Intensive Care Med (1985) 11:68 – 75

Intensive Care Medicine

Decelerating inspiratory flow waveform improves lung mechanics and gas exchange in patients on intermittent positive-pressure ventilation

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Accepted: 25 September 1984

Abstract. The effects of two inspiratory flow waveforms (WFs), decelerating and constant have been studied in 14 patients undergoing intermittent positive-pressure ventilation (IPPV). With tidal volume (VT), inspiratory time, inspiratory-expiratory (I/E) ratio and frequency being kept constant, the decelerating waveform produced statistically significant reduction of peak pressure, total respiratory resistance, work of inspiration, ratio of dead space to tidal volume (VD/VT) and alveolar-arterial gradient for oxygen (A-a)PO2. There was significant increase in total static and kinetic compliances and PaO2, with no significant changes in PaCO2, in cardiac output (CO) and in other haemodynamic measurements.

Key words: IPPV – Inspiratory WFs – VD/VT ratio – PaO2 – (A-a)PO2 and CO

For many years conflicting views have been expressed over the relative effectiveness of different inspiratory flow patterns in patients undergoing intermittent positive pressure ventilation (IPPV). In particular, opinion is divided on whether improvements in lung mechanics and gas exchange can be attributed to a particular type of inspiratory flow pattern. Studies on analogue models and experimental clinical observations have produced contrary results [4–6, 16, 22, 26, 30–34].

Fahri [13] stated that, in the vast majority of patients with pulmonary disease, it is the failure of adequate matching of ventilation to perfusion which incapacities the patient and any therapeutic approach must be guided to a large extent by this observation.

Bergman [6] suggested that, if an alteration in breathing pattern resulted in a small shift of ventilation into poorly ventilated but well perfused lung compartment, a rather significant improvement in blood oxygenation might occur. Some of this controversy may be related to the fact that in these studies, changing the inspiratory flow waveforms produced changes in tidal volume, frequency and I/E ratio. The principle objective of the present study therefore, was to determine whether the profile of the inspiratory flow waveforms could influence gas exchange in patients on IPPV, when the tidal volume, minute volume, I/E ratio and frequency are kept constant. It should be then possible to assess in isolation the effect of different inspiratory flow waveforms on gas exchange.

Materials and methods

Equipment testing

Prior to the patients study, a series of laboratory measurements was made on the equipment.

1. A Wright spirometer was used to measure expired VT. It was calibrated with a graduated syringe and also tested against an independent electronic respirometer (the Kontron respirometer 3111 S) both connected in series to the expiratory port of the ventilator. The response of the Wright spirometer to different VTs and peak flows was tested in the following way: the ventilator was made to ventilate a standard lung model [see (3) below]. VT was then adjusted using the electronic respirometer, so that the expired VT was always constant. Three different ranges of VT (0.4, 0.8 and 1.0 l) were studied, each of these VTs was maintained while the peak flow was changed from 10–80 l/min in steps of 10 l/min.

2. A Fleisch pneumotachograph was used to measure flow and to check the profile of the waveforms of the ventilator. The pneumotachograph was calibrated with air supplied from a compressed air cylinder at

constant flow in the range of 0 – 60 l/min against a previously calibrated rotameter.

3. *The ventilator.* This is a volume-cycled, flow regulated ventilator (BOC, Medishield IMV Pneumotron). The accurate measurement of tidal volume and minute volume are the key functions on which this ventilator relies for its operational performance. To measure these volumes, this ventilator uses two flow transducers, one on the inspiratory limb and the other on the expiratory limb. These two transducers are the same as those of the electronic respirometer [8] and are relatively insensitive to the density, viscosity, temperature and humidity of the gases they measure. The two transducers are connected via a feedback system that compares the inspiratory VT with the expired VT.

To test the accuracy of VT delivery, the ventilator was connected to a standard lung model [7] which consisted of two 20-l bottles, each with a compliance of 0.018 l/cm H_2O and a combined compliance of 0.037 l/cm H_2O, and a circuit resistance of 10.47 cm H_2O/l per second. The Fleisch pneumotachograph with a differential pressure transducer was inserted between the Y-piece of the ventilator and the two bottles. Another pressure transducer was connected to the third port of the Fleisch pneumotachograph. The Wright spirometer and the electronic respirometer were connected to the expiratory port of the ventilator. Different combinations of compliances and resistances were used. VT was changed from 0.4 l to 1.2 l in steps of 0.2 l. With each VT different ranges of peak flow were used (10, 20, 30, ... up to 80 l/min).

