Respiratory Insufficiency Following Open Heart Surgery *

JOHN J. OSBORN, M.D., ROBERT W. POPPER, M.D.,
WILLIAM J. KERTH, M.D., FRANK GERBODE, M.D.

From The Institute of Medical Sciences, Presbyterian Medical Center, San Francisco, and the Departments of Surgery and Pediatrics, Stanford University School of Medicine, Palo Alto, California

THE PATIENT, in difficulty after perfusion and open-heart surgery (in common with other sick surgical patients), may exhibit cyanosis, hypotension, tacchycardia or increased respiratory effort. These signs often are ascribed to respiratory acidosis with hypoventilation or to metabolic acidosis due to reduction in cardiac output or to a combination of both. In this paper, we will describe quantitative measurements of respiratory and cardiac function in patients following open-heart surgery.

The data showed that the sickest patients often hyperventilated and had normal or increased cardiac output. The most likely anatomical explanation was diffuse atelectasis.

Methods

Computing and recording equipment was redesigned and adapted for use in the surgical recovery room for the following general groups of measurements:

Respiratory Measurements. A small, tight-fitting mask was adapted from a standard anesthesia mask, by incorporation of a section of 120 mesh stainless steel screen to make a simple functional pneumotachygraph (Fig. 1). A pressure sensing catheter was led from the inside of the mask to a sensitive strain gauge, allowing recording of air pressure gradient (inside mask to room air) across the screen which is equivalent to air flow. A second small catheter led from inside the mask to a Liston-Becker rapid response infra-red CO_2 analyser,*** to give a continuous record of expired CO_2 concentration. The mask can be held over a patient's nose and mouth for about 30 seconds, to obtain a complete record of respiratory rate, respiratory flow and expired air PCO_2. Such a mask is not

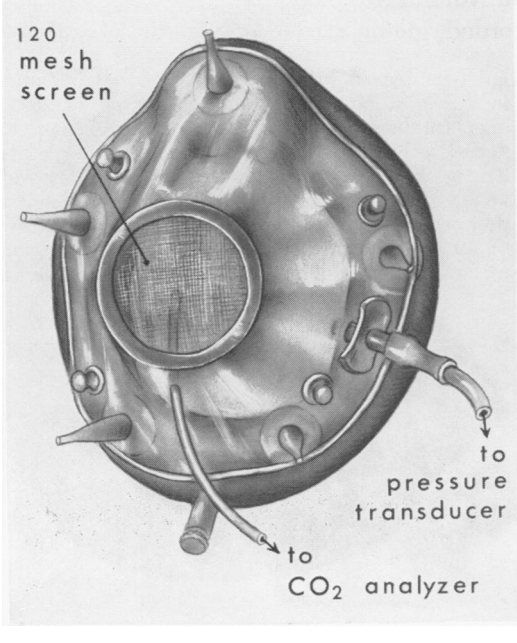

FIGURE 1.

* Presented before the American Surgical Association, Washington, D. C., May 9–11, 1962.

** Aided in part by grants from the United States Public Health Service and The San Francisco Heart Association.

*** Spinco Division Beckman Instruments, Palo Alto, Calif., and Electronics for Medicine, Inc., White Plains, N. Y.

