The Effects of Major Surgery on Cardiac Output and Shunting

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Cardiac output, resting lung volume (FRC) and physiologic shunting were measured in 12 patients immediately before and after upper abdominal surgery. FRC and physiologic shunt did not change. Cardiac output, however, was reduced significantly, and considerable individual variation was observed. In addition, shunt estimated with an assumed $Ca_{O_2} - C\bar{v}_{O_2}$ of 4.5 vol per cent frequently differed greatly from the shunt actually determined. Results of the study indicate that changes in cardiac output can lead to inaccuracy in the calculation and interpretation of intrapulmonary shunting of blood. The greater the shunting, the more significant are changes in cardiac output in the determination of shunt. In the seriously-ill patient in whom changes in cardiac output are expected, management of cardiopulmonary problems is more effectively accomplished when both $A - aD_{O_2}$ and $Ca_{O_2} - C\bar{v}_{O_2}$ are determined.

CLINICAL ASSESSMENT of intrapulmonary shunting of blood is frequently made by measuring the alveolar–arterial oxygen gradient ($A - aDo_2$). This method of estimating shunt, however, requires the use of an assumed arterial–mixed venous oxygen content difference ($Cao_2 - C\bar{v}o_2$). The validity of shunt estimations without measurement of mixed venous blood may be questioned, particularly in situations where changes in cardiac output can be expected. Accordingly, a series of 12 patients was studied to determine the effects of upper abdominal surgery on resting lung volume (FRC), cardiac output, and $Cao_2 - C\bar{v}o_2$, and the effects of changes in these parameters on the estimation of intrapulmonary shunting.

Methods

Patients selected for study were about to undergo major upper abdominal surgery. Many were seriously ill. Operations performed included bilateral adrenalectomy, gastrectomy, aortic aneurysmectomy, total colectomy, and repair of a massive ventral hernia with bowel resection in a 422-pound patient with Pickwickian syndrome (Patient 12). Patients, 2, 3, 7, and 12 had significant chronic pulmonary disease. The patients selected, therefore, cannot be considered representative of a normal surgical population. The mean age of the patients was 55 (37–68) years. One hour after premedication with 100 mg pentobarbital (Nembutal) and 0.6 mg atropine, intramuscularly, the patient was brought to the operating room and the preoperative studies were done with the patient supine either on the operating table or on a stretcher.

A 36-inch central venous catheter was passed through the basilic vein into the right ventricle for collection of mixed venous blood samples. Venous pressure was monitored with a water manometer during advancement of the catheter; proper placement of the catheter tip was determined by fluctuations and rise in the pressure consistent with mean right ventricular pressure. Either the brachial or the radial artery was cannulated with a one-inch 22-gauge needle for collection of arterial blood samples. A #8 soft French catheter, for collecting either inspiratory or end-expiratory gas samples, was passed through the nose until the tip was just visible in the posterior pharynx, then taped in place. During breathing of air,

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expired air was collected for two to four minutes in a meterological balloon, utilizing a mouthpiece, nose clip, Sierra nonrebreathing valve and Collins two-way valve. Expired-air analyses and volume were used in computing the respiratory exchange ratio (R) and oxygen consumption ($\dot{V}o_2$). Arterial and mixed venous blood samples were drawn coincident with collection of expired air and analyzed immediately for *p*H, Pco_2, and Po_2.

Denitrogenation was then carried out for ten minutes, utilizing a mouthpiece, nose clip and Sierra nonrebreathing valve, the latter connecting a five-liter anesthesia bag to a face mask. A 50-ml end-expired air sample was collected from the posterior pharynx and analyzed for O_2 and CO_2.

$P_{A_{O_2}}$ of the end-expired gas following ten minutes of denitrogenation was first measured with a Beckman C-2 Analyzer, and CO_2 content with an Anesthesia Associates CO_2 Analyzer. CO_2 analysis assured the validity of the end-expired sample; the remainder of the sample was then analyzed for O_2 and CO_2 in the laboratory with a Beckman 160 Analyzer. Arterial and mixed venous blood samples were drawn at the time of collection of end-expired air.

