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Research Article

UNILATERAL HYPOVENTILATION IN MAN DURING TEMPORARY OCCLUSION OF ONE PULMONARY ARTERY *

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Occlusion of the right or left pulmonary artery forces the opposite lung to perform the total gas exchange. The alveolar ventilation of the one functioning lung must increase if it is to maintain normal arterial blood oxygen and carbon dioxide tensions (1). One would predict that total ventilation would increase about 80 to 90 per cent to achieve the necessary increase in alveolar ventilation. Söderholm, however, found only a 30 per cent increase in total ventilation when one pulmonary artery was occluded even though arterial blood oxygen saturation, carbon dioxide tension and pH did not change (2). Other investigators (3, 4) have noted even smaller changes in total ventilation during temporary unilateral pulmonary arterial occlusion (TUPAO). If tidal volume, frequency of breathing and oxygen consumption remain the same, these findings could be explained only if some or all of the ventilation of the nonperfused lung were shifted to the functioning lung.

Moore, Humphreys and Cochran (5) have shown that such a shift of ventilation did occur in most dogs when one pulmonary artery was occluded by tightening a ligature brought out through the chest wall. They attributed this shift to a loss of erectile support of the vascular bed in the lung. Venrath, Rotthoff, Valentin and Bolt (6) also reported a redistribution of ventilation in dogs when one pulmonary artery was occluded by a balloon. They believed that the shift was caused by the low CO_2 on the nonperfused side, which is consistent with Nisell's observation (7) that bronchoconstriction occurred in excised cat lungs when alveolar CO_2 was reduced. On the other hand, Folkow and Pappenheimer (8), Julian, Travis, Robin and Crump (9), and Carlens, Hanson and Nordenström (10) found no significant shift of ventilation during TUPAO.

We decided to reinvestigate the effect of unilateral cessation of pulmonary blood flow on the distribution of ventilation in dogs and man. In dogs (11) we have confirmed the findings of Nisell and of Venrath and co-workers that the low alveolar P_{CO_2} in the lung with no pulmonary arterial blood flow produces bronchoconstriction which results in redistribution of ventilation favoring the opposite lung. Our present studies indicate that such redistribution also occurs in man during TUPAO and is initiated by the same mechanism—i.e., the decrease in alveolar P_{CO_2} in the nonperfused lung.

METHODS AND MATERIALS

The plan of the investigation involved *a*) studies during and after temporary unilateral pulmonary arterial occlusion in patients before bronchospirometry and *b*) repetition of the studies in the same patients during bronchospirometry. Table I gives the physical, clinical, and pulmonary function data for the patients; all of the patients had some pulmonary disease. Control and TUPAO studies were performed 9 times in 8 patients without bronchospirometry, and once in each of the 9 patients during bronchospirometry.

Studies before bronchospirometry. With the patient supine on a fluoroscopy table, we placed a triple lumen cardiac catheter in the pulmonary artery of choice, via an antecubital vein. We then inflated a balloon near the tip of the catheter to obstruct the flow of mixed venous blood to that lung (3). The first measurements were made during the period of vascular occlusion; this allowed the control studies to follow after deflation of the balloon and eliminated any intervening period of fluoroscopy and manipulation of the catheter. We collected expired gas in a Tissot spirometer which recorded tidal volume and frequency of breathing. After three preliminary collections, we made the final collection for a 3 minute period; we drew systemic and pulmonary arterial blood samples during the second minute. We

