VENTILATION AND BLOOD GASES IN ANAESTHETIZED PATIENTS*

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RECENTLY the safety of intermittent positive pressure breathing (IPPB) from the standpoint of oxygenation has been questioned. An increase in physiologic dead space (V_D (phys)) and ventilation-perfusion inequality have been demonstrated during IPPB in both awake and anaesthetized subjects.[1-11] An anatomical shunt with venous admixture may result from ventilation with constant volumes. The degree of this disturbance, the time relationships between onset and maximum hypoxaemia, and its clinical significance remain to be evaluated.

The purpose of this study was to evaluate the significance of blood gas changes during anaesthesia and IPPB. Although evidence is available to the contrary, we wanted to test the validity of the assumption that during the awake, spontaneously breathing state and the anaesthetized IPPB state similar inhaled oxygen tensions ($P_{I_{O_2}}$), tidal volumes (V_T), and respiratory rates (f) should provide similar blood gas values in the same patient. Results proved that the assumption was not valid, although in many instances higher arterial oxygen tensions occurred at tidal volumes considerably less than are used during the management of clinical anaesthesia.

EXPERIMENTAL METHOD

Surgical patients were placed on the operating table one hour after the intramuscular injection of 100 mg. pentobarbital and 0.5 mg. atropine. A Cournand needle was placed in the brachial artery and an intravenous infusion of 5 per cent dextrose and water initiated. A mouthpiece was inserted and a nose clip applied. After a tight seal was ensured, a recently calibrated Wright respirometer was attached to measure expiratory V_T and minute volume ($\dot{V}_E$). Two arterial samples were withdrawn in the conscious state, the first while the patient was breathing air. A non-rebreathing valve and reservoir bag with 100 per cent oxygen were then attached. This was followed by 5 minutes of denitrogenation and, in turn, succeeded by 5 minutes of inhalation of 50 per cent oxygen in nitrogen through the same breathing system. The second arterial sample (the awake control) was then withdrawn while $\dot{V}_E$ and f were again measured.

Thiopental was then injected intravenously followed by succinylcholine. The trachea was then intubated. Fifty per cent O_2 in N_2O-halothane was inhaled and the patients' respirations were controlled with the same pressure-regulated ventilator during all subsequent measurements. This O_2 concentration was maintained and monitored at a point distal to the inspiratory valve in a circle CO_2 absorption

*This investigation was supported by Public Health Service Research Grant HE-06429 from the National Institutes of Health.

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system by means of a Beckman D-2 paramagnetic analyser. The patient was given periodic injections of d-tubocurarine to ensure apnoea while on the ventilator with the same VT and f as his awake control. In a few patients either VT or f was varied, after which arterial samples were withdrawn and volume measurements made.

Blood samples were withdrawn at 15-minute intervals after the ventilator was set to inflate the lungs at the control values. Surgery lasted from 30 minutes to five hours in the 13 patients studied. The arterial blood was placed in heparinized syringes and iced for analysis of arterial oxygen tension (Pa_{O_2}) by means of an Astrup radiometer unit and an oxygen electrode calibrated with gases. The pH meter and equilibration technique were used to analyse arterial carbon dioxide tension (Pa_{CO_2}). Calibration gases were analysed by the Scholander method; all arterial samples were analysed three times.

RESULTS

In Table I the physiologic information regarding the 13 patients is shown. Tables II, III, and IV display the individual Pa_{O_2}, Pa_{CO_2} and $\dot{V}_E$ data of each patient breathing air and 50 per cent O_2 in N_2 awake and, subsequently, anaesthetized. Samples were withdrawn each 15 minutes after IPPB was initiated. Pa_{CO_2} was determined each 30 minutes. Those 22 samples withdrawn at VT and/or f not identical to the awake control are indicated by an asterisk (*).

Figure 1 displays the mean ±1 S.D. of the Pa_{O_2}, Pa_{CO_2} and $\dot{V}_E$ (including VT, f, and estimated $\dot{V}_A$) for the 13 patients during the three conditions: (*a*) awake, spontaneously breathing air; (*b*) awake, spontaneously breathing 50 per cent O_2 in N_2 for five minutes after denitrogenation; and (*c*) anaesthetized with 50 per cent O_2 in N_2O-halothane during IPPB at VT and f almost identical to those during situation (*b*).

