PULMONARY GAS EXCHANGE: A COMPARISON OF THE EFFECTS OF SIGHS USING HELIUM AND OXYGEN WITH THE EFFECTS OF POSITIVE EXPIRATORY PRESSURE PLATEAU

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PERIODIC HYPERINFLATIONS or "sighs" have been advocated by some to re-expand atelectatic lung[1] thereby increasing arterial oxygen tension (PaO_2) and pulmonary compliance and reducing alveolar-arterial oxygen gradient ($A\text{-}aDO_2$) and venous admixture or right-to-left pulmonary shunts (Qs/Qt).[2] Other investigators[3,4,5] have failed to demonstrate any improvement in oxygenation after "sighs". On the other hand, Positive Expiratory Pressure Plateau (PEPP) has been shown to improve oxygenation by a similar mechanism.[6]

Inert gases, particularly nitrogen,[7] are thought to have a direct role in preventing atelectasis. Helium is an inert gas which may be of benefit in this regard since its low solubility results in less absorption while its low density allows it to enter and leave small diameter airways easily, thus preventing "gas trapping", absorption and micro-atelectasis.[8]

This study is an attempt to compare "sighs" using oxygen and helium with PEPP and their effects on pulmonary gas exchange in a group of post-operative cardiac patients. They had cardiopulmonary by-pass procedures and had significant right-to-left shunts in the immediate post-operative period.

MATERIALS AND METHODS

The subjects for study were ten patients in the recovery room. They were free of chest disease or overt congestive heart failure. Median sternotomies with no entry into the pleural cavities had been done in all cases. Seven subjects had coronary by-pass graft procedures (CABG) for refractory angina pectoris. Two subjects (M.S. and M.B.) had mitral valve replacement (MVR) for refractory heart failure; the remaining one (D.P.) had a repair of a ventricular septal defect (VSD). The clinical details are shown in Table I.

All subjects were intubated and on controlled ventilation with varying concentrations of oxygen using the same BIRD Mark 7 Respirator. They were ventilated with tidal volumes which ranged from 7 to 10 cc/Kg without "sighs" prior to the study period when the control or "initial" samples were taken. Endotracheal suction for secretion removal was performed on all subjects prior to the study period. They had indwelling arterial and central venous lines for continuous monitoring of blood pressures and pulse waves and for sampling of blood for analysis.

Two separate series of four "sighs," 20 seconds in duration and 30 cm water

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TABLE I

DETAILS OF PATIENTS STUDIED

Patient	Age	Sex	Weight (Kg)	Surgical procedure
PC	51	M	78.2	Coronary artery bypass graft
MB_1	28	F	56	Mitral valve replacement
JT	50	M	83.6	Coronary artery bypass graft
DP	54	M	76	Ventricular septal defect repair
GMcI	55	M	70.8	Coronary artery bypass graft
PR	42	M	87	Coronary artery bypass graft
AP	50	M	75.4	Coronary artery bypass graft
BMcM	51	M	68.5	Coronary artery bypass graft
MS	35	F	64	Mitral valve replacement
MB_2	54	F	61.5	Coronary artery bypass graft

pressure in magnitude were administered over 80-second intervals, using 100 per cent oxygen and 100 per cent helium with an Ambu bag. The terminal outlet of the bag was blocked to prevent dilution with room air. PEPP was applied by submerging the expiratory line 5 cm below water surface. The inflating pressures and tidal volumes were monitored at the endotracheal connector site. Ventilator settings with PEPP were adjusted with the aid of a Wright's Respirometer and a stopwatch to maintain the tidal volumes and time cycling present before the study, to prevent hypoventilation resulting from the increased airway resistance due to PEPP.

End-tidal gas samples were collected at the endotracheal connector site in 50 cc syringes and blood samples were taken for analysis 10 minutes after each series of "sighs" or after 10 minutes of PEPP. The 10-minute period was allowed for alveolar-arterial-venous equilibration of carbon dioxide to a 90 per cent limit under controlled ventilation.[9]

Monitoring of alveolar carbon dioxide concentrations was done by connecting an endobronchial catheter to an infra-red rapid carbon dioxide analyzer* previously calibrated with Scholandered gas samples. Alveolar oxygen concentration was based on oxygen concentration in the end-tidal gas samples. These were measured by a paramagnetic oxygen analyzer† previously calibrated with Scholandered samples.