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TABLE I

Clinical and routine pulmonary function data of ten patients studied *

Patient	Age, Sex BSA	Diagnosis	VC	MBC	RV/TLC	SB O_2	Maximal flow rates Before/After isoproterenol aerosol MEFR	MIFR	Sa_{O_2} Breathing air	Pa_{O_2} Breathing O_2	Dco
			% *predicted*		%	% N_2	*L./min*		%	*mm Hg*	% *predict.*
A.D.	59 M 1.82	Bilat. nodular scars, esp RUL	152		33	3.5	333/'	300/'	96	595	68
F.Fr.	67 M 1.68	Ca in LLL old tbc RUL	117	119	33	4.0	273/375	186/214	91		64
J.J.	33 M 1.86	Bilat. midlung tbc infilt.	131	120	13	3.5	500/'	375/'	96	650	111
D.K.	23 M 1.71	Emphysema; bilat. bronchiect.	79	35	41	11.0	75/83	255/280	97		76
L.M.	63 M 1.48	Ca in RUL; inactive bilat. apical tbc	82	64	48	6.5	83/88	115/115	88	525	34
L.P.	50 M 1.91	Oat cell Ca in RLL	139		23	4.0	250/'	214/'	93		172
N.P.	60 M 1.73	LLL bullae	104	134	34	3.5	430/'	375/'	96		80
E.T.	52 M 1.74	Ca in RLL; inactive bilat. apical tbc	96	50		5.0	123/150	123/136	85	659	
E.D.	62 M 1.70	Ca + abscess in SS, LLL	105	81	46	5.0	57/117	96/110	89		
A.G.	71 M 1.98	RUL tumor	101		54	5.0	214/200	209/200	95	510	

* Abbreviations: VC = vital capacity; MBC = maximal breathing capacity; RV/TLC = residual volume per total lung capacity; SB O_2 = rise N_2 concentration between 750 and 1,250 ml expired after a single breath of pure O_2 (1); MEFR and MIFR = maximal expiratory and inspiratory flow rates (1); Sa_{O_2} = % arterial oxygen saturation; Pa_{O_2} = arterial oxygen tension; Dco = breath-holding diffusing capacity for carbon monoxide at rest; RUL = right upper lobe; SS, LLL = superior segment of left lower lobe; Ca = carcinoma.

measured the O_2 and CO_2 concentrations of expired gas (using the Scholander apparatus) and of blood (using the Van Slyke apparatus). We determined systemic arterial O_2 and CO_2 tensions with special electrodes (12). We calculated O_2 uptake, CO_2 elimination, respiratory quotient, cardiac output, physiological dead space and alveolar ventilation.

We calculated anatomic dead space during and after TUPAO from continuous recordings of expired CO_2 concentration and flow (13); CO_2 concentration was obtained with the Beckman Spinco apparatus and flow with a Fleisch pneumotachygraph. The lag of the CO_2 meter was measured at the time each patient was studied; it averaged 0.1 second.

In order to determine whether venous to arterial shunts were present during the vascular occlusion, we gave two patients (O.B. and A.G.) pure oxygen to breathe for 20 minutes before we deflated the balloon; they continued to breathe oxygen during and after balloon deflation. We measured the oxygen tension of arterial blood (12) just before deflation and 1 minute afterwards. Finally, to determine whether shunts were due to easily reversible atelectasis, we asked the patients to take several deep breaths and repeated the measurement of arterial blood P_{O_2}.

Studies during bronchospirometry. At the completion of the previous studies, we injected intravenously 0.75 to 1.0 mg of atropine sulfate and 75 to 100 mg of pentobarbital sodium as premedication for bronchospirometry. The patient then sat up and we anesthetized his airway with 1 per cent tetracaine, and passed a Carlens catheter (French 41) by indirect laryngoscopy. With the patient again supine and breathing air, we collected expired gas from each lung simultaneously in meteorologic balloons. We noted the time of collection and the respiratory rate so that we could calculate mean tidal volume. We determined the oxygen uptake, carbon dioxide output, minute and tidal volumes of each lung. Under fluoroscopic control we then reoccluded the pulmonary artery to one lung and repeated the above studies.

We then measured the anatomic dead space in each lung in sequence, starting with the perfused lung. During each determination, a sample of systemic arterial blood was drawn for measurement of CO_2 tension. Low tidal volumes, especially on the side of the pulmonary arterial occlusion, made measurements of the anatomic dead space difficult, but they were nevertheless reproducible within a limit of 5 ml.[1] While monitoring CO_2 and air flow of the lung with a blocked pulmonary artery, we then deflated the occluding balloon. Figure 1 shows an example of the increase in alveolar (end tidal) CO_2 and flow.[2]

[1] Calculated anatomic dead space volumes were corrected for 15 ml of apparatus dead space (catheter lumen and tubing) so that they represent volumes "below the carina."