The functional residual capacity (FRC) of the lungs was measured by the closed-circle helium-dilution technique.[1]

The surgical operation was then performed. Anesthetics and techniques used were at the discretion of the anesthesiologist and included nitrous oxide, cyclopropane, halothane or lmethoxyflurane, with or without muscle re axants. Controlled respiration was employed in all cases. Following operation, preoperative studies were repeated, but not until the patient had recovered from the anesthetic sufficiently to cooperate and respond appropriately. This was from one to four hours following operation in the recovery room. Narcotic medication was omitted in the postoperative period while blood gas studies were done. Occasionally postoperative body temperatures were lower than preoperative values, but appropriate corrections of blood tensions were always made. Shivering, which might have affected oxygen consumption, was not encountered.

Calculations

Oxygen content of blood (Cb_{O_2}) was calculated from the equation:

$$Cb_{O_2} = (Hgb \times 1.34)So_2 + (Pb_{O_2} \times 0.0031) \quad (1)$$

Whole-blood saturation (So_2) was calculated from the oxygen tension of blood, using the Severinghaus calculator, which corrects for *p*H, Pco_2 and temperature.[2] Pulmonary capillary blood oxygen content (Cc_{O_2}) was calculated by assuming full equilibration of pulmonary capillary blood at the observed $P_{A_{O_2}}$. The oxygen content of each of 34 samples of arterial and mixed venous blood from seven patients was also determined in duplicate polarographically, using a mixture of deoxygenated potassium ferricyanide and blood.[3]

$P_{A_{O_2}}$ during inhalation of air was calculated from the standard alveolar air equation employing corrections for the respiratory exchange ratio (R) and assuming that $P_{A_{CO_2}}$ was equal to Pa_{CO_2}.

$P_{A_{O_2}}$ during inhalation of oxygen was assumed to equal the end-expired oxygen tension following at least ten minutes of denitrogenation. It was not calculated by the usual approach using the formula $P_{A_{O_2}} = P_B - (P_{A_{CO_2}} + P_{H_2O})$ for the following reasons: In dealing with the critically-ill, and sometimes restless patient we have found it not always possible to eliminate occasional entrainment of air during denitrogenation, and in spite of what appears to be a suitable length of time, the expired air may contain some nitrogen. Also, many of these patients with ventilation problems would require more than 30 minutes of oxygen inhalation for complete washout. In such a circumstance we believe that direct measurement of a reasonably stable end-expired O_2 (and CO_2) provides a more accurate assessment of $P_{A_{O_2}}$ since it actually can detect air entrainment and avoids a period of prolonged denitrogenation. Last, the assumption that $Pa_{CO_2} = P_{A_{CO_2}}$ may not be valid in the presence of shunts of more than 20 per cent.

Cardiac output during breathing of air was determined according to the Fick principle:

$$\text{C.O. (l/min)} = \frac{\dot{V}o_2}{Cao_2 - Cv_{O_2}} \quad (2)$$

TABLE 1. Effects of Surgery on Shunting in 12 Patients Breathing Room Air and 100 Per Cent Oxygen