Patients

Fourteen patients (Table 1), 7 males and 7 females with an age range of 20 – 71 years (mean 52.00 ± 17.95) were studied. They were in respiratory failure, intubated or tracheotomized and required IPPV as part of their therapy.

Table 1. Patients' sex, age and diagnosis

Patients	Sex	Age	Diagnosis
1	F	68	Respiratory failure secondary to acute mycoplasma pneumonia
2	M	58	Neurofibromatosis, bilateral broncho-pneumonia
3	F	53	Respiratory failure due to pneumonia
4	F	64	Acute on chronic respiratory failure
5	F	32	Barbiturate overdose
6	M	20	Sickle cell lung disease
7	M	62	Adult respiratory distress syndrome
8	M	24	Adult respiratory distress syndrome
9	M	59	Acute on chronic respiratory failure
10	F	67	Acute on chronic respiratory failure
11	M	27	Adult respiratory distress syndrome
12	M	63	Adult respiratory distress syndrome
13	F	71	Cardio-respiratory arrest
14	F	60	Systemic lupus erythematosus, liver cirrhosis, septicaemia and pneumothorax

Artificial ventilation

Ventilator. All patients were ventilated with the IMV Pneumotron ventilator (see equipment testing) which allowed variation in inspiratory flow waveforms (Fig. 1) [7]. Standard connections and tubings were used including a heat-moisture exchanger (Bennett cascade II humidifier).

Ventilatory pattern employed. The choice of the ventilatory pattern used in each patient was determined by the clinical state of the patient.

Two inspiratory flow WFs were compared, i.e. constant and decelerating. In 11 patients the sequence of WF studied was constant, decelerating and back to constant. In two of these patients on separate occasions and in three further patients the sequence was decelerating, constant and then decelerating.

Constancy of tidal volume (VT) and minute volume ($\dot{V}$). In order to compare any effects of changing the inspiratory WFs, it was considered essential to maintain the constancy of both VT and $\dot{V}$ in each patient.

Constant WF produces peak and mean flows of the same value whilst the decelerating WF results in the peak flow being greater than the mean flow. Therefore, in order to maintain frequency and $\dot{V}$ constant it was necessary to increase the peak flow when the decelerating WF was in use. Expired VT was

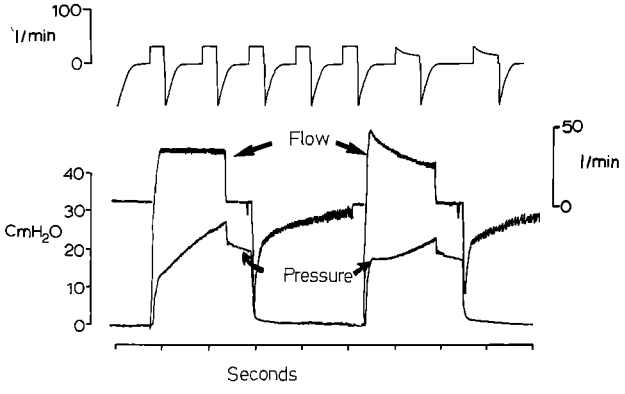

Fig. 1. The upper trace shows signals obtained from the Fleisch pneumotachograph for both constant and decelerating WFs when the ventilator was connected to the lung model. The lower trace shows flow and airway pressure signals in one patient for both WFs. Flow signals were obtained from the ventilator recording output

measured at the beginning and the end of WF study period (see equipment testing for calibration procedure).

Inspiratory-expiratory (I/E) ratio and frequency. In order to attribute any change in gas distribution to changes in inspiratory WFs, the I/E ratio was kept constant at 1 : 2 and low frequencies were used to minimize any effects frequency might have on gas distribution, these being kept constant in each study.

Inspired oxygen concentration (FIO2). FIO2 was determined by the clinical state of the patient, and was maintained constant throughout the period of the study. It was measured using a Servomex paramagnetic oxygen analyzer which was calibrated with the following gases: 100% N2, air and 100% O2. Inspired air was sampled down stream to the humidifier.

Airway pressure. Airway pressure was recorded continuously with an Elcomatic transducer (Elcomatic SE 750), calibrated in the usual way. This was connected between the Y-piece of the breathing tubes and the endotracheal/or tracheostomy tube. The signal was amplified and recorded on a multichannel recorder (UV Oscilloscope, SE Labs.).

End-inspiratory pause (EIP). An end-inspiratory pause (EIP) of 0.55 s was inserted for 15 s during the time of measurement of breathing mechanics. To test the effect of using an EIP for short period on the breathing mechanics, a pilot study on two patients was carried out, in which an EIP of 0.5 and 1.0 s were used one at a time and with each WF. Measurement of airway pressure, VT and I/E flow was made every minute for 5 min, then every 5 min for 30 min.

