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Aspiration of dead space allows normocapnic ventilation at low tidal volumes in man

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Abstract *Objective:* Aspiration of dead space (ASPIDS) improves carbon dioxide (CO_2) elimination by replacing dead space air rich in CO_2 with fresh gas during expiration. The hypothesis was that ASPIDS allows normocapnia to be maintained at low tidal volumes (V_T).
Design: Prospective study.
Setting: Adult intensive care unit in a university hospital.
Patients: Seven patients ventilated for neurological reasons were studied. All patients were clinically and haemodynamically stable and monitored according to clinical needs.
Interventions: ASPIDS implies that, during expiration, gas is aspirated through a catheter inserted in the tracheal tube. Simultaneously, a compensatory flow of fresh gas is injected into the inspiratory line. ASPIDS was achieved with a computer/ventilator system controlling two solenoid valves for aspiration and injection.
Results: At the basal respiratory rate of 12.6 breaths min^{-1}, with ASPIDS V_T decreased from 602 to 456 ml, as did the airway pressures to a corresponding degree. $PaCO_2$ and PaO_2 remained stable. At a frequency of 20 breaths min^{-1}, with ASPIDS V_T was further reduced to 305 ml with preserved normocapnia. ASPIDS did not interfere with the positive end-expiratory pressure (PEEP) level. No intrinsic PEEP developed. All patients remained stable. No haemodynamic or other side effects of ASPIDS were noticed.
Conclusion: The results of this study suggest that ASPIDS may be a useful and safe modality of mechanical ventilation that limits alveolar pressure and minute ventilation requirements while keeping $PaCO_2$ constant.

Key words Mechanical ventilation · Dead space · Airway pressure · Barotrauma · Tracheal gas insufflation

Introduction

Barotrauma or volutrauma with alveolar and microvascular damage is a serious adverse effect of high airway pressures and overdistension that may result from efforts to achieve adequate gas exchange in patients with severe lung disease [1]. Such knowledge has incited approaches for reducing tidal volume (V_T) and airway pressure during critical illness such as extracorporeal CO_2 removal, high frequency ventilation, partial liquid ventilation and permissive hypercapnia. The ultimate value of these modalities remains unproved.

Another way to improve alveolar ventilation, thus increasing CO_2 clearance, is to minimise dead space ventilation. This can be achieved by diluting the CO_2-laden airway dead space with fresh gas, by either expiratory flushing of the airways [2, 3] or tracheal gas insufflation [4, 5, 6]. We have previously explored an alternative approach in animals,: aspiration of the dead space (ASPIDS) [7]**.** During ASPIDS, in the late part of expi-

Table 1 Patients characteristics

Patients	Sex	Age years	Weight Kg	Diagnosis	C_{RS} ml cmH_2O^{-1}	PaO_2/FiO_2	VD_{aw} ml
1	M	39	70	PNS assistance	87.8	428	190
2	M	80	80	PNS assistance	101.7	225	220
3	M	25	60	Brain Trauma	88.9	225	220
4	F	44	65	SAH	71.7	284	200
5	M	67	75	Brain Ischemia	64.1	137	230
6	F	80	50	Brain Ischemia	47.6	358	210
7	F	73	55	Brain Ischemia	44	391	220
Mean		58	65		72.3	294	213
± SD		22	11		21.8	106	14

Definition of abbreviations: C_{RS}: compliance of the respiratory system; VD_{aw}: airway dead space; M: male; F: female; PNS: post neurosurgery; SAH: subarachnoid haemorrhage

ration, the gas in the ventilator tubing, Y-piece, filter and tracheal tube is aspirated from the tip of the tracheal tube and replaced with fresh gas through the ordinary inspiratory line of the ventilator. Consequently, by reducing the airway dead space (VD_{aw}) that returns to the alveoli during inspiration, ASPIDS augments alveolar ventilation, allowing a decrease in V_T and airway pressure.

In a clinical setting the hypothesis was tested that V_T and airway pressures could be reduced with ASPIDS while CO_2 levels remained constant. A further hypothesis was that a reduction in dead space allowed the use of a higher respiratory rate (RR) permitting still lower V_Ts.

Materials and methods

The study, conducted according to the Helsinki principles, was approved by the Ethical Committee of the University of Naples "Federico II", and informed consent was obtained from patients' next of kin. Seven patients suffering from coma due to various neurological pathologies and requiring positive pressure ventilation were studied (Table 1). Patients were studied when they were stable with respect to clinical condition, intracranial pressure, haemodynamics and body temperature.