	P_{O_2}		P_{CO_2}		pH		$C_{\bar{v}O_2} - C_{aO_2}$ (vol percent)		$C_{aO_2} - C_{\bar{v}O_2}$ (vol percent)		$\dot{Q}_s/\dot{Q}_t \times 100$	
	Pre-op	Post-op	Pre-op	Post-op	Pre-op	Post-op	Pre-op	Post-op	Pre-op	Post-op	Pre-op	Post-op
Room Air												
Pt. 1	70	82	40	44	7.46	7.38	0.49	0.36	4.7	3.7	10	9
Pt. 2	63	62	41	40	7.40	7.40	0.81	1.23	2.1	3.3	30	27
Pt. 3	55	50	30	29	7.54	7.51	1.03	1.95	2.3	9.0	31	18
Pt. 4	77	80	35	36	7.54	7.47	0.75	1.58	5.0	7.0	13	17
Pt. 5	66	69	38	42	7.48	7.36	0.98	0.89	2.5	2.3	28	28
Pt. 6	77	72	32	38	7.46	7.38	0.52	0.23	3.3	6.3	13	13
Pt. 7	62	66	35	40	7.44	7.36	1.01	0.90	6.0	5.3	15	17
Pt. 8	69	73	37	45	7.48	7.42	0.64	0.48	3.7	3.7	15	13
Pt. 9	84	68	36	47	7.50	7.41	0.62	0.40	4.7	4.4	12	14
Pt. 10	62	62	28	34	7.44	7.42	1.22	1.43	3.7	5.0	24	22
Pt. 11	67	64	36	42	7.40	7.37	1.19	0.90	4.0	4.9	23	15
Pt. 12	56	52	37	36	7.28	7.35	2.07	2.53	3.8	5.2	44	33
$\bar{X}\pm SE$	68± 2.6	66± 2.7	35±1.0	39±1.4	7.46±0.01	7.40±0.01	1.10±0.18	1.07±0.19	3.8±0.32	5.1±0.51	21.5±2.6	18.8±2.0
100 Per Cent Oxygen												
Pt. 1	435	430	39	47	7.48	7.39	0.43	0.49	3.8	4.8	8.4	9.4
Pt. 2	480	380	39	44	7.48	7.38	0.31	0.74	1.9	4.9	14.7	13.1
Pt. 3	365	240	27	32	7.60	7.49	0.45	0.40	4.4	6.9	9.3	5.5
Pt. 4	500	375	39	36	7.52	7.44	0.45	0.40	4.0	7.0	10.0	5.3
Pt. 5	370	455	30	47	7.58	7.34	0.48	0.57	3.9	2.8	11.0	10.9
Pt. 6	440	375	34	42	7.45	7.41	0.23	0.74	4.6	5.9	4.8	11.0
Pt. 7	305	400	36	44	7.44	7.30	0.43	0.59	4.8	5.4	8.2	7.5
Pt. 8	515	570	34	34	7.45	7.56	0.48	0.27	4.7	3.9	9.4	6.5
Pt. 9	460	520	40	46	7.48	7.44	0.40	0.32	3.7	4.0	9.7	7.5
Pt. 10	430	365	22	33	7.55	7.39	0.77	1.02	4.1	5.2	16.3	17.0
Pt. 11	355	370	42	34	7.42	7.49	0.64	0.57	3.9	4.9	14.1	10.0
Pt. 12	425	455	39	43	7.28	7.37	1.36	1.16	4.6	5.7	23.0	16.9
$\bar{X}\pm SE$	430±23.7	412±18.0	35±3.6	40±1.6	7.48±0.02	7.42±0.02	0.53±0.15	0.54±0.06	4.0±0.21	5.1±0.32	11.6±1.1	10.6±1.2

TABLE 2. Effects of Surgery on FRC, $\dot{V}o_2$, and Cardiac Output

	FRC (ml)		$\dot{V}o_2$ (ml)		C.O. (l/min)	
	Pre-op	Post-op	Pre-op	Post-op	Pre-op	Post-op
Pt. 1	1,930	2,490	249	244	5.3	6.6
Pt. 2	1,350	1,540	200	198	9.5	6.0
Pt. 3	2,210	2,070	120	207	5.2	2.3
Pt. 4	2,539	2,290	285	213	5.7	2.8
Pt. 5	1,990	1,720	195	184	7.8	8.0
Pt. 6	1,920	2,190	171	145	5.2	2.3
Pt. 7	1,370	1,560	186	164	3.1	3.1
Pt. 8	3,250	3,230	229	204	6.2	5.5
Pt. 9	2,260	2,310	193	202	4.1	4.6
Pt. 10	2,080	2,348	199	200	5.4	4.0
Pt. 11	2,313	2,117	200	196	5.0	4.0
Pt. 12	1,550	1,250	357	432	9.4	8.3
$\bar{x}\pm SE$	2,064±145	2,093±172	215±16	216±20	6.0±.54	4.8±.58

When air was inhaled, the total physiologic shunt was estimated from the following equation:

$$\dot{Q}_s/\dot{Q}_t = \frac{Cc_{O_2} - Ca_{O_2}}{Cc_{O_2} - Cv_{O_2}} \quad (3)$$

which relates the shunted portion ($\dot{Q}_s$) of the cardiac output to the total output ($\dot{Q}_t$). The difference between the actual oxygen content of artieral blood (Ca_{O_2}) and that which was theorectically possible if all blood entering the lungs were to reach full equilibration with the alveolar oxygen (Cc_{O_2}) represented the alveolar–arterial oxygen content gradient ($Cc_{O_2} - Ca_{O_2}$). During inhalation of air this gradient results from blood coming through intrapulmonary anatomical shunts, through atelectatic lung as well as from areas of low ventilation relative to perfusion.