Measurements

All the patients were studied in the supine position. In all 14 patients the mechanics of breathing were measured in the following way: each WF was applied for 20 min and at the beginning and the end of this period, records of airway pressure, flow and a measurement of VT was made.

Figure 2 illustrates the type of airway pressure tracing produced. During the inflation of VT, airway pressure changes in the following way:

As flow ceases (t2) there is a fall in pressure (P1-P3) which is the total flow resistance (R) at the maximal lung volume. The pressure then slowly declines to reach a stable value. This decline in pressure (P3-P2) is called pressure stress relaxation [17]. Total flow resistance of the respiratory system (gas flow + lung tissue + chest wall tissue) was obtained by the same

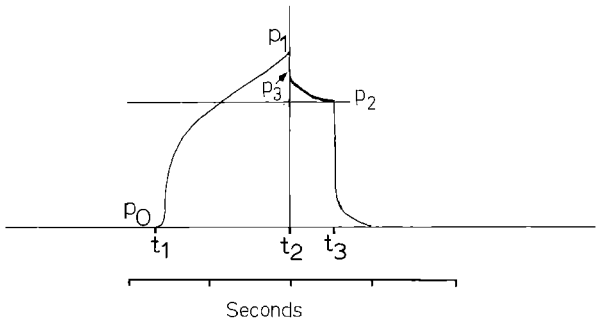

Fig. 2. A typical airway pressure trace re-touched. Flow occurred between t1 and t2, static conditions established at t3, t3-t2 = EIP. P0 = atmospheric pressure, P1-P0 = peak pressure, P2-p0 = plateau pressure. P3 is measured where a projection backwards from the slow decline in pressure (P2 to P3) meets a vertical line from P1

method of Ingelstedet et al. [18]. Total static compliance (Cst) (chest wall + lung) was calculated [11] by dividing the introduced volume (VT) by the pressure (P2-P0) established at the end of end-inspiratory pause (EIP).

A second index of compliance was calculated from the pressure (P3-P0). This pressure was found by projecting the slope of the slow decline in pressure backwards (Fig. 2) to meet a vertical line from P1. When the introduced volume is divided by this pressure it provides a measure of kinetic compliance (Ck). This term is used because dynamic compliance is calculated from the pressure difference between points of zero flow during a tidal volume [11]. Pressure stress relaxation was presented in two ways:

1. as the fall in pressure per unit initial pressure ((P3-P2/(P3-P0) × 10) mmH2O/ cmH2O;
2. as the rate of fall in pressure, obtained by dividing (P3-P2) by EIP (t3-t2) (cm H2O/s).

Arterial blood gas analysis

Five ml of arterial blood were collected in heparinised plastic syringes from an indwelling arterial catheter, the volume in the dead space having previously been discarded. The time from collection to analysis was never more than 10 min, and analysis was carried out using an Instrumentation Laboratory machine (Model 415). This was calibrated in the standard fashion immediately prior to each measurement. Each sample was measured twice, the mean value being calculated.

Haemodynamic measurements

In six patients, pulmonary artery balloon thermo-dilution flotation catheters (Edwards laboratories) were inserted for clinical reasons. Cardiac output measure-

ments were made at the beginning and the end of each study period. Five ml of ice-cold 5% dextrose were rapidly injected into the right atrium, and values were obtained from an Edwards Laboratories cardiac output computer (Model 9520). The mean of 3 sets of measurements were calculated.

Both pulmonary and systemic arterial pressures were measured using a Statham P 23 Dc transducer connected to a Kontron medical 128A monitor. Dynamic and mean pressures were measured on each occasion. Central venous pressure was monitored from a right atrial catheter.

Expired gas collection

In five patients, expired gas was collected from the ventilator outlet into a 100-l Douglas bag over a 5-min period. The volume was determined with the Wright spirometer which was fitted between the expiratory port of the ventilator and Douglas bag. The expired CO2 concentration (FECO2) was measured using an infra-red gas analyzer (P K Morgan, Model 901), which was calibrated with air, 5% CO2 in N2 and 10% CO2 (BOC, special gases).

Derived parameters [(A-a)PO2 gradient]. The ideal alveolar air equation [27] was used to calculate the alveolar oxygen tension (PAO2) using the measured inspired oxygen tension and assuming RQ to be 0.85 [25].

$$PAO2 = PIO2 - \frac{1-(1-R)FIO2}{R} PaCO2 .$$

VD/VT ratio was calculated from the Enghoff modification of the Bohr equation [12]:

$$VD/VT = \frac{PaCO2 - PECO2}{PaCO2} .$$

Mean airway pressure (Paw̄p̄) was calculated [23] by measuring the area enclosed between the pressure curve and the line of zero pressure of one respiratory cycle divided by the duration of the cycle using a Numonics digitizing pad linked to an Alpha LSI-II minicomputer.