All patients were transorally intubated using cuffed endotracheal tubes with an inner diameter of 7–8.5 mm. The cuff was inflated and frequently tested to avoid air leakage. Volume-controlled ventilation at a constant inspiratory flow pattern was given with a Servo Ventilator 900 C (Siemens-Elema, Sweden). On average, minute ventilation was 7.6 l min^{-1}, RR 12.6 breaths min^{-1}, positive end-expiratory pressure (PEEP) 5 cmH_2O and FIO_2 0.38. Inspiratory time was 33% and post-inspiratory pause time 5%. A moisture exchanger and bacterial/viral filter (AC53/FE62NST, Europe Medical, Bourgen Bresse, France) and a connector to the Y-piece were used. In all patients monitoring included electrocardiogram, invasive or non-invasive arterial blood pressure, central venous pressure and temperature. An infusion of thiopental sodium 2.4 mg kg^{-1} h^{-1} was given to patients in light coma as clinically required. Blood gas analysis was performed on an ABL 505 blood gas analyser (Radiometer, Copenhagen, Denmark).

A computer/ventilator interface (CVI) conveyed measurement signals from the Servo Ventilator to an IBM compatible computer, and signals allowing the computer to control the ventilator in the opposite direction. The computer was equipped with a card for A/D and D/A conversion and digital in- and outputs (PC-30, Eagle technology, South Africa). The CVI was used for computer control of both the ventilator during measurements and the solenoid valves of the ASPIDS system (Fig. 1). Signals from the ventilator transducers representing airway pressure in the expiratory line (Paw), inspiratory and expiratory flow in the ventilator, and the CO_2 signal from a mainstream CO_2 analyser (CO_2 Analyzer 930, Siemens Elema, Sweden) were A/D converted at 50 Hz. The system was constructed and programmed at the Department of Clinical Physiology, University of Lund, Sweden.

The airway dead space was calculated from a plot of expired CO_2 against expired V_T according to Fletcher et al. [8, 9]. The elastic pressure volume curve (P_{el}/V) of the respiratory system was recorded during a 6 s long insufflation of gas following an expiration to the elastic equilibrium volume at zero PEEP [10, 11, 12, 13, 14]**.** Compliance of the respiratory system (C_{RS}) for the linear part of the P_{el}/V curve was calculated as previously described [15]. Peak airway pressure (Paw_{peak}), post-inspiratory plateau pressure (Paw_{plat}), and mean airway pressure (Paw_{mean}) were read from the digital display of the ventilator. Intrinsic positive end-expiratory pressure (PEEPi) was calculated as the difference between the Paw read at the end of expiration and Paw read after 3 s of an end-expiratory pause.

Aspiration of dead space gas and injection of a compensatory fresh gas together constitute ASPIDS (Fig. 1). The computer controlled the two solenoid valves of the ASPIDS system through the CVI. Aspiration was performed through a polyethylene end-hole catheter (ID 2 mm, OD 3 mm). This was inserted into the tracheal tube through a swivel adapter connecting the tube to the ventilator circuit. The tip of the catheter was positioned 2 cm proximal to the tip of the tracheal tube. On activation of the aspiration solenoid valve, the catheter was connected to a 3-l damping reservoir coupled to the central hospital vacuum source. In order to achieve a suitable flow rate of aspirated gas, the degree of vacuum in the reservoir was modified with an adjustable leakage valve leading from the reservoir to the atmosphere.

The injected fresh gas was delivered from a second outlet of the gas mixer that feeds compressed gas to the ventilator. A variable pressure regulator reduced the pressure of the gas. The injection line further comprised a solenoid valve, a variable pneumatic resistor and a tube connected to the inspiratory line of the ventilator circuit. The compensatory flow rate was adjusted by setting the pressure regulating valve and the resistor. The computer and the CVI together performed continuous tests of proper system func-