[2] Low end tidal CO_2 values were useful as a criterion of the unilateral blockade of perfusion. However, oxygen

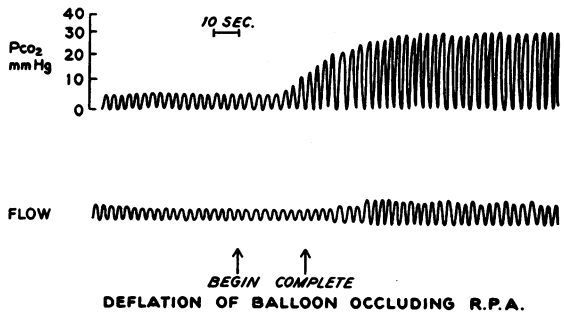

FIG. 1. INSPIRED AND EXPIRED P_{CO_2} AND FLOW TRACINGS IN PATIENT L.P. MONITORED AT THE EXTERNAL ORIFICE OF THE RIGHT LUMEN OF THE BRONCHOSPIROMETRIC CATHETER BEFORE, DURING AND AFTER DEFLATION OF THE BALLOON BLOCKING THE RIGHT PULMONARY ARTERY.

We again measured anatomic dead space on each side. The time between the dead space measurements of the right and left lung during either the control period of unilateral pulmonary arterial occlusion was less than 5 minutes.

In the last two patients (E.T. and E.P.) we tested the effect of administering 6 per cent CO_2 (in air or oxygen) to the unperfused lung to determine the effect of changing concentrations of CO_2 on the distribution of tidal volume between the two lungs.

uptake data showed that the occlusion was not always complete, even when an allowance was made for an enlarged bronchial collateral flow of blood and the mild hypoxemia associated with the patient's disease and the nature of the procedure (see Discussion).

RESULTS

Table II shows the results of nine studies in eight patients with the airway undivided. Associated with the occlusion, there was no significant increase in total ventilation. The unchanged arterial CO_2 tension suggests that alveolar ventilation remained normal even though there was no increase in total ventilation. Mean values for alveolar ventilation (total minus physiologic dead space ventilation) were 4.21 L per minute after, and 4.10 during the occlusion, a decrease of 3 per cent (not statistically significant). The mean oxygen uptake and carbon dioxide output each decreased 8 per cent (the differences again were not statistically significant) and the respiratory quotient stayed approximately the same. The physiologic dead space increased slightly while the mean anatomic dead space decreased slightly. The mean values for both the arteriovenous oxygen difference and the cardiac index decreased slightly but not to a statistically significant degree.

TABLE II

Studies "at the mouthpiece" (undivided airway) after and during temporary unilateral pulmonary arterial occlusion *

Patient	PA occluded side		$\dot{V}_E$	f	V_T	P_{aCO_2}	Physiol. V_D	$\dot{V}_A$	Anat. V_D	$\dot{V}_{O_2}$	$\dot{V}_{CO_2}$	RQ	AVD	CI
		min	*L/min*	*per min*	*ml*	*mm Hg*	*ml*	*L/min*	*ml*	*ml/min*	*ml/min*		*ml/L*	*L/min/m²*
A.D.	O		7.10	16	444	39	210	3.74		216	176	0.814	40	2.98
	R	15	6.84	11	622	41	313	3.40		205	168	0.818	43	2.63
F.Fr.	O		9.23	22	419	46	272	3.25	148	208	171	0.821	46	2.69
	L	15	9.92	22	451	45	301	3.30	130	227	175	0.770	42	3.21
J.J.	O		8.86	12	738	39	286	5.43	170	297	236	0.796	58	2.76
	R	20	10.48	14	749	40	376	5.22	140	300	236	0.787	45	3.60
D.K.	O		9.75	20	487	36	226	5.23		273	215	0.788	40	4.00
	R	20	11.61	21	552	34	256	6.23		282	240	0.851	42	3.93
	O		10.35	22	470	36	247	4.92		256	207	0.808	52	2.92
	R	15	11.30	20	565	34	307	5.16		264	198	0.750	38	3.95
L.M.	O		9.53	22	433	39	264	3.72	90	209	167	0.799	41	3.44
	R	75	10.05	24	418	37	264	3.71	100	174	158	0.910	42	2.81
L.P.	O		9.20	14	657	40	338	4.47	90	276	208	0.754	38	3.66
	R	15	6.06	14	433	42	239	2.71	80	178	134	0.798	33	2.54
N.P.	O		7.44	13	572	40			105					
	L	86	7.78	12	648	41			100					
E.T.	O		10.89	26	419	33	252			242	205	0.848	35	3.97
	R	20	9.60	24	400	35	250	3.60		206	167	0.815	39	3.03
Mean control			9.15	19	516	39	262	4.21	121	265	212	0.861	44	3.31
TUPAO		31	9.30	18	536	39	288	4.10	110	246	197	0.870	41	3.21