The work of breathing (inspiration), defined as the work done when a litre of gas moves in response to a pressure gradient of 1 kilo Pascal, was calculated by the product of the volume inspired (VT) and the pressure generated (PP) (Kpa · l) [24].

Statistical analysis

Statistical analysis was performed by using paired student t-tests. Each investigation started and ended with the same type of flow pattern. All the parameters were compared in the following set of pairs, according to the two types of gas flow used: Constant-decelerating, decelerating-constant. Paired differences were also calculated between these start and end settings. p values of ≤ 0.05 were regarded as significant.

Results

Equipment testing

Table 2 shows the correlation equations for VT delivered by the ventilator measured with the Wright spirometer and the electronic respirometer when two peak flows (10 and 80 l/min) were used with the constant and decelerating WFs respectively. It is evident from the table that there is a good correlation between the two methods of VT measurement, although the intercept values were high for the lower peak flows particularly with decelerating WF. However, during the course of this study the minimum VT used was 600 ml and the lowest peak flow 30 l/min. Examination of the equations show the error of measurement of such VT to be less than 5%. In the same table the lower equation represent the correlation equation of the Wright spirometer and the graduated syringe and shows an excellent correlation with an intercept value of zero.

The accuracy of the ventilator VT delivery

Tidal volume values calculated from the output of the Fleisch pneumotachograph were within ± 5% of the set value. This was independent of peak flow, flow pattern, or the load on the ventilator. There were also a good correlation between these values and those measured by the Wright spirometer.

Table 2. The correlation equations for VT measured with the Wright spirometer and the electronic respirometer at two different peak flows (10 and 80 l/min. y = Electronic respirometer)

	10 l/min	80 l/min
Constant WF	y = 28.3 + 0.97x r = 0.99 sd = 15.6	y = 13.9 + 0.98x r = 0.99 sd = 33.0
Decelerating WF	y = 42.4 + 0.97x r = 0.99 sd = 16.2	y = 18.7 + 0.99x r = 0.99 sd = 13.9

y = 1.0233x; r = 0.99; sd = 35.17. The lower is the correlation equation of the Wright spirometer and the graduated syringe, y = Wright spirometer, sd = standard deviation

Waveform profile

The upper trace in Figure 1 shows the output obtained from the Fleisch pneumotachograph with constant and decelerating WFs when the ventilator was connected to the lung model. The lower trace shows flow and airway pressure signals in one patient for both WFs. The flow signals were obtained from the ventilator recording output.

The effect of using EIP of 0.55 s for 15 s

The increase in total static compliance was only evident after 2 min of EIP application, reaching maximal value at 15 min. After 2 min there was an increase of less than 5% of the control value, with no changes in total resistance.

Breathing mechanics

Seventeen observations in 14 patients (Table 3) showed that the decelerating WF produced significant reduction in peak pressure (PP) of 16% ($p<0.0001$), kinetic pressure (P K) of 9% ($p=0.0037$), plateau pressure (P PL) of 8% ($p=0.0016$), total resistance of the respiratory system of 31% ($p<0.0001$). Total static (Cst) and kinetic (Ck) compliances showed a significant increase of 17% ($p=0.024$) and 15% ($p=0.01$) respectively.

Although mean airway pressure (PAWP) was higher with the decelerating WF this was not significance. Pressure stress relaxation per unit initial pressure (SR/PK) was found to be significantly lower when the flow was changed from constant to decelerating ($p=0.04$). On returning to constant it increased by 9% which was not significant. The rate of fall in pressure stress relaxation (SR/EIP) was significantly lower with decelerating WF ($p=0.016$). The work of inspiration was found to be significantly lower by 22% ($p=0.004$) with the decelerating WF. In five of the patients where VD/VT ratio was measured, decelerating WF produced a reduction of 16% ($p=0.035$).

Gas exchange

Sixteen observations in 14 patients (Table 4) showed that the decelerating WF produced an increase of 14% ($p=0.027$) in PaO2 with no significant changes in PaCO2. In the nine patients where the (A-a)PO2 was calculated, the decelerating WF produced a decrease of 4% ($p=0.01$).