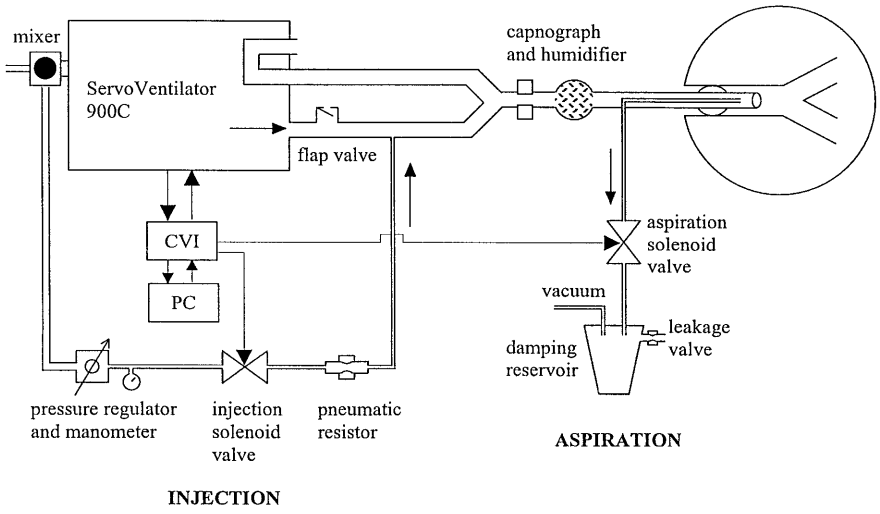

Fig. 1 Equipment. When the solenoid valves for aspiration and injection open under control of the personal computer (PC) and the computer/ventilator interface (CVI) ASPIDS is performed as described in the text

tioning. The ASPIDS valves could only be opened during expiration. A flap valve in the inspiratory line served as a safety measure against accidental development of a negative pressure in the circuit. Importantly, all alarm functions of the ventilator were continuously in function during treatment with ASPIDS.

Recognising the phases of ventilation, the computer was set simultaneously to open both valves from 50 % to 85 % of the expiratory time (Fig. 2). This period is denoted the ASPIDS phase. The volume of aspirated gas needed to wash out the dead space down to the tip of the tracheal tube during each breath (V_{wash}) comprises both the dead space of the tubing and the volume of gas expired by the patient during the ASPIDS phase:

$$V_{wash} = VD_{tubing} + VE_{late} \quad (1)$$

where VD_{tubing} is the dead space of the connections comprising humidifier, capnograph, connectors and the tracheal tube: it was determined to be 150–155 ml in vitro depending upon the size of the tracheal tube. VE_{late} is the volume expired during the ASPIDS phase: VE_{late} was measured as the integral of flow rate during a period corresponding to the ASPIDS phase, but before ASPIDS was started. To set the ventilator and the ASPIDS system the subsequent procedure was followed:

1. Minute ventilation (V_E) was reduced by ($VD_{tubing} \times RR$). The tidal volume to be used during ASPIDS (VT_{aspids}) was then read from the ventilator display.
2. VE_{late} was measured to calculate V_{wash} according to Eq. 1.
3. The injection solenoid valve was activated and injection flow adjusted until the expiratory tidal volume (VT_E) read on the ventilator was equal to ($VT_{aspids} + V_{wash}$).
4. The aspiration solenoid valve was activated. The leakage valve was adjusted until the VT_E read on the ventilator was brought back close to VT_{aspids}. In order to provide a slight surplus of compensatory gas over aspirated gas, VT_E was maintained a few millilitres higher than VT_{aspids}.

Procedure

The effect of ASPIDS in each subject was first studied at the basal RR chosen by the attending physicians, on average 12.6 breaths min^{-1}. While the patient was in a steady state, a blood gas sample was taken and data representing the basal ventilation were read. A study of mechanics and dead space was made. ASPIDS was then started as described. After 20 min, when changes in blood gas parameters would have stabilised [16], data collection was repeated and ASPIDS stopped. The mechanics were measured immediately after the return to basal ventilation, as the observations cannot be made during ASPIDS.

The frequency was increased to 20. V_E was increased so as to maintain a stable CO_2 elimination in ml/min^{-1} monitored with the capnograph. After a new steady state had been reached the procedure was repeated at the higher frequency.

Statistical analysis

All data are expressed as mean ± SD. Two-tailed Student's *t*-test was used to compare findings from different study periods. Significance was considered at $p < 0.05$.