TABLE I

CLINICAL INFORMATION

Patient	Sex	Age	Ht. (in.)	Wt. (lbs.)	Surgery	Thiopental (mg.)	d-Tubocurarine (mg.)
1. J.G.	M	43	71	150	resection hand tumour	200	Succinylcholine drip
2. M.L.	F	76	62	100	cholecystectomy	—	24
3. E.T.	M	45	68	150	gastrectomy	250	57
4. B.A.	F	42	63	125	expl. lap.	200	24
5. A.S.	F	63	62	140	lysis of abd. adhesions	150	21
6. J.H.	M	66	68	130	Appendectomy & colostomy	150	24
7. L.D.	M	47	66	187	ing. herniorrhaphy	225	27
8. G.W.	F	48	62	110	hysterectomy	275	21
9. A.K.	M	78	69	160	perineal prostatectomy	200	21
10. A.G.	M	55	70	188	radical neck dissection	100	48
11. E.W.	F	65	66	176	sympathectomy	225	42
12. E.B.	F	51	64	125	cholecystectomy	175	38
13. M.S.	F	37	64	140	vein ligation and stripping	175	15

TABLE II

Pa_{O_2} (mm. Hg)

	Patient no.														
	1	2	3	4	5	6	7	8	9	10	11	12	13	Mean	S.D.
Awake															
air	94	75	—	87	—	—	83	78	79	78	71	81	93	82	7
50% O_2 in N_2	277	210	—	271	—	—	198	179	221	197	135	188	202	208	40
Minutes after initiation of IPPB															
15	235	151	—	261	217	—	104	—	200 188	207	121	201	210	190	45
30	224	—	286	250	176	168 150	95	—	191	175	118	199	196	186	50
45	238	93	—	240	167	146	82	89	193	165	122	191	212 207	165	53
60		—	254	—	113	147	85	82	187	166	118	183	206	154	53
75		83	—	234		145	70		183	147	117	185	203	152	52
90		—	238	245		143	68		159	146	127	—	214	168	57
105		98	263	231		—	88		—	141	136	183 140	194	164	55
120		108	—	238		161 170	95*		174 184	143	134	186		168	34
135		109	251*	254			97*		167	146	140	177*		163	49
150		127	247*				102*		171	164*	161	183*		153	19
165		136	249*						186	166*	166			163	21
180		151	253*							169*					
195		141	263*							162*					
210		130	263*							185*					
225			—							179*					
240			276*							178*					
255			273*							174*					
270			265*							152					
285										158					
300										156					
315										163					
mean	232	121	260	244	168	154	85	86	182	160	133	184	205		

*Sampled at VT and/or f other than awake, control. These data not included in calculating means.

TABLE III

Pa_{CO_2} (mm. Hg)

	Patient no.														
	1	2	3	4	5	6	7	8	9	10	11	12	13	Mean	S.D.
Awake															
air	42	—	—	39	—	—	—	—	—	—	—	38	38	39	2
50% O_2 in N_2	41	43	—	—	—	—	42	40	38	46	52	36	39	42	4
Minutes after initiation of IPPB															
	38												40		
30	42	44	33		31	32			28	46	53	41	38	39	7
													38		
60	46							42	30		51	40	35	40	6
													41		
													40		
90		54	43	21		34	30			54	50	38	38	40	10
120				21					36		50	42	47	39	10
										56*		44*			
150		46					30*			56*	48	42*		50	4
180			26*								50				
										69*					
210		48	24*							50*					
240															
			24*												
270			20*							46					
										58					
300															
330										60					
mean	42	48	38	21	31	33	30	42	32	55	50	40	40		

*Sampled at VT and/or f other than awake, control. These data not included in calculating means.

TABLE IV

VE (ml./min.)