During inhalation of oxygen, the true shunt was measured; it represented blood shunted past atelectatic lung and through anatomic shunts. The equation for determining the true shunt was:

$$\dot{Q}_s/\dot{Q}_t = \frac{(P_{A_{O_2}} - Pa_{O_2})\,0.0031}{(P_{A_{O_2}} - Pa_{O_2})\,0.0031 + (Ca_{O_2} - Cv_{O_2})} \quad (4)$$

Results

Effect of Operation on FRC, Cardiac Output and Shunting

FRC, which is known to be linearly and inversely related to the total physiologic shunt,[4] remained remarkably stable following operation (table 2). Prior to operation the mean total physiologic shunt was 22 per cent of the cardiac output; the true shunt, during breathing of oxygen, was 12 per cent. No significant changes in shunting occurred following operation whether air or oxygen was inspired. Thus, approximately half the total physiologic shunting in this group of patients was due to a ventilation–perfusion abnormality unchanged by surgery and anesthesia. Cardiac output, however, was reduced following operation. Prior to operation the mean cardiac output was 6 l/min. It fell to 4.8 l/min after operation. The change was significant ($P < 0.025$).

Relationship of Alveolar–Arterial Oxygen Content Gradient and Cardiac Output to Shunting

A positive linear correlation was demonstrated between the alveolar–arterial oxygen content difference and $\dot{Q}_s/\dot{Q}_t$ during breathing of oxygen. ($r = 0.86$) The regression equation best expressing the relationship (data from table 1) was $y = 13.9x + 3.4$ where y = per cent $\dot{Q}_s/\dot{Q}_t$ and $x = Cc_{O_2} - Ca_{O_2}$ (vol per cent). During breathing of air the correlation of the alveolar–arterial oxygen content gradient with the total physiologic shunt was much less apparent ($r = 0.49$), although the regression equation ($y = 13.6x + 3.8$) was similar to that during breathing of oxygen.

No significant correlation was noted between cardiac output and $\dot{Q}_s/\dot{Q}_t$ during breathing of oxygen or air ($r = 0.28$, $P < 0.2$).

Significance of Arterial–Mixed Venous Oxygen Content Difference in Calculating Shunt

The mean preoperative $\dot{V}o_2$ of 215 ml/min was unchanged following surgery (table 2). Since changes in $\dot{V}o_2$ at rest are known to be small, alteration of cardiac output was determined principally by changes in the arterial–mixed venous oxygen content difference ($Ca_{O_2} - Cv_{O_2}$). An increase in the mean $Ca_{O_2} - Cv_{O_2}$ occurred following operation when both air and oxygen were inhaled, and during oxygen inhalation the change was significant ($P < .05$, table 1). Since the determination of shunt is directly influenced by changes in the $Ca_{O_2} - Cv_{O_2}$, the effects of an assumed $Ca_{O_2} - Cv_{O_2}$ of 4.5 vol per cent and the actual $Ca_{O_2} - Cv_{O_2}$ in computing the physiologic shunt were determined (table 3). The choice of 4.5 vol per cent was arbitrary. This represented, however, the mean of all the pre- and postoperative values obtained while our patients were breathing air and oxygen. The value also is that obtained by Cournand *et al.*

Table 3. Physiologic Shunt Determined by Actual *vs.* Assumed $Ca_{O_2} - Cv_{O_2}$ of 4.5 Vol. Per Cent