Results

The airway dead space, from the Y-piece to and including the conducting airways was 213 ml on average (Table 1). The typical pattern of pressure and flow during basal ventilation and during ASPIDS is shown in Fig. 2. During ASPIDS the set PEEP level was maintained and PEEPi did not change at either the lower or higher RR (Fig. 2 and Table 2). All patients remained haemodynamically stable throughout the experimental period (Table 2). No side effects of ASPIDS were noticed.

At the basal respiratory rate of 12.6 breaths min^{-1}, V_E during ASPIDS decreased from 7.6 to 5.8 l min^{-1} (Table 2). V_T, Paw_{peak}, Paw_{plat} and Paw_{mean} decreased significantly. $PaCO_2$ was stable and other blood gas data showed no significant changes. C_{RS} was 72 ± 22 ml/cmH_2O^{-1} at basal ventilation and 64 ± 11 ml cmH_2O^{-1} immediately after ASPIDS ($p > 0.05$). At the frequency of 20 the baseline blood gases and haemodynamics

were similar to the baseline observations at the lower frequency (Table 2). At ASPIDS, V_T and airway pressures were reduced by the same proportion as at the lower frequency. During ASPIDS at the higher frequency V_T was reduced by nearly 50 % compared to the basal V_T at the lower frequency. C_{RS} was 72 ± 25 ml cmH_2O^{-1} at basal ventilation and 68 ± 28ml cmH_2O^{-1} after ASPIDS ($p > 0.05$).

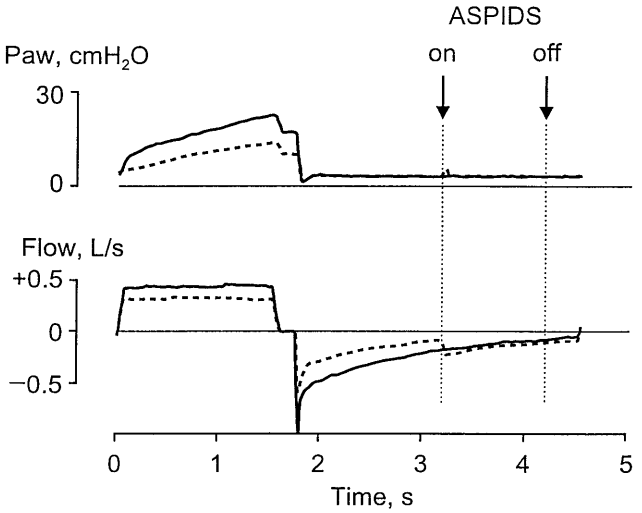

Fig. 2 Airway pressure (Paw) and flow rate at basal RR. Curves representing baseline ventilation (*continuous line*) are superimposed by those obtained at ASPIDS at reduced V_E (*interrupted lines*). *Dotted lines* indicate opening and closure of the aspiration and injection valves. Injection was performed at a slightly higher flow rate compared to aspiration

Discussion

The material chosen for this study reflects the limited objective to document the feasibility of ASPIDS in a clinical setting. No evidence of clinically significant lung disease was observed. The compliance of the linear segment of the pressure volume curve was, however, lower than in healthy subjects [12]. It is noteworthy that the linear segment represents the maximum slope of the pressure-volume curve and is higher than compliance calculated over an ordinary tidal volume. A "jet tube" in which extra lumen is used for aspiration is particularly useful for ASPIDS, as has been shown [7]. However, it was not considered acceptable to change the tube in the selected group of patients, particularly as the duration of the continuously supervised study was less than 1 h.

Our hypothesis that ASPIDS allows a substantial reduction of tidal volumes and airway pressures at a constant CO_2 level was confirmed. It was also verified that a particularly low V_T could be used at a high RR combined with ASPIDS. Previous results, obtained in animals with an ASPIDS system of an earlier generation, were confirmed [7]. The present computer/ventilator system allows continuous monitoring as well as recording of pressure and flow rate. The safety level of the system achieved by the combination of the internal safety system of the ventilator and continuous control of the function of the complete system allows its use in patients. However, the present system should still be regarded as experimental and it should be used under the surveillance of a well-informed operator. A system for routine clinical use would need to be modified. The measurement of tracheal pressure distal to the aspiration port, through a separate tube in a complex tracheal tube, is probably needed. When the tracheal pressure

Table 2 Mean values of ventilatory, gas exchange, and haemodynamic variables during the two studies at low and high respiratory rate