* Abbreviations: $\dot{V}_E$ =total ventilation; f =respirations per minute; V_T =tidal volume; P_{aCO_2} =arterial CO_2 tension; Physiol. V_D =physiologic dead space uncorrected for 100 ml apparatus dead space; $\dot{V}_A$ =alveolar ventilation; Anat. V_D =anatomic dead space; $\dot{V}_{O_2}$ =oxygen uptake; $\dot{V}_{CO_2}$ =carbon dioxide output; RQ =respiratory quotient; AVD =arteriovenous O_2 content difference; CI =cardiac index.

TABLE III

Ventilation studies during temporary unilateral pulmonary arterial occlusion and bronchospirometry *

Patient	Duration of PAO	Insp. gas	$\dot{V}_E$	f	Lung	V_T	% of total	$\dot{V}_{O_2}$ Bilat.	% of total	$\dot{V}_{CO_2}$ Bilat.	% of total	$Paco_2$	$PETco_2$	Anat. V_D
	min		*L/min*	*per min*		*ml*		*ml/min*					*mm Hg*	*ml*
A.D.	O	Air	6.50	10	R	297	48	302	50			40	35	
					L	323	52		50				35	
	R15	Air	10.94	13	R	280	35	299	2	218	5	40	4	
					L	527	65		98		95		39	
F.Fr.	O	Air	5.24	22	R	176	41	169	42			46	32	35
					L	253	59		58				28	45
	L10	Air	8.91	22	R	287	62	206	100	146	95	41	34	45
					L	156	38		0		5		3	25
J.J.	O	Air	17.99	15	R	489	44	324	50	318	48	29	22	45
					L	620	56				52		22	45
	R5	Air	21.59	18	R	519	46	444	16	401	21	34	4	15
					L	590	54		84		79		28	15
D.K.	O	Air	12.20	20	R	282	49	276	39	252	39	33	29	
					L	286	51		61		61		32	
	R5	Air	12.18	23	R	128	27	352	8	251	9	33	6	
					L	372	73		92		91		33	
L.M.†	O	Air	16.35	14	R	473	42	311	38	278	32	45	31	21
					L	665	58		62		68		26	14
	R10	Air	4.51	15	R	60	20	215	9	107	8	50	16	19
					L	240	80		91		92		39	13
L.P.	O	Air	10.03	20	R	155	38	443	56			43	29	20
					L	255	62		44				33	20
	R24	Air	9.31	19	R	40	9	284	0	238	1	45	4	20
					L	408	91		100		99		33	15
N.P.	O	Air	6.45	14	R	235	55	248	54			42	13	30
					L	195	45		46				12	25
	L13	Air	6.39	16	R	240	65	249	82			42	17	20
					L	130	35		18				9	15
E.T.	O	Air	14.52	21	R	219	36	294	32	206	33	32	20	
					L	395	64		68		67		20	
	R5	Air	18.94	26	R	198	28	349	13	302	15		9	
					L	499	72		87		85		21	
	R5	6% CO_2 Air	15.83	26	R	207	36	295	1	221	0		36	
					L	373	64		99		100		23	
E.P.	O	Air	10.05	12	R	485	60	333	72	263	66		33	
					L	322	40		28		34		30	
	L25	Air	12.65	16	R	611	80	269	96	235	95	38	30	
					L	151	20		4		5		4	
	L35	O_2 6% CO_2	14.80	20	R	385	54						28	
					L	325	46						43	
Mean	Control		10.37	17	Ips	277	45	299	44	260	37	38	26	31
					Con	337	55		56		63		27	
TUPAO			12.61	19	Ips	200	29	274	8	256	9	40	11	19
					Con	490	71		92		91		29	

