.H. & Fuhrmann,C.: Application of transtracheal pressure oscillations as a modification of "diffusion respiration". Br J Anaesth 44:627, 1972.

10. Man,G.C.W., Man,S.F.P. & Kappagoda,C.T.: Effects of high-

frequency oscillatory ventilation on vagal and phrenic nerve activities. J Appl Physiol 54:502-507, 1983.

11. Marchak,B.E., Thompson,W.K., Duffty,P., Miyaki,T., Bryan,M.H. & Bryan,A.C.: Treatment of RDS by high-frequency oscillatory ventilation: A preliminary report. J Pediatr 99:287, 1981.

12. Norsted,T., Jonzon,A. & Sedin,G. Inhibition of phrenic nerve activity during positive pressure ventilation at high and low frequencies. Acta Anaesth Scand 30:521-528, 1986.

13. Nunn,J.F.: Applied respiratory physiology with special reference to anaesthesia. Butterworths 1969.

14. Sedin,G.: Hochfrequente Uberdrucksbeatmung in der Neugeborenen-Intensivbehandlung. In: Pohlandt F, ed. Pädiatrische Intensivmedizin, Vol. V, INA Bd 41, Stuttgart. Georg Thieme Verlag, 54-57, 1983.

15. Sedin,G.: Positive-pressure ventilation at moderately high frequency in newborn infants with respiratory distress syndrome (IRDS). Acta Anaesth Scand 30:515-520, 1986.

16. Sedin,G., Jonzon,A. & Norsted,T.: Intermittent positive pressure ventilation at high and low frequencies. Clinical and experimental experiences. SAFE 88, Scandinavian Association for ECMO, 2nd SAFE Meeting, May 27-29, 1988.

17. Sedin,G., Jonzon,A. & Kaniklides,C.: Superimposed oscillations in the treatment of neonatal pulmonary interstitial emphysema. In manuscript 1990.

18. Sempik,A. & Patrick,J.M.: The effect of thoracic vibration on ventilation and breath-holding in man. Respiration Physiology 44:381-391, 1980.