	Preoperative				Postoperative			
	Actual	Assumed	Per-cent Error	P	Actual	Assumed	Per-cent Error	P
Room air $\dot{Q}_s/\dot{Q}_t \times 100$								
Pt. 1	10	9.8	−2		9	7.4	−17	
Pt. 2	30	15.2	−49		27	21.4	−20	
Pt. 3	31	18.6	−40		18	30.2	+68	
Pt. 4	13	14.2	+9		17	26.0	+52	
Pt. 5	28	17.9	−36		28	16.5	−41	
Pt. 6	13	10.4	−20		13	4.8	−63	
Pt. 7	15	18.3	+27		17	16.6	−2	
Pt. 8	15	12.4	−17		13	9.6	−26	
Pt. 9	12	12.1	0		14	8.1	−42	
Pt. 10	24	21.3	−11		22	24.1	+9	
Pt. 11	23	20.9	−9		15	16.6	+7	
Pt. 12	44	37.2	−15		33	36.0	+9	
$\bar{x}\pm SE$	21.5±2.6	17.3±2.0	−13.6 ±4.8	<0.005	18.8±2.0	18.1±2.6	−5.5 ±6.3	<0.80
100 Per cent oxygen $\dot{Q}_s/\dot{Q}_t \times 100$								
Pt. 1	8.4	8.7	+3		9.4	9.8	+4	
Pt. 2	14.7	6.4	−56		13.1	14.1	+7	
Pt. 3	9.3	6.4	−3		5.5	8.1	+47	
Pt. 4	10.0	9.0	−10		5.3	8.1	+52	
Pt. 5	11.0	9.6	+12		16.9	11.2	−33	
Pt. 6	4.8	4.8	0		11.0	14.1	+28	
Pt. 7	8.2	8.0	−2		7.5	11.5	+53	
Pt. 8	9.4	9.6	+12		6.5	5.6	−15	
Pt. 9	9.7	8.1	−16		7.5	6.6	−12	
Pt. 10	16.3	14.6	−10		17.0	15.6	−8	
Pt. 11	14.1	12.4	−12		10.0	11.2	+10	
Pt. 12	23.0	23.2	+1		16.9	20.4	+20	
$\bar{x}\pm SE$	11.6±1.1	10.2±4.2	−8.5 ±4.2	<0.10	10.6±1.2	11.3±1.1	+12.7 ±5.0	<0.70

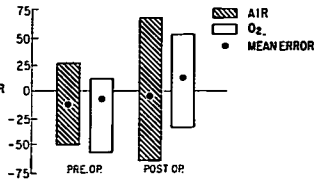

FIG. 1. Range and mean error from actual shunt when estimating shunt using an assumed $Ca_{O_2} - Cv_{O_2}$ of 4.5 vol per cent (N = 12).

under rigidly controlled conditions in resting man.[5]

Prior to operation, the mean total physiologic shunt during inhalation of air would have been significantly underestimated if an assumed $Ca_{O_2} - Cv_{O_2}$ of 4.5 vol per cent had been used in computing shunt. Table 1 contains evidence that the patients had mild respiratory alkalosis, probably due to apprehension associated with the study and contributing to increases in cardiac output. Mean values for total physiologic shunt after operation and true shunt both before and after operation, however, were not affected by assuming a $Ca_{O_2} - Cv_{O_2}$ of 4.5 vol per cent for the group of 12 patients.

In individual cases, however, the shunt computed by using an assumed value of 4.5 vol per cent frequently differed considerably from the shunt computed with the actually-determined value (table 3). In only four of the twelve patients studied were the actual and assumed values for shunt within 10 per cent of each other. These differences reflected the variability of cardiac output among patients and changes induced by surgical operation. Had these differences in cardiac output been ignored, estimation of intrapulmonary shunt in individual cases would have been off as much as 50 per cent (fig. 1).

Figure 2 depicts the relationships of the pulmonary ($Cc_{O_2} - Ca_{O_2}$) and the systemic ($Ca_{O_2} - Cv_{O_2}$) oxygen content gradients to the physiologic shunt. The arrows in Figure 2 span the pre- and postoperative values for these gradients for each of the 12 patients during breathing of oxygen. It is apparent from this figure that changes in the systemic gradient played a significant role in estimation of shunt, and reliance on changes in the pulmonary gradient alone would have led to erroneous estimations. The figure further illustrates that, with a fall in cardiac output and an increase in the systemic oxygen gradient, the intrapulmonary gradient would also increase, yet the true shunt would be unchanged. Thus, a reduction in cardiac output *per se* does not affect shunt size but can reduce further the saturation of the mixed venous blood flowing through the shunt. While this venous admixture will increase the intrapulmonary gradient by lowering the arterial oxygen tension, it will not affect the size of the shunt. Seven of ten patients who had increased systemic oxygen gradients following surgery actually had no change or reductions in the sizes of the shunts.