* Abbreviations as in Table II plus: % = per cent of the bilateral function performed by that lung; $PETco_2$ = end tidal CO_2 tension; Ips = ipsilateral lung, of which pulmonary artery was or was subsequently blocked; Con = contralateral lung. Anatomic dead space values are corrected for 15 ml apparatus dead space (bronchospirometric tubing) so that the volumes are "below the carina."

† Morphine sulfate, 5 mg, given i.v. because of paroxysmal coughing, so decreased the ventilation that only the R:L distribution values are included in the mean data for the group.

Table III shows the results of TUPAO during bronchospirometry in nine patients. The total ventilation (mean) increased 22 per cent; however, the ventilation of the lung with the occluded pulmonary artery was only 29 per cent of the total as compared with a control value of 44 per cent. The anatomic dead space of that lung also decreased. When 6 per cent CO_2 in air or oxygen was breathed by the unperfused lung (Patients E.T. and E.P.), the fraction of the total ventilation of that lung returned to control values.

DISCUSSION

Unilateral pulmonary arterial obstruction in a normal individual must create a considerable increase in physiologic dead space, if ventilation of alveoli on this side continues. Under these conditions, total ventilation should increase about 80 per cent to maintain normal O_2 and CO_2 tensions in systemic arterial blood. Nevertheless, previous investigators (2–4) have not found an increase in total ventilation of the magnitude predicted, and our study confirms this.

There are several possible ways of explaining how arterial blood O_2 and CO_2 tensions can remain normal during TUPAO with little or no increase in over-all ventilation.

1. The pulmonary circulation may have been occluded by disease *before* the balloon occlusion. This was not true in our patients; bronchospirometric studies showed that the O_2 consumption of the experimental lung averaged 44 per cent of the total for the two lungs in the control period, and in only one patient (E.P.) was it less than 30 per cent of the total.

2. TUPAO may be accompanied by a decrease in body metabolism which occurs either coincidentally (change in environment) or by some reflex action. A decrease in O_2 consumption may be responsible in part in Patients L.P. (Table II) and L.M. (Table III), but the mean values of O_2 consumption were only 8 per cent lower than during the control period, which could not explain the data observed.

3. Breathing may change to a slower and deeper pattern so that alveolar ventilation may increase, although over-all minute ventilation does not. This mechanism may afford a partial explanation in Patient A.D. (Table II), but in the other patients there was either little change in respiratory frequency or there was an increase.

4. Anatomic dead space may decrease. The mean value decreased from 121 to 110 ml during TUPAO. This change is too small to explain the data observed.

5. Redistribution of inspired air may occur so that a large fraction of it enters perfused alveoli and little enters the nonperfused lung.

We believe that the major factor in the maintenance of normal arterial blood O_2 and CO_2 tensions during TUPAO is a redistribution of inspired air. The data obtained during bronchospirometry show unequivocally that the percentage of inspired air directed to the nonperfused lung decreases (mean decrease from 45 to 29 per cent) and that to the other lung increases (mean increase from 55 to 71 per cent). There is a suggestion that the shift is more effective when the airway is not divided, since the change in total ventilation was less than that noted during bronchospirometry. Atropine, used as premedication for bronchospirometry, may act to prevent bronchoconstriction, since it is known to increase the anatomic dead space (14). However, in our animal studies, atropine did not alter the ventilatory shift associated with the unilateral occlusion (11). Topical anesthesia may also play a role. It is also possible that the procedure of bronchospirometry leads to an increase in sympathetic nervous system activity and that this tends to decrease the response. We have been able to block the response completely by administering isoproterenol as an aerosol to the side with the arterial occlusion or by infusing epinephrine into the main pulmonary artery in animals (11). A mechanism such as this may have been responsible for the negative results in some previous studies in dogs (9) and patients (10). Patient J.J. failed to show any decrease in his ipsilateral tidal volume during TUPAO; his mean systemic arterial pressure rose gradually from a normal level to 150 mm Hg during bronchospirometry and TUPAO.