Table 3. Mean values ± SD and the difference between the means ± SD

	PP cmH_2O	PK cmH_2O	SR/PK mmH_2O/cmH_2O	SR/EIP cmH_2O/s	PPL cmH_2O	R $cmH_2O/l/s$	Cst ml/cmH_2O	Ck ml/cmH_2O	VT ml	VD/VT ratio	Pawp cmH_2O	W KP a·l
Constant WF												
$\bar{X}$	29.5	24.2	1.56	5.75	20.9	8.72	48.8	40.0	798	0.38	13.30	2.49
±SD	11.4	11.2	1.4	3.6	10.4	3.7	28.3	15.4	122	0.06	5.9	1.16
Decelerating WF												
$\bar{X}$	24.9	22.1	1.35	4.55	19.27	6.00	56.9	46.1	802	0.32	14.04	2.03
±SD	11.3	10.7	1.13	2.83	9.5	2.9	39.7	20.7	121	0.04	·6.0	1.14
N	16	16	16	16	16	16	16	16	16	5	15	16
$\Delta\bar{X}$	4.62	2.05	0.22	1.2	1.62	2.7	−8.1	−6.1	−3.7	0.06	−0.7	0.45
±SD	3.4	2.38	0.39	1.8	1.7	1.95	12.9	8.3	13.1	0.05	1.9	0.55
% Change	16%	9%	13%	21%	8%	31%	17%	15%	0.5%	16%	6%	18%
p	<0.0001	0.0037	0.04	0.016	0.0016	<0.0001	0.02	0.01	0.3	0.035	0.14	0.004
Constant WF												
$\bar{X}$	25.5	22.4	1.39	4.67	19.72	6.02	55.1	45.1	814	0.32	13.82	2.11
±SD	11.0	10.4	1.18	2.6	9.5	3.1	37.9	20.2	127	0.04	6.4	1.15
Decelerating WF												
$\bar{X}$	30.4	25.0	1.52	5.93	21.59	9.08	46.9	38.4	810	0.37	13.22	2.49
±SD	11.2	10.7	1.35	3.8	9.9	3.9	25.2	14.3	124	0.04	6.2	1.2
N	17	17	17	17	17	17	17	17	17	5	15	17
$\Delta\bar{X}$	−4.86	−2.6	−0.13	−1.27	−1.87	−3.06	8.2	6.63	3.7	−0.05	0.61	−0.38
±SD	2.77	1.98	0.31	1.62	1.5	2.19	14.3	9.18	12.7	0.02	1.35	0.2
% Change	19%	12%	9%	27%	9%	51%	15%	15%	0.5%	16%	4%	18%
p	<0.0001	<0.0001	0.95	0.005	<0.0001	<0.001	0.031	0.009	0.25	0.005	0.1	<0.0001

PP = pressure peak; PK = pressure kinetic; SR = pressure stress relaxation; pPL = pressure plateau, R = total resistance, Cst = total static, Ck = total kinetic compliances, Pawp = mean airway pressure, W = work of breathing

Table 4. Mean values ± SD for gas exchange and haemodynamics

	PaO_2 mmHg	$PaCO_2$ mmHg	PAO_2 mmHg	(A-a)PO_2 mmHg	CO l/min	HR B/min	BP mmHg	PAP mmHg	PAWP mmHg	CVP cmH_2O
Constant WF										
$\bar{X}$	73.73	36.36	313.9	246.3	5.02	88.5	84.5	22.33	11.0	10.0
± SD	25.4	5.5	65.9	69.1	1.7	10.8	13.5	5.05	5.8	6.0
Decelerating WF										
$\bar{X}$	84.30	35.3	315.0	236.1	4.98	84.4	83.2	23.33	12.7	9.3
± SD	31.2	4.9	67.4	72.3	1.6	12.3	12.5	5.7	4.6	5.9
N	15	15	9	9	5	8	6	6	6	5
$\Delta\bar{X}$	−1.41	1.06	−1.14	10.21	0.034	4.13	1.33	−1.0	−1.67	0.7
± SD	1.3	2.7	3.18	9.28	0.17	6.17	6.3	1.4	2.8	0.6
% Change	14%	3%	0.4%	4%	1%	5%	2%	4%	15%	7%
p	0.0009	0.16	0.31	0.011	0.67	0.1	0.63	0.14	0.21	0.052
Constant WF										
$\bar{X}$	88.65	35.27	321.8	236.1	5.04	86.0	84.2	22.67	12.0	9.7
± SD	38.3	4.9	67.0	69.7	1.5	11.8	12.1	5.5	5.0	5.9
Decelerating WF										
$\bar{X}$	80.03	35.3	322.2	245.0	5.08	85.3	82.4	26.17	11.8	9.3
± SD	34.7	4.7	66.1	66.8	1.6	10.0	13.5	5.4	5.3	6.5
N	16	16	10	10	5	8	6	6	6	5
$\Delta\bar{X}$	1.15	−0.03	−0.38	−8.93	−0.04	0.75	1.79	−3.5	0.17	0.4
± SD	1.06	1.85	2.37	7.86	0.19	7.3	5.8	6.19	1.17	1.19
%Change	10%	0.1%	0.1%	4%	1%	1%	2%	15%	1%	4%
p	0.0006	0.95	0.63	0.0058	0.69	0.78	0.49	0.22	0.74	0.5