Arterial blood gas tensions and pH were measured with a Radiometer‡ pH/blood gas monitor and microelectrode unit at 37 degrees Centigrade (°C). Calibration of this apparatus was made before and after each set of determinations. Corrections to body temperature were made when necessary. Samples were taken in heparinized plastic syringes, immediately iced and placed in a refrigerator at 4° C and analyzed within 30 minutes.

Alveolar to arterial oxygen gradients and carbon dioxide gradients were derived directly, e.g. $AaDO_2 = P_{ET}O_2 - P_aO_2$ where $P_{ET}O_2 = (\%O_{2ET})\ (P_B - P_{H_2O}) = P_AO_2$ in the shunt equation. The equation used for calculating shunt in the present study was:[10]

$$Qs/Qt = \frac{C_cO_2 - C_aO_2}{C_cO_2 - C_{\bar{v}}O_2} = \frac{0.0031\ (P_AO_2 - P_aO_2)}{C_aO_2 - C_vO_2 + 0.0031\ (P_AO_2 - P_aO_2)}$$

*Beckman Medical Gas Analyzer LB-1.
†Beckman Model E2 Rapid Oxygen Analyzer. ‡Copenhagen.

where Qs/Qt is the ratio of quantity of blood flowing through the shunt to total quantity of blood flow (cardiac output) and C_cO_2, C_aO_2, $C_{\bar{v}}O_2$ are oxygen contents of pulmonary capillary, arterial and mixed venous blood, respectively. When P_aO_2 is greater than 150 mmHg and haemoglobin is fully saturated the right-hand portion can be used.[10] When the P_aO_2 was less than 150 mmHg and haemoglobin is not saturated, C_cO_2 equalled 0.0031 (P_AO_2) + S_cO_2 (1.34) (Hb)[11,12] and C_aO_2 equalled 0.0031 (P_aO_2) + S_aO_2 (1.34) (Hb).[11,12] $C_aO_2 - C_{\bar{v}}O_2$ was assumed to be six volumes/100 ml.[10] This was checked with the calculated oxygen content using arterial and central venous blood, which approximates true mixed venous blood, and no significant difference was found in this study.

Statistical evaluations were done using standard methods of calculation based on the "Student t-test" and Bessel's correction of the sample standard deviation.[13]

Results

End-tidal oxygen concentrations immediately after the 100 per cent helium "sighs" were 20–25 per cent and no anoxia occurred. Cardiac output was not measured but no significant change occurred in heart rate, arterial pulse wave form, arterial blood pressure, central venous pressure or urine output during the study period.

Pulmonary gas exchange is summarized in Table II. Statistical analysis of these results are shown in the following table.

Discussion

This study showed that PEPP increased arterial oxygen tension in the post-operative patient and confirmed its beneficial effect as documented by other authors.[6,14,15,16,18]

"Sighs" performed with 100 per cent oxygen and 100 per cent helium failed to improve oxygenation of arterial blood in post-operative patients. Although ventilation improved after "sighs" of oxygen or helium as demonstrated by the increased $AaDCO_2$ gradient, there was no reduction in $AaDO_2$ gradient or in venous admixture. Despite the theoretical advantages of helium in preventing airway collapse,[8] its use with "sighs" was as ineffective as oxygen in the correction of perfusion abnormalities.[17,18] This improvement in oxygenation is likely to be due to the reduction in venous admixture (Qs/Qt) as a result of PEPP, as demonstrated in the patients in this series as well as in others.[6,14,16] There is no evidence that other mechanisms responsible for improved oxygenation are involved. The partial pressure of oxygen in alveolar gas ($P_{ET}O_2$) showed no significant increase; there is no change in $PaCO_2$ or in $AaDCO_2$ to suggest improvement in ventilation or improvement in ventilation-perfusion ratio. McIntyre *et al.*[6] suggested that PEPP decreases venous admixture by the prevention of collapse of terminal airspace during expiration and thus PEPP may prevent atelectasis. Changes in diffusion probably did not play a significant role. The decrease in $AaDO_2$ with PEPP corroborates this.