The mechanism responsible for the redistribution of inspired air in man appears to be the same as in the dog: TUPAO leads to decreased elimination of CO_2 ipsilaterally and to a decrease in alveolar CO_2 tension; this in turn leads to bronchoconstriction. Addition of CO_2 to the gas inspired

by the experimental lung, so that its alveolar P_{CO_2} remains normal despite TUPAO, prevents the redistribution (6, 7, 11). Pressure upon the bronchus by the inflated balloon in the pulmonary artery cannot be responsible, because bronchograms in two subjects could not demonstrate any encroachment upon the airway near the balloon (Figure 2). The low pressure distal to the occluding balloon in the pulmonary artery does not initiate the redistribution, as was postulated by Moore and colleagues (5), because the shift fails to occur when the balloon is inflated if the inspired gas contains 6 per cent CO_2 (11).

Airway-narrowing during TUPAO can proceed to the point of atelectasis. When we deflated the occluding balloon in two patients breathing oxygen, the systemic arterial blood O_2 tension decreased abruptly, indicating that there was a pulmonary arterial to venous shunt on that side. Since the arterial P_{O_2} rose to or toward control values after deep breathing, we suspect that the shunt was through atelectatic regions which could be opened up by voluntary hyperinflation (Table IV).

Unilateral bronchoconstriction may also be responsible for delayed exhalation by the involved lung. This was suggested by the alteration in the "alveolar plateau" (when expired CO_2 was monitored at high paper speed) during unilateral pulmonary arterial occlusion in some of the patients. As can be seen in Figure 3, the normal slight upward slope of the alveolar plateau was changed to an irregular downward sloping pattern, somewhat similar to that described by West and Hugh-Jones in studies in animals (15).

TABLE IV

The effect of deflation of pulmonary arterial (PA) balloon and of deep breathing on Pa_{O_2} (patients breathing O_2 throughout)

Patient	PAO	Arterial O_2 tension (mm Hg)		
		Before deflation of PA balloon	Immediately after deflation of PA balloon	After deep breathing
O.B.	L	520	420	520
A.G.	R	590	400	510

We were not always able to eliminate the oxygen uptake on the side of the TUPAO. Despite roentgenographic confirmation of the position of the balloon, the blockade was probably incomplete in two cases (J.J. and N.P., Table III). The mean residual oxygen uptake of the "unperfused" lung may be in part attributable to an enlarged collateral circulation bringing slightly desaturated bronchial arterial blood into the pulmonary capillary bed. The arterial oxygen tensions were in the range of 70 to 80 mm Hg; however, no correlation was present between the size of the oxygen uptake on the side of the occluded artery and the