See Material and methods for abbreviations

Haemodynamics

The decelerating WF produced no significant changes in pulmonary artery pressure (PAP), pulmonary capillary wedge pressure (PCWP), systemic blood pressure (BP), heart rate (HR), central venous pressure (CVP) or cardiac output (CO).

There were no significant changes between the beginning and the end set of measurements for all the variables, i.e. breathing mechanics, gas exchange and haemodynamics.

Discussion

The use of decelerating WF in patients on IPPV significantly lowered total resistance, increased total compliance (static and kinetic) of the respiratory system and improved oxygenation of the blood. It also significantly decreased alveolar-arterial gradient for oxygen, the ratio of VD/VT, and the work of inspiration. Furthermore, the decelerating WF produced the lowest peak pressure and plateau pressure. These improvement in lung mechanics and gas exchange occurred without deleterious effect on haemodynamics.

Watson [30–34] showed in conscious totally paralysed patients with normal lung function that inspiratory flow WFs had no effect on VD/VT ratio and lung compliance for any given duration of inspiration.

These findings have been confirmed by Bergman in dogs [4] and in anaesthetized subjects with normal cardio-respiratory function [5] and Adams et al. [1] in normo-, hypo- and hypervolaemic dogs.

In two separate studies Johansson [20, 21] showed that in patients requiring IPPV for various reasons and in anaesthetized patients with normal respiratory and cardiac function [19] the decelerating WF increased VD/VT; improved dynamic compliance but produced no significant changes in PaO2 when compared to constant WF.

Baker et al. [2, 3] showed that the most favourable WF for most physiological variables in dogs was the decelerating WF which resulted in decreased VD/VT and PaCO2, with increased PaO2, and total dynamic and lung compliances, when compared with the accelerating WF. Both decelerating and accelerating were significantly better than constant.

The discrepancies in the various studies are most likely due to the fact that changing the WF produced secondary changes in VT, frequency and I/E ratio. It is therefore essential, in order to determine the precise effect of changing the WF, that secondary changes in

these parameters do not occur. In most reported studies these criteria have not been fulfilled.

Watson [30–33] and Adams et al. [1] had used their own experimental ventilators. Watson's ventilator [34] was a pressure generator, the performance of which would be modified by the subjects respiratory mechanics. Watson's observations on the dead space were based on using the rebreathing technique to obtain PaCO2 which requires disconnecting the patient from the ventilator and assumed fixed a-v CO2 tension differences. With small changes in VD/VT ratio this technique is too insensitive to detect such changes.

In Adams et al. [1] study the geometry of the WF was different from the one used in our study. Three of the WFs used all had their peak flow in the middle third of inspiration, i.e. the variation in timing may be too small to have any significant effect on the inspiratory WF. Apparently the peak flows were also very similar.

Finally, in their protocol they stated that they always started with the sine WF, but they do not indicate if after changing the WF they return to the original WF, to verify the reproducibility of the system. The lung volume in their animals was also distorted by periodic hyperventilation, so that the compliance and lung volume at the beginning of each investigatory period presumably varied. This in turn could override any differences between the effects of different WFs. Nevertheless Adams et al. [1] had suggested that the inspiratory flow pattern, with a fixed inspiratory time and constant VT might affect the cardio-respiratory function by changing the cardiac output or altering the distribution of ventilation or blood flow within the lung.

Bergman [4–6] used different ventilators (pressure generators) to produce different WFs resulting in variations in inspiratory time, VT, frequency and expiratory pressure, thus making it difficult to relate the results to change in the WFs.

Similarly, there were some inconsistencies in Johansson's studies particularly in the constancy of the VT, as values indicated in the table differ from those calculated from the values given for compliance and end-inspiratory pressure [20–21]. Furthermore, Johansson and Löfström [19] used low VT (mean = 407 ± 63 and 411 ± 55 ml) with a calculated VD of 273 ml. Despite these inconsistencies the (A-a)PO2 was lower with the decelerating WF.