Discussion

When measured during inhalation of both air and oxygen, the total physiologic shunt may be divided into two major components: that due to overperfusion relative to ventilation and that due to the true right-to-left shunt, most commonly attributed to atelectasis. The grouped results of the present study indicate that anesthesia and major operation do not have an appreciable effect on the amount of true shunt (atelectasis) or that portion of the total physiologic shunt due to ventilation–perfusion abnormalities. Moreover, in individual cases, shunt values did not change appreciably following surgery, whether the shunt was high or low in the preoperative period. In addition, no reduction in resting lung volume (FRC) occurred following surgery. Evidence has also been reported for the lack of development of atelectasis during and following halothane anesthesia and spontaneous respiration for surgery on the periphery in healthy patients.[6,7]

However, major operation and anesthesia are known to be responsible for changes in cardiac output,[8–10] and in the present study a significant reduction in cardiac output was present in the immediate postoperative period. Had not the true $Ca_{O_2} - Cv_{O_2}$ been used in computing shunt, the total physiologic shunt prior to operation would have been underestimated. Highly significant variations in cardiac output and $Ca_{O_2} - Cv_{O_2}$ occurred among patients and between pre- and postoperative values for each patient, which all but precludes the use of a fixed $Ca_{O_2} - Cv_{O_2}$ in estimating shunt. Data are available which

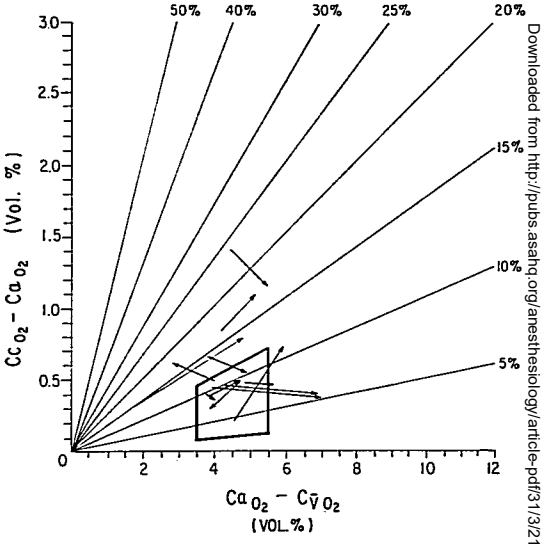

FIG. 2. Relationship of the pulmonary ($Cc_{O_2} - Ca_{O_2}$) and systemic ($Ca_{O_2} - C\bar{v}_{O_2}$) oxygen content gradients to physiologic shunt. Boxed area encompasses arbitrary limits of normal values. Arrows span the pre- and post-operative coordinates for each of 12 patients during breathing of oxygen.

indicate that even under strictly controlled resting conditions the average maximal variation in cardiac output is 17 per cent in individual subjects.[11] An attempt to estimate intrapulmonary shunt without incorporating the effects of these changes becomes increasingly speculative in situations promoting increases in cardiac output. Febrile, apprehensive or anemic patients are likely to have increased cardiac output. Since the relationship between cardiac output and $Ca_{O_2} - C\bar{v}_{O_2}$ is not linear but hyperbolic, reductions in $Ca_{O_2} - C\bar{v}_{O_2}$ in the presence of a large cardiac output will have a proportionally greater effect on estimation of shunt than when the cardiac output is low. Shunt estimations, then, are less accurate in the presence of high output, the accuracy dependent both on the standard error of measurement of blood oxygen content and on the validity of the mixed venous blood sample as a truely representative sample.

SOURCES OF ERROR IN DETERMINING $Ca_{O_2} - C\bar{v}_{O_2}$

Blood oxygen content was estimated from *p*H, Po_2, temperature, and hemoglobin concentration of blood, using the Severinghaus calculator, which utilizes a new composite oxygen dissociation curve.[2] Since many of our patients had mild anemia and many were smokers, blood oxygen content was also measured directly in 68 samples of blood from seven of 12 patients to compare with the contents estimated using the calculator. The mean $C\bar{v}_{O_2}$ values of 34 samples were 13.9 vol per cent determined directly and 14.3 vol per cent using the calculator. The mean Ca_{O_2} values of 34 samples were 18.5 and 19.2 vol per cent, respectively. Thus, the mean $Ca_{O_2} - C\bar{v}_{O_2}$ determined by calculation would be 0.3 vol per cent greater than that determined by direct measurement of blood oxygen content. The effect of this difference on the estimation of shunt in the presence of a normal cardiac output would be minimal. Use of the calculator, however, probably is not justified when greater accuracy is required for estimating saturation in markedly anemic patients[12] or heavy smokers,[13] or in other clinical circumstances in which altered dissociation curves can be expected.