	Patient number														
	1	2	3	4	5	6	7	8	9	10	11	12	13	Mean	S.D.
Awake															
air	6000	—	—	6138	—	—	9300	7923	6474	10603	4970	6600	9800	7534	1847
50% O_2 in N_2	7600	6900	6300	8344	9450	8800	8556	7581	7620	8358	5400	5700	6600	7178	1032
Minutes after initiation of IPPB															
15	8100	6300	—	9100	8250	—	8349	—	10400 7800	8260	4576	5650	6900	7608	1565
30	7600	—	6300	8400	9000	8800 8800	9100	—	9240	8260	5595	5650	7200	7829	1288
45	7600	5600	—	10150	10500	8800	8760	7448	7800	8900	5580	5900	7200 7900	7857	1514
60		—	5950	—	10725	10592	8880	7942	8004	8060	6450	5700	6700	7900	1686
75		6650	—	7800		10560	8280		7848	7940	6000	6500	6700	7586	1288
90		—	6300	9375		9840	8448		8148	7900	5400	—	7200	7826	1395
105		7000	6300	8400		—	8400		—	7800	5100	5850 6000	7200	6894	1102
120		7000	—	8400		9024 9000	10470*		7848 7848	7800	5100	6100		7569	1231
135		7000	9360*	8400			10800*		7452	7088	5850	10000*		7300	857
150		7000	9180*				10800*		7620	7020*	5475	8000*		6698	901
165		7000	9900*						7776	6960*	5250			6675	1065
180		7000	9248*							7068*					
195		8400	9972*							6852*					
210		7000	9900*							12204*					
225										12180*					
240			9396*							11400*					
255			9900*							11400*					
270			15000*							8000					
285										8240					
300										8240					
315										8240					
mean	7767	6905	6212	8753	9619	9427	8602	7695	8149	8111	5489	5919	7125		

*Sampled at VT and/or f other than awake, control. These data not included in calculating means.

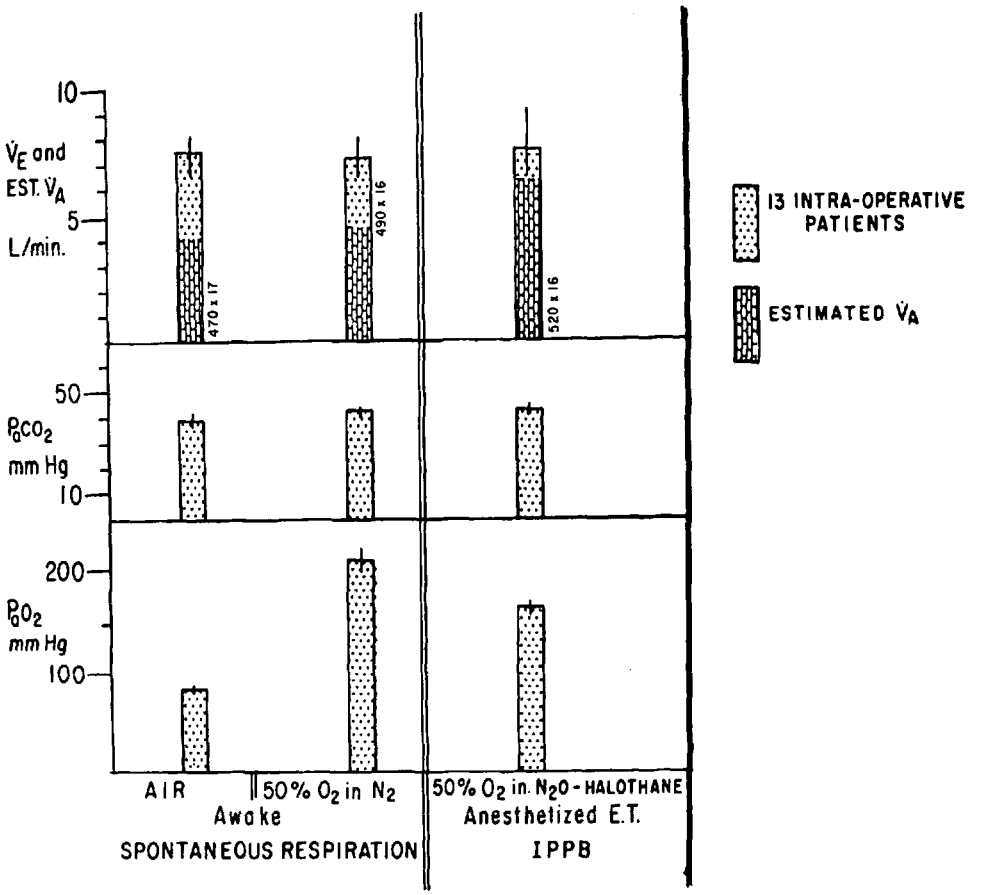

FIGURE 1. Volumes and gas tensions during spontaneous and intermittent positive pressure breathing.