It seems therefore, that there is now good evidence demonstrating that decelerating WF improves gas distribution and uptake in the lung without deleterious effect on the circulation.

One explanation for this improvement is that the bulk of the VT is delivered early in inspiration due to a high initial peak flow which is followed by a slow decline in flow allowing more time for gas to enter the alveoli [28]. This may be particularly relevant to those alveoli with prolonged time constants. Furthermore, this early delivery of the VT is accompanied by a higher initial airway pressure allowing a longer residence time for the fresh gas in the alveoli thus favouring gas distribution [29].

We found that the decelerating WF produced a higher mean intrathoracic pressure which might be expected to overdistend those alveoli with a short time constant, thus compressing neighbouring alveoli and their capillaries. This would result in an increased physiological dead space. Furthermore, the higher mean intrathoracic pressure also has the potential for reducing cardiac output. In our study we found no evidence of this occurring. This is in agreement with Fairley [14] who stated that it should be possible to rase intrathoracic pressure enough to improve gas distribution, lowering VD/VT and improving oxygenation without exceeding the critical closing pressure of the pulmonary capillaries.

End-inspiratory pause (EIP) is a maintained inflation of VT. This is associated with a slow fall in airway pressure, although lung volume is not altered [10]. This fall in pressure results in part from stress relaxation. Other contributing factors are the redistribution of the inspired gas, absorption of gas into the blood stream and adiabatic compression of the gas in the lung. Don and Robson [11] reported that in anaesthetized patients, a maintained inflation of the respiratory system of 1 l, introduced at a rate of 1 l/s, is associated with a decline in airway pressure of approximately 0.2 cm H_2O for each cm H_2O inflation pressure. The rate of fall in pressure was found to be approximately 1.5 cm H_2O/s.

The findings in our study were 0.16 cm H_2O/cm H_2O and 5.75 cm H_2O/s respectively.

Fuleihan et al. [15] studied the effect of EIP on gas exchange in 10 ventilated adult patients with acute respiratory insufficiency. They showed that when they used an EIP of 0.6 and 1.2 s compared to a zero EIP there were no changes in arterial oxygenation, but VD/VT ratio and PaCO2 dropped significantly. The measurement in their study were made 45 to 60 min after EIP application.

In our study when EIP of 0.5 and 1.0 s was tested on two patients there were no changes in total compliance and resistance after the first min of EIP application, but there was 5% increase in total compliance with no changes in total resistance after 2 min of EIP application. It is highly unlikely therefore, that the use of 15 s EIP to obtain measurement could influence the gas distribution in our patients. Furthermore, the expired gas collection for CO2 concentra-

tion measurement was made after EIP had been discontinued.

Clinical implications

As the application of the decelerating WF results in a lower airway resistance and improved gas distribution it may be of benefit in those patients with high airway resistance. Similarly the improvement in PaO2 and (A-a)PO2 gradient also implies probable benefit in patients with severe $\dot{V}_A/\dot{Q}$ abnormalities. This would allow reduction in FIO2. In addition the prolonged alveolar residence of the gas could be beneficial in patients with impaired gas diffusion. Similarly, those patients with reduced compliance due to atelectasis may also benefited by the higher mean airway pressure.

Our results suggest therefore, that the decelerating WF should be more generally employed in patients requiring IPPV.

Acknowledgements. We acknowledge the help and cooperation of all the staff in the Intensive Therapy Unit. In addition the clerical assistance of Mrs. Betty Simpson, the word processing expertise of Mr. Nawzer Mehta, and the graphical expertise of Mr. Mike Birch.