	Basal RR: 12.6		RR: 20	
	Baseline	ASPIDS	Baseline	ASPIDS
RR, breath min^{-1}	12.6 ± 0.8	12.6 ± 0.8	20	20
V_E, L min^{-1}	7.6 ± 0.7	5.8 ± 0.7**	9 ± 0.8	6.1 ± 0.8**
V_T, ml	602 ± 77	456 ± 76**	429 ± 74	305 ± 43**
Paw_{peak}, cmH_2O	26.1 ± 5.8	16.7 ± 2.1**	24.7 ± 3.4	15.3 ± 3.6**
Paw_{plat}, cmH_2O	20.5 ± 5.3	13 ± 1.8*	17.1 ± 2.3	11.1 ± 4*
Paw_{mean}, cmH_2O	11.3 ± 8.6	8.6 ± 1.1**	11.5 ± 1.7	8.2 ± 1.4**
$PEEP_{tot}$, cmH_2O	5.7 ± 0.9	5.7 ± 1	5.7 ± 0.9	5.7 ± 0.9
PEEPi, cmH_2O	0.37 ± 0.34	0.44 ± 0.44	0.59 ± 0.4	0.42 ± 0.13
$PaCO_2$, mm Hg	35.8 ± 3.4	35.5 ± 3.5	37.9 ± 6.6	38.3 ± 7.1
PaO_2, mm Hg	108 ± 30	97 ± 32	111 ± 28	111 ± 24
pH	7.45 ± 0.06	7.46 ± 0.05	7.44 ± 0.06	7.44 ± 0.05
HR, beats min^{-1}	97 ± 12	96 ± 20	102 ± 18	97 ± 14
MAP, mm Hg	80 ± 17	80 ± 19	82 ± 13	81 ± 19

Definition of abbreviations: RR, respiratory rate; V_E, minute ventilation; V_T, tidal volume; Paw_{peak}, peak pressure; Paw_{plat}, plateau pressure; Paw_{mean}, mean airway pressure; $PEEP_{tot}$: total positive end-expiratory pressure PEEPi, intrinsic positive end-expiratory pressure; HR, heart rate; MAP, mean artery blood pressure

In comparison between baseline and ASPIDS: * $p < 0.01$; ** $p < 0.001$

deviates inappropriately from the pressure measured in the ventilator, the ASPIDS system should automatically be switched off. The safety flap valve, which makes patient triggering impossible in the present system, would be void.

Injection of gas into the trachea through an additional catheter or lumen of the tracheal tube, i.e. tracheal gas insufflation (TGI), has been studied during the last decade [2, 3, 4, 5]. The efficiency of TGI reflects different mechanisms [6, 17, 18, 19]. TGI applied during expiration will dilute the CO_2 present in the dead space at the end of expiration. If the TGI is delivered as expiratory pulses it may even have an effect analogous to high frequency ventilation [2]. TGI applied during inspiration implies that dead space is bypassed. TGI is associated with some recognised drawbacks. The warming and humidification of compressed gas used for TGI constitutes a problem. Jet streams into the trachea may possibly be harmful. Expiratory TGI increases the intratracheal pressure, augments PEEPi and impedes expiration. When an expiratory flow rate continues throughout expiration, TGI may only dilute, and not flush the dead space free of, the CO_2. ASPIDS, which does not share the above-mentioned limitations of TGI, might be useful under some clinical circumstances.

In the adult respiratory distress syndrome, it is considered that a respiratory pattern should open up closed units and maintain aeration and stability throughout the respiratory cycle [20]. This may be accomplished by the use of rather high PEEP [21]. If ASPIDS allows the tidal volume to be reduced by about 150 ml, as in the present material, adequate PEEP might be used at constant CO_2 levels without the risks associated with high peak pressures and high tidal volumes [1].

In obstructive lung disease PEEPi is associated with a continuous expiratory flow. Because of uneven ventilation/perfusion of the lungs, the gas expired late is particularly rich in CO_2 [8, 9]. ASPIDS, which may eliminate CO_2 in a large part of the airway dead space, would then be particularly efficient. ASPIDS should be continued until the end of expiration, especially in the presence of a significant end-expiratory flow. In the present study the ASPIDS phase was interrupted at 85 % of the expiratory time in order to make observations of the effects on PEEP clearer.

We have shown that ASPIDS is technically feasible in a clinical setting and allows an important decrease in tidal volume and airway pressures. It merits further tests in patients with critical lung disease.

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