$\bar{P}a_{O_2}$ with air was 82 mm. Hg, 208 mm. during 50 per cent O_2 and 167 mm. during IPPB and anaesthesia (mean value from 99 blood samples). This represents a drop of 41 mm. Hg ($p < .05$). $\bar{P}a_{CO_2}$ during conditions (*a*), (*b*), and (*c*) was 39, 42, and 42 mm. Hg respectively. $\bar{V}_E$ was 7.53, 7.18 and 7.59 L./min. during conditions (*a*), (*b*), and (*c*). There was, therefore, a significant drop in $\bar{P}a_{O_2}$ despite almost identical $\bar{V}_E$, $\bar{V}_T$, $\bar{f}$, $\bar{P}_{IO_2}$ and $\bar{P}a_{CO_2}$.

Figure 2 plots the average Pa_{O_2} and $\dot{V}_E$ at 15-minute intervals and Pa_{CO_2} at 30-minute intervals in the 13 patients (see Table II). The data were plotted up to 165 minutes of IPPB. Not all patients had measurements during each 15-minute interval. While $\dot{V}_E$ and Pa_{CO_2} remained at levels close to the awake control, the Pa_{O_2} gradually fell, reaching its lowest levels at 60 and 75 minutes.

DISCUSSION

It is accepted that hypoxaemia may occur during anaesthesia and IPPB and that: (*a*) V_D (phys) increases and (*b*) a physiologic shunt occurs.[1-11] Further, pulmonary mechanics during IPPB are different from those during spontaneous

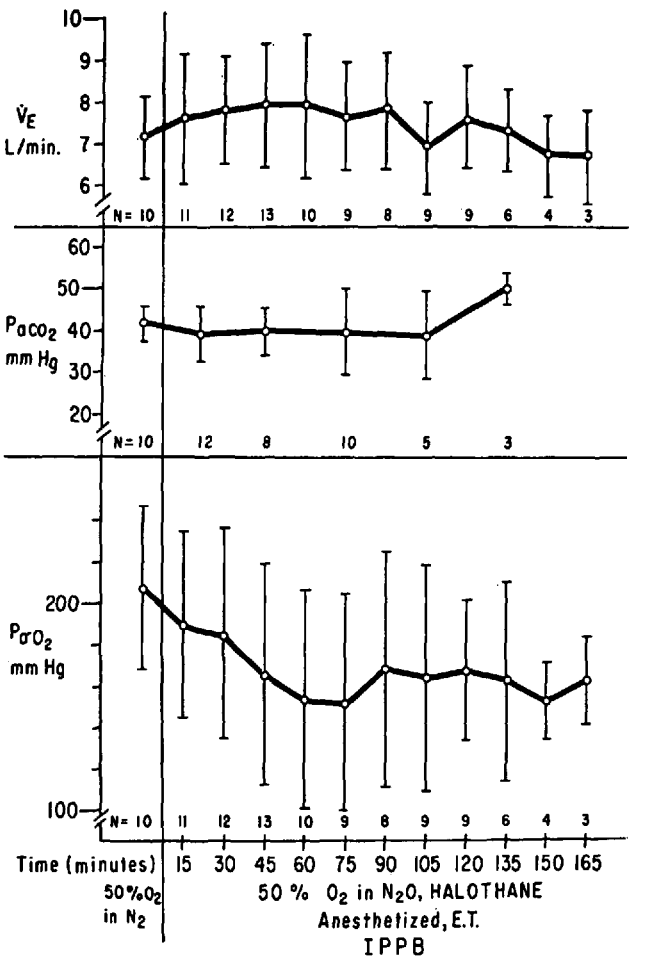

FIGURE 2. Variation in blood gases with time during intermittent positive pressure breathing.

breathing.[12] A decrease in pulmonary compliance (CL) in both awake and anaesthetized states occurs.[5,8,13,14] The magnitude and incidence of these abnormalities and therefore their significances are not known. It is well to realize that during anaesthesia and spontaneous breathing, an increase in venous admixture and a decrease in CL also occur.[14,15]

In this study, the reduction of $\bar{P}a_{O_2}$ from 208 to 167 mm. Hg ($p < .05$) in the passage from the awake to the anaesthetized state during IPPB, despite similar $P_{I_{O_2}}$, VT, and f, indicates the existence of some abnormality. This cannot be explained on the basis of decreased effective $\dot{V}_A$ since $\bar{P}a_{CO_2}$ did not change from the awake to the anaesthetized state during IPPB.

In addition to the lowered Pa_{O_2} under these conditions, there was a wide variation in individual Pa_{O_2} and Pa_{CO_2} response. Of the 13 normals, three patients (#2, 7, and 8) had Pa_{O_2} values below 100 mm. Hg at some time during anaesthesia, and of these only patient #2 had concomitant hypercarbia. None of the low Pa_{O_2} values was significantly lower than the awake control Pa_{O_2} on room air.