The use of PEPP did not result in an increased $P_{ET}O_2$ as expected. When the Air-Mix venturi is functioning, as it was in this study, the BIRD respirator is a

TABLE II

THE EFFECTS OF "SIGHS" AND PEPP ON PULMONARY GAS EXCHANGE

Group	Patient	$P_{ET}O_2$*	P_aO_2*	A-aDO_2*	P_aCO_2*	P_ACO_2*	A-aDCO_2*	Qs/Qt (%)
Initial	PC	440	245	195	34.0	28.6	5.4	9.2
	JT	389	209	180	29.0	29.6	0.6	8.5
	DP	564	265	299	38.0	35.7	2.3	13.4
	GMcI	550	287	263	32.0	30.0	2.0	12.0
	PR	264	195	69	44.8	45.5	0.7	3.4
	AP	655	381	274	36.5	30.0	6.5	15.6
	BMcM	543	352	191	40.0	32.8	7.2	9.8
	MS	556	320	236	39.5	39.2	0.3	10.9
	MB_1	374	289	95	41.0	35.0	6.0	4.7
	MB_2	445	292	153	36.0	35.6	0.4	7.3
Mean		478	284	196	37.1	34.2	3.14	9.5
±s.d.		±36.7	±18.6	±22.6	±1.44	±1.58	±0.85	±0.96
"Sigh" (O_2)	PC	415	245	170	29.0	21.4	7.6	8.1
	JT	403	176	127	28.0	27.1	0.9	6.2
	DP	545	280	265	35.2	34.2	1.0	12.0
	GMcI	553	281	272	31.8	27.1	4.7	12.3
	PR	261	210	51	43.8	44.2	0.4	2.4
	AP	660	370	290	33.5	30.0	3.5	15.0
	BMcM	550	325	225	37.2	32.1	5.1	11.6
	MS	545	311	234	36.5	28.6	7.9	10.9
	MB_1	400	299	101	40.8	34.2	6.6	5.1
	MB_2	406	238	168	31.5	30.0	1.5	8.0
Mean		474	273	191	34.7	30.9	3.83	9.2
±s.d.		±36.8	±19.1	±24.0	±1.59	±1.72	±0.83	±1.11

TABLE II (*Concluded*)

Group	Patient	$P_{ET}O_2$*	P_aO_2*	A-aDO_2*	P_aCO_2*	P_ACO_2*	A-aDCO_2*	Qs/Qt(%)
"Sigh" (He)	PC	416	260	156	29.5	28.6	0.9	7.4
	JT	353	196	157	25.0	28.6	3.6	7.5
	DP	570	252	318	34.0	32.1	1.9	14.1
	GMcI	516	302	214	27.5	24.2	3.3	10.0
	PR	256	232	24	44.5	42.8	1.7	1.1
	AP	604	352	252	34.5	28.5	6.0	13.0
	BMcM	545	318	227	38.0	31.4	6.6	11.7
	MS	546	302	244	36.0	38.6	2.6	11.2
	MB_1	378	300	78	35.8	31.4	4.4	3.9
	MB_2	388	245	143	30.0	27.8	1.2	6.9
Mean		459	276	184	33.5	31.0	3.21	8.7
±s.d.		±36.8	±14.0	±26.2	±1.7	±1.57	±0.55	±1.23
PEPP	PC	478	315	163	28.0	27.1	0.9	7.7
	JT	324	225	99	32.5	28.6	3.9	4.8
	DP	603	292	311	46.0	39.2	6.8	13.8
	GMcI	578	340	238	36.2	32.4	3.8	10.9
	PR	254	245	9	46.0	48.5	2.5	0.5
	AP	618	410	208	38.5	35.6	2.9	10.8
	BMcM	515	332	183	43.0	35.7	5.3	9.4
	MS	624	350	274	36.0	32.1	3.9	12.4
	MB_1	410	306	104	45.0	38.0	7.0	5.1
	MB_2	368	275	93	27.0	28.5	1.5	4.6
Mean		476	309	168	37.8	34.6	3.50	8.0
±s.d.		±41.5	±17.0	±27.9	±2.14	±1.82	±0.62	±1.25

*Units of mm Hg.