References

1. Adams AP, Economides AP, Finlay WEI, Sykes MK (1970) The effects of variations of inspiratory flow waveform on cardiorespiratory function during controlled ventilation in normo-, hypo- and hypervolaemic dogs. Br J Anaesth 42:818
2. Baker AB, Babington PCB, Colliss JE, Cowie RW (1977) Effects of varying inspiratory flow waveform and time in intermittent positive pressure ventilation. I. Introduction and methods. Br J Anaesth 49:1207
3. Baker AB, Colliss JE, Cowie RW (1977) Effects of varying inspiratory flow waveform and time in intermittent positive pressure ventilation. II. Various physiological variables. Br J Anaesth 49:1233
4. Bergman NA (1963) Effect of different pressure breathing patterns on alveolar-arterial gradients in dogs. J Appl Physiol 18:1049
5. Bergman NA (1967) Effects of varying inspiratory waveforms on gas exchange. Anesthesiol 28:390
6. Bergman NA (1969) Effect of varying respiratory waveforms on distribution of inspired gas during artificial ventilation. Am Rev Respir Dis 100:518
7. Chackrabarti MK, Sykes MK (1976) Evaluation of the lung ventilator performance analyser. Anaesthesia 31:521
8. Cox LA, Almeida AP, Robinson JS, Horsley JK (1974) An electronic respirometer. Br J Anaesth 46:302
9. Cox LA, Chapman EDW (1974) A comprehensive volume cycled lung ventilator embodying feedback control. Med Biol Eng 12:160
10. Don H (1977) The mechanical properties of the respiratory system during anaesthesia. Int Anaesthesiol Clin 15(2):113
11. Don HF, Robson JG (1965) The mechanics of the respiratory system during anesthesia. Anesthesiol 26(2):168
12. Enghoff H (1938) Volumen Inefficax Bemerkungen zur Frage des schadlichen Raumes. Upsala Lak Foren Forh 44:191
13. Fahri LE (1966) Ventilation-perfusion relationships and its role in alveolar gas exchange. In: Collin G Care (ed) Advances in Respiratory Physiology. Edward Arnold, London, p 178
14. Fairley HB (1970) Artificial ventilation. In: Scurr C, Feldman S (eds) Scientific foundations of anaesthesia. William Heinemann, London, p 192
15. Fuleihan SF, Wilson RS, Pontoppidan H (1976) Effect of mechanical ventilation with end-inspiratory pause on blood-gas exchange. Anesth Analg 55(1):122
16. Herzog P, Norlander OP (1968) Distribution of alveolar volumes with different types of positive pressure gas flow patterns. Opuscula Medica 13:27
17. Hughes R, May AJ, Widdicombe JG (1959) Stress relaxation in rabbit's lungs. J Physiol (Lond) 146:85
18. Ingelstedt S, Jonson B, Nordstrom L, Olsson S-G (1972) A servo-controlled ventilator measuring expired minute volume, airway flow and pressure. Acta Anaesth Scand (Suppl) 47:7
19. Johansson H, Löfström JB (1975) Effects on breathing mechanics and gas exchange of different inspiratory gas flow patterns during anaesthesia. Acta Anaesth Scand 19:8
20. Johansson H (1975a) Effects on breathing mechanics and gas exchange of different inspiratory gas flow patterns in patients undergoing respirator treatment. Acta Anaesth Scand 19:19
21. Johansson H (1975b) Effects of different inspiratory gas flow patterns on thoracic compliance during respirator treatment. Acta Anaesth Scand 19:89
22. Lyager S (1968) Influence of flow pattern on the distribution of the respiratory air during intermittent positive-pressure ventilation. Acta Anaesth Scand 12:191
23. Mushin WW, Rendell-Baker L, Thompson PW, Mapleson WW (1980) Automatic ventilation of the lung. Third edn, Blackwell, Oxford
24. Nunn JF (1977) Applied respiratory physiology. Butterworths, London
25. Nunn JF, Bergman NA, Coleman AJ (1965) Factors influencing the arterial oxygen tension during anaesthesia with artificial ventilation. Br J Anaesth 37:898
26. Otis AB, Mckerrow CB, Bartlett RA, Mead J, Mellroy MB, Selverstone NJ, Radford EP (1956) Mechanical factors in distribution of pulmonary ventilation. J Appl Physiol 8:427
27. Otis AB (1964) Quantitative relationship in steady-state gas exchange. In: Fenn O, Rahn H (eds) Handbook of physiology, section 3. Respiration, vol 1. American Physiological Society, Washington D.C., p 681
28. Robinson JS (1978) Respiratory care and the principle of ventilation. In: Hanson GC, Wright PL (eds) The medical management of the critically ill. Academic Press, London, p 5
29. Robinson JS (1973) The influence of differing flow patterns on the gas distribution and uptake during artificial ventilation. 7th International Anaesthesia Postgraduate Course. H. Egermann, Vienna, p 277
30. Watson WE (1962a) Some observations on dynamic lung compliance during intermittent positive pressure respiration. Br J Anaesth 34:153
31. Watson WE (1962b) Transmural central venous pressure during intermittent positive pressure respiration. Br J Anaesth 34:278
32. Watson WE (1962c) Observations on physiological dead space during intermittent positive pressure respiration. Br J Anaesth 34:502
33. Watson WE (1962d) Observations on the dynamic lung compliance of patients with respiratory weakness receiving intermittent positive pressure respiration. Br J Anaesth 34:690
34. Watson WE, Spalding JMK, Smith AC (1962) A respirator pump to provide a variable waveform. Br J Anaesth 34:149

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