Since N_2 is less soluble than N_2O and might effectively prevent absorption atelectasis, it might be believed that the difference in background gases (N_2 *vs.* N_2O) from the awake control to the anaesthetized state could have contributed to the lower Pa_{O_2} during anaesthesia and IPPB. In a preliminary report, Webb and Nunn[16] studied venous admixture in two groups of patients, one inhaling N_2 and the other N_2O; both groups breathed both spontaneously and mechanically but did not develop hypercarbia. A greater per cent admixture with N_2 than N_2O was found, and we must therefore presume that N_2O did not contribute to a lower Pa_{O_2} during IPPB in this study.

Despite the variety of Pa_{O_2} responses, the remaining ten patients who did not develop any degree of hypoxaemia maintained Pa_{O_2} values between 150 and 250 mm. Hg. These patients were ventilated at a VT and f almost identical to their awake control values with 50 per cent O_2 inhalation. Patients #1, 7, 9, 11, and 13 had, on occasion, a "low" VT or f. Patient 5 was an exception since his Pa_{O_2} began to fall but never reached hypoxic levels. The surgery lasted one hour and further data are lacking.

The Pa_{CO_2} information yielded a variety of patient responses. In general, values were equal to or lower than their respective awake controls. Patient #10 had an elevated Pa_{CO_2} in the presence of a Pa_{O_2} at the 150 mm. Hg level. When the VT was increased to 1000 ml. (2½ times control), and the f dropped from 20 to 12, the Pa_{CO_2} also dropped. The Pa_{O_2} changed little in either case. Patient #11 also had slightly elevated Pa_{CO_2}, but his control value was 52 mm. Hg.

The magnitude of the alveolar-arterial carbon dioxide gradient ($A\text{-}aDco_2$) is considerably smaller than the alveolar-arterial oxygen gradient ($A\text{-}aDo_2$)[17] partly because of the more linear dissociation curve at the normal physiological level and partly because of the smaller difference between the mixed venous and arterial carbon dioxide tensions. With respect to large shunts, one might therefore predict a smaller change in the $A\text{-}aDco_2$ than $A\text{-}aDo_2$. For example, with a normal venous to arterial Pco_2 difference of 5 mm. Hg, a 10 per cent shunt will yield only a 0.5 mm. Hg $A\text{-}aDco_2$ but an $A\text{-}aDo_2$ of approximately 150 mm. Hg when 50 per cent O_2 is inhaled.[18] The approximately 40 mm. Hg drop in Pa_{O_2} from 50 per cent O_2 in N_2 to 50 per cent O_2 in N_2O-halothane is equivalent to an increase in shunt of about 3 per cent (Fig. 1).

Figure 2 is a chronologic plot of the mean data at 15-minute intervals. At 60 and 75 minutes after the initiation of IPPB, during anaesthesia at a VT and f determined by the patient when awake, the lowest Pa_{O_2} values occurred. After 75 minutes no further decrease developed and an upward trend was noted. Others have noted a similar progressive decline in Pa_{O_2} during IPPB.[5,6] We may conjecture that homeostatic mechanisms, perhaps reflex in nature during such constant volume ventilation, become manifest at approximately one hour and cause a reversal of the probable physiologic shunt.[19]

Summary and Conclusions

Blood gases, tidal volumes, and respiratory rates were measured preoperatively and during surgery with intermittent positive pressure breathing of 50 per cent

oxygen. The ventilator was set to provide a tidal volume and rate closely approximating that in the awake state.

$\overline{P}a_{O_2}$ dropped significantly during IPPB despite the similar $P_{I_{O_2}}$, V_T, f, and $\dot{V}_E$. $\overline{P}a_{CO_2}$ remained the same. As time elapsed, a progressive fall in $\overline{P}a_{O_2}$ occurred. The $\overline{P}a_{O_2}$ reached its minimum at approximately one hour after the initiation of IPPB. Individual patient responses varied considerably for both changes in Pa_{O_2} and Pa_{CO_2}. Although three patients developed Pa_{O_2} values below 100 mm. Hg, none was considered hypoxaemic since the respective Pa_{O_2} was at a level above the air control. Some Pa_{O_2} values dropped approximately 150 mm. Hg below the awake control while others remained at the 250 mm. Hg level. It should be realized that the tidal volumes used were lower than that during the usual clinical administration of anaesthesia. Changes in Pa_{CO_2} were not usually related to changes in Pa_{O_2}, and hypocarbia and acceptable estimated $\dot{V}_A$ were frequently associated with a Pa_{O_2} lower than during the control. Therefore, under the same conditions, it is concluded that if the tidal volume and respiratory rate used during IPPB are identical to those of the awake control, then during the inhalation of 50 per cent O_2 patients are able to maintain normal Pa_{CO_2} and slightly lower Pa_{O_2}.