TABLE III

SUMMARY OF STATISTICAL ANALYSIS

1. A highly significant increase in PaO_2 ($p = 0.01$) with PEPP and no significant change with "sighs" of oxygen (O_2) or helium (He).
2. A significant decrease in $AaDO_2$ with PEPP but no significant change with "sighs" of O_2 or He.
3. A highly significant decrease in Qs/Qt ($p = 0.01$) with PEPP and no significant change with "sighs" of O_2 or He.
4. A highly significant decrease in $PaCO_2$ ($p = 0.01$) with "sighs" of O_2 or He and no significant change with PEPP.
5. A significant increase in $AaDCO_2$ ($p = 0.05$) with "sighs" of O_2 and no significant change when "sighs" of He or PEPP were used.
6. No significant difference between $PaCO_2$ resulting from "sighs" of O_2 compared to "sighs" of He.
7. No significant change in $P_{ET}O_2$ with "sighs" or PEPP.

constant pressure generator.[19] Thus as patient resistance increases or as PEPP is added or increased, increased back pressure results and one would expect increased oxygen flow (the driving gas) to cycle the BIRD ventilator. The alteration in ventilator settings to counteract the decreased flow rates, peak pressure changes, decreased tidal volumes and cycling changes was not accompanied by a significant change of $P_{ET}O_2$ with 5 cms of PEPP. This suggests the pattern of ventilation initially and after PEPP was similar.[20] A recent study by Davies,[21] using a test lung, has graphically shown the detrimental changes which may occur with PEPP using a BIRD Respirator and emphasizes that the respirator will be dangerously ineffective if mechanical defects in the venturi, diaphragms or cycling system are present.

Adverse effects of PEPP, e.g. tension pneumothorax, decreased cardiac output, pulmonary capillary collapse, hypoventilation and/or arrhythmias did not occur. The use of a small pressure (5 cm H_2O) for PEPP avoids complications and is adequate for improvement in oxygenation. $AaDO_2$ and Qs/Qt changes can be monitored as indices of improvement in each subject.

CONCLUSIONS

PEPP (with 5 cm of water) is superior to "sighs" in the post-operative patient with right-to-left shunts of 8 per cent or more. Use of PEPP results in a highly significant decrease in shunt (Qs/Qt) and highly significant increases in PaO_2. PEPP applied on an intermittent basis may be anticipated as being more useful than "sighs" in the prevention of shunts and/or atelectasis. "Sighs" with oxygen or helium improve ventilation but not perfusion in the post-operative patient with a significant right-to-left shunt.

The BIRD Respirator can be adapted to function with PEPP and this increases the number of clinical situations where PEPP may be used.

SUMMARY

A comparison of periodic hyperinflations or "sighs" and positive expiratory pressure plateau (PEPP) was done in ten post-operative cardiac patients. Highly

significant decreases in shunts (Qs/Qt) and highly significant increases in P_aO_2 occurred with PEPP. Improvement in ventilation occurred with "sighs" but no change in oxygenation or perfusion was observed. A BIRD respirator was used and was adapted to PEPP.

Résumé

On a fait une comparaison entre l'hyperinflation périodique ou "soupirs" et le plateau de pression positive à l'expiration (PPPE) chez dix cardiaques en période post-opératoire. Il y a eu une diminution importante des shunts et une augmentation importante de la P_aO_2 avec le PPPE. Il y a eu amélioration de la ventilation avec les "soupirs" mais on n'a observé aucun changement dans l'oxygénation ou la perfusion. On a utilisé un respirateur Bird et on l'a adapté au PPPE.

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