The error in determination of content from saturation is also dependent on the accuracy of the polarographic technique, which can be estimated conservatively at ±2 mm Hg. In measurement of mixed venous blood, an error of ±2 mm Hg may represent an error of ±5 per cent saturation, which could be equivalent to ±1 vol per cent in oxygen content. A similar polarographic error of ±2 mm Hg in measuring content by a direct method[3] would produce a content error of ±0.3 vol per cent. Therefore, the latter method offers at least a threefold increase in accuracy of measurement of blood which is from 20 to 80 per cent saturated. In our laboratory, the direct method of measuring oxygen content of blood[3] agrees within ±0.2 vol per cent with the content determined by the Van Slyke method, regardless of the degree of saturation. Although it is not readily available in most clinical laboratories, the direct method of measurement of content offers the advantages of speed and accuracy over the method based on estimation from saturation.

Mixed Venous Sampling Errors

Barratt-Boyes and Wood[11] have demonstrated the importance of the position of the sampling catheter tip in determining the validity and variability of the oxygen content in mixed venous blood samples. Rapidly-drawn duplicate samples from the pulmonary artery indicated minimal variation in saturation due to changes in cardiac output and laminar blood flow. While somewhat more variability was noted in duplicate samples drawn from the right atrium or ventricle, no systematic difference between oxygen saturation of samples from these sources and from the pulmonary artery was evident. They found, however, that mixed venous samples drawn from the superior vena cava averaged 3.2 per cent lower in saturation than right atrial or ventricular blood.

Effect of Cardiac Output on Shunting

No significant relationship between cardiac output and shunting was evident. However, the three patients with the largest shunts when breathing air (28, 30, 44 per cent) also had the greatest cardiac outputs. The effect an increase in cardiac output might have on the degree of shunting can only be a matter of conjecture without data on the changing size of the pulmonary capillary bed. If the oxygen consumption and size of the pulmonary capillary bed remain relatively unchanged, an increase in cardiac output will raise Cv_{O_2} and shorten the transit time of blood through the pumonary capillary, and may alter the distribution of pulmonary blood flow relative to ventilation. During breathing of air, a decrease in transit time would reduce the likelihood of complete equilibration of venous blood with alveolar air. The higher content of oxygen in mixed venous blood, associated with high cardiac output, however, would favor rapid equilibration of blood with the alveolar air. If the pulmonary capillary bed is capable of expansion through opening of reserve capillary areas, as during exercise, then the transit time through the lung may remain near normal in the face of an increased cardiac output. The net effect of the elevated Cv_{O_2} and relatively normal transit time would be a reduction of the total physiologic shunt, by improving perfusion relative to ventilation. It could be reasoned that our three patients with the largest intrapulmonary shunts might have experienced some reduction in shunt by increasing both the cardiac output and the pulmonary vascular bed. It is also conceivable that during inhalation of oxygen certain areas of the lung could be so overperfused and have such a short transit time that full equilibration of mixed venous blood with alveolar oxygen would not occur. A measurement of true shunt during breathing of oxygen would register blood coming from these areas as blood flowing through anatomical shunts or areas of atelectasis. With an expanded pulmonary capillary bed and an increase in the Cv_{O_2} following an increase in cardiac output, this portion of the true shunt might well be reduced.

Before the direct effects of cardiac output on shunting can be determined, then, consideration must be given to the size and flexibility of the pulmonary capillary bed in relation to cardiac output. The dynamics of the pulmonary capillary bed may be as important as cardiac output in determining the transit time and the ultimate degree of shunting.