Résumé

Chez 13 malades, nous avons mesuré à toutes les 15 minutes avant et durant l'anesthésie et la chirurgie, alors que le malade respirait sous pression positive intermittente, nous avons mesuré les gaz du sang, les volumes courants et la vitesse de la respiration. Le ventilateur fournissait spécifiquement un air courant et une vitesse respiratoire indentiques à ceux du malade réveillé. Le taux d'oxygène inhalé était de 50 pour cent.

La Pa_{O_2} a baissé de façon marquée durant la pression positive intermittente; la Pa_{CO_2} est demeurée la même. Le changement de la Pa_{O_2} a été progressif et a atteint son sommet au bout d'une heure. Bien que, chez certains malades, la Pa_{O_2} ait été au-dessous de 100 mm. Hg, nous n'avons pas estimé qu'il existait d'hypoxémie puisque la Pa_{O_2} est demeurée au-dessus du contrôle lorsque le malade respirait de l'air. Nous avons observé des variations individuelles considérables de la Pa_{O_2} et de la Pa_{CO_2}. Les changements de la Pa_{CO_2} n'étaient pas habituellement à l'opposé de ceux de la Pa_{O_2} et l'hypocarbie en présence d'un volume/minute adéquat s'accompagnait généralement d'une Pa_{O_2} plus basse que durant le contrôle.

Acknowledgments

The authors acknowledge the enthusiastic help of Mr. F. E. Knowles III, Miss Barbara Eerligh, and Mrs. Lillian Chambliss.

Abbreviations

V_T	tidal volume
$\dot{V}_E$	minute volume

f	respiratory rate/minute
$\dot{V}_A$	alveolar ventilation*
Pa_{O_2}	arterial oxygen tension
Pa_{CO_2}	arterial carbon dioxide tension
A-aD_{O_2}	alveolar-arterial oxygen gradient
A-aD_{CO_2}	alveolar-arterial carbon dioxide gradient
V_D (phys)	physiologic dead space
$P_{I_{O_2}}$	inhaled oxygen tension
$\dot{Q}s/\dot{Q}t \times 100$	= Per cent physiologic shunt†

*$\dot{V}_A$ was estimated by: (*a*) multiplying the f by the combined anatomical (1 lb. body weight equals 1 ml.) and mechanical (mouthpiece, E. T. tube, connectors, Wright respirometer) dead spaces, and (*b*) subtracting this figure from $\dot{V}_E$.

†This was estimated by using the simplified version of the alveolar gas equation for 50 per cent O_2:

$$P_{A_{O_2}} = \tfrac{1}{2}(P_B - 47) - Pa_{CO_2}.$$

Here $P_{A_{O_2}}$ is alveolar oxygen tension, P_B is barometric pressure, and 47 is water vapour pressure.

The per cent shunt was estimated from

$$\dot{Q}s/\dot{Q}t = (Cc_{O_2} - Ca_{O_2}) \div (Cc_{O_2} - C\bar{v}_{O_2}).$$

Here $Cc_{O_2} = O_2$ content of blood leaving alveolar capillaries; $Ca_{O_2} = O_2$ content of arterial blood; $C\bar{v}_{O_2} = O_2$ content of mixed venous blood. Therefore,

$$\dot{Q}s/\dot{Q}t = (P_{A_{O_2}} - Pa_{O_2})\ 0.0031 \div (Cc_{O_2} - C\bar{v}_{O_2}).$$

It follows then, if we assume $Cc_{O_2} - C\bar{v}_{O_2}$ equals 4.5 volumes per cent, that with a $P_{A_{O_2}}$ of 316 mm. Hg, and Pa_{O_2} of 200 mm. Hg,

$$\dot{Q}s/\dot{Q}t = (316 - 200)\ 0.0031/4.5 = 7.5\% \text{ shunt.}$$

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