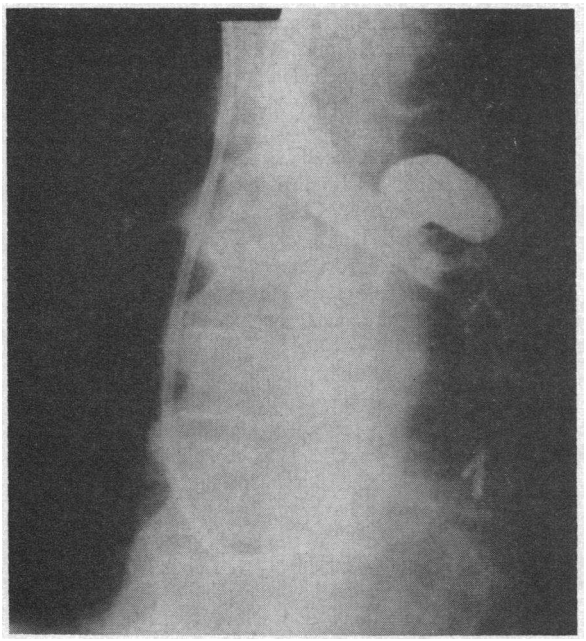

FIG. 2. LEFT-SIDED BRONCHOGRAM VIA THE LEFT LUMEN OF THE BRONCHOSPIROMETRIC CATHETER IN PATIENT F.FR. DURING TEMPORARY OCCLUSION OF HIS LEFT PULMONARY ARTERY.

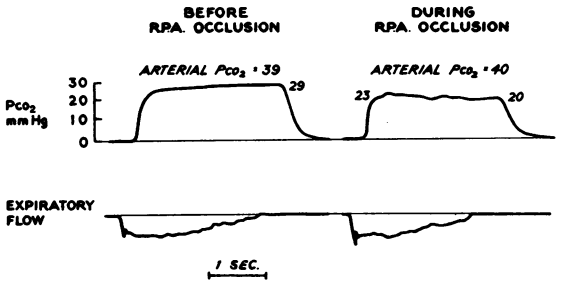

FIG. 3. CO_2 AND AIR FLOW TRACINGS AT THE MOUTHPIECE (UNDIVIDED AIRWAY) BEFORE AND DURING RIGHT PULMONARY ARTERIAL OCCLUSION IN PATIENT J.J. Note the changes in the "alveolar plateau" as well as the arterial-alveolar CO_2 tension differences associated with the occlusion.

arterial oxygen tension or saturation before vascular occlusion when the airway was undivided. The CO_2 output of the "unperfused" lung was consistently greater (mean of 9 per cent of the bilateral CO_2 output) than the contribution of that lung to the total oxygen uptake. The high respiratory quotient resulting is explainable on the basis of the high CO_2 gradient and ready diffusibility of this gas from the bronchial collateral (systemic arterial) blood to the alveoli of the lung whose supply of mixed venous blood is blocked.

SUMMARY

1. In eight patients with chronic lung disease, studied during and after temporary unilateral pulmonary arterial occlusion (TUPAO) in order to determine the ventilatory response to one-sided obstruction of the flow of mixed venous blood: *a*) On the average, the total ventilation was unchanged. *b*) At the same time, the mean arterial P_{CO_2} did not rise, indicating that alveolar hypoventilation had not occurred. *c*) The physiologic dead space increased less than might have been expected considering that 45 per cent of the total ventilation and oxygen uptake was originally performed by the lung whose pulmonary artery was later blocked. *d*) In several cases the "alveolar plateau" of CO_2 became irregular during the occlusion and sloped downward; this was interpreted as delayed emptying of the lung whose CO_2 output was curtailed from lack of perfusion.

2. In nine patients, studied by bronchospirometry and unilateral pulmonary arterial occlusion: *a*) After obstructing the flow of blood to one lung the tidal volume of that lung decreased so that its contribution to the total ventilation diminished from a mean of 45 to 29 per cent during the occlusion. *b*) During unilateral pulmonary arterial occlusion the anatomic dead space in that lung (below the carina) decreased from a mean of 31 ml before, to 19 ml during occlusion. *c*) Although the total ventilation increased by a mean of 22 per cent, under these circumstances the shift in the distribution of ventilation allowed the contralateral lung to increase its ventilation by a mean value of 63 per cent (5.73 L per minute before, 9.32 during occlusion); the normal arterial P_{CO_2} figures (mean of 38 before, 40 mm Hg during) attest to the magnitude of this shift. *d*) Allowing the "unperfused" lung to breathe 6 per cent CO_2 in two cases resulted in a return to the preocclusion values for distribution of inspired gas to the right and left lungs.

3. We suggest that the immediate reduction of ventilation in one lung following the occlusion of its pulmonary artery is due to bronchoconstriction which occurs in response to local airway hypocapnia and leads to regional atelectasis.

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