Problems with Estimating Shunt from A-aDo_2

Changes in the arterial oxygen tension or A-aDo_2 frequently are interpreted as indicating changes in the intrapulmonary shunt.[7, 14, 15] Our data, which closely relate the A-aDo_2 to degree of shunting, support the validity of this association. Kelman, however, in a theoretical study, had emphasized that reductions in cardiac output alone may account for increases in A-aDo_2 without changes in the underlying shunt.[16] It can be estimated from figure 2 that in the presence of a 20 per cent shunt A-aDo_2 could increase from 310 to 550 mm Hg (an increase of 0.75 vol per cent) on the basis of an increase in the $Cao_2 - Cvo_2$ from 4 to 7 vol per cent alone, without a change in the size of the shunt. Had $Cao_2 - Cvo_2$ been assumed constant, the shunt would have been calculated at 30 per cent. The clinical significance in such a situation becomes apparent in determining the principal form of therapy: Had an increase in the $Cao_2 - Cvo_2$ and its effect on the A-aDo_2 not been recognized, the main effort might have been to improve ventilation to correct an apparently-worsening intrapulmonary shunt, rather than to treat simultaneously a failing circulation. It also can be inferred from figure 2 that the degree of intrapulmonary shunting will be affected more by a change in cardiac output when the shunt is larger than when it is small.

In many acute clinical situations, then, it may be necessary to determine to what extent changes in arterial tension reflect changes in the intrapulmonary shunt or in cardiac output. With a change in oxygen content following a change in cardiac output, mixed venous blood, on passing through an existing intrapulmonary shunt, will directly alter A-aDo_2. Unless this change due to venous contamination through the shunt is recognized, an erroneous estimation of the intrapulmonary shunt will result. When the cardiac output is normal and reasonably stable, however, measurement of A-aDo_2, because of its simplicity, remains a useful index of intrapulmonary shunting.

References

1. Colgan, F. J., and Whang, T. B.: A method for measuring the functional residual capacity and dynamic lung compliance during oxygen and halothane inhalation, Anesthesiology 28: 559, 1967.
2. Severinghaus, J. W.: Blood gas calculator, J. Appl. Physiol. 21: 1108, 1966.
3. Theye, R. E.: Blood O_2 content measurement using the O_2 electrode, Anesthesiology 28: 773, 1967.
4. Colgan, F. J., Whang, T. B., and Gillies, A. J.: Atelectasis and pneumothorax: Effect on lung function and shunting, Anesthesiology 29: 923, 1968.
5. Cournand, A., Riley, R. L., Breed, E. S., Baldwin, E. DeF., and Richards, D. W.: Measurement of cardiac output in man using the technique of catheterization of the right auricle or ventricle, J. Clin. Invest. 24: 106, 1945.
6. Panday, J., and Nunn, J. F.: Failure to demonstrate progressive falls of arterial Po_2 during anaesthesia, Anaesthesia 23: 38, 1968.
7. Colgan, F. J., and Whang, T. B.: Anesthesia and atelectasis, Anesthesiology 29: 917, 1968.
8. Heibrunn, A., and Allbritten, F. F.: Cardiac output during and following surgical operations, Ann. Surg. 152: 197, 1960.
9. Clowes, G. H. A., Jr., Del Guercio, L. R., and Barwinsky, J.: The cardiac output in response to surgical trauma, Arch. Surg. 81: 60/212, 1960.
10. Snyder, J. C.: The cardiac output and oxygen consumption of nine surgical patients before and after operation, J. Clin. Invest. 17: 571, 1938.
11. Barratt-Boyes, B. G., and Wood, E. H.: The oxygen saturation of blood in the venae cavae, right-heart chambers, and pulmonary vessels of healthy subjects, J. Lab. Clin. Med. 50: 93, 1957.
12. Rodman, T., Close, H. P., and Purcell, M. K.: The oxyhemoglobin dissociation curve in anemia, Ann. Int. Med. 52: 295, 1960.
13. Roughton, F. J. W.: In Handbook of Physiology, vol. 1: section 3, Respiration. Williams & Wilkins, Baltimore, 1964, p. 779.
14. Diament, M. L., and Palmer, K. N. V.: Venous/arterial pulmonary shunting as the principal cause of postoperative hypoxaemia, The Lancet, 1: 15, 1967.
15. Bergman, N. A.: Components of the alveolar–arterial oxygen tension difference in anesthetized man, Anesthesiology 28: 517, 1967.
16. Kelman, G. R., Nunn, J. F., Prys-Roberts, C., and Greenbaum, R.: The influence of cardiac output on arterial oxygenation: A theoretical study, Brit. J. Anaesth. 39: 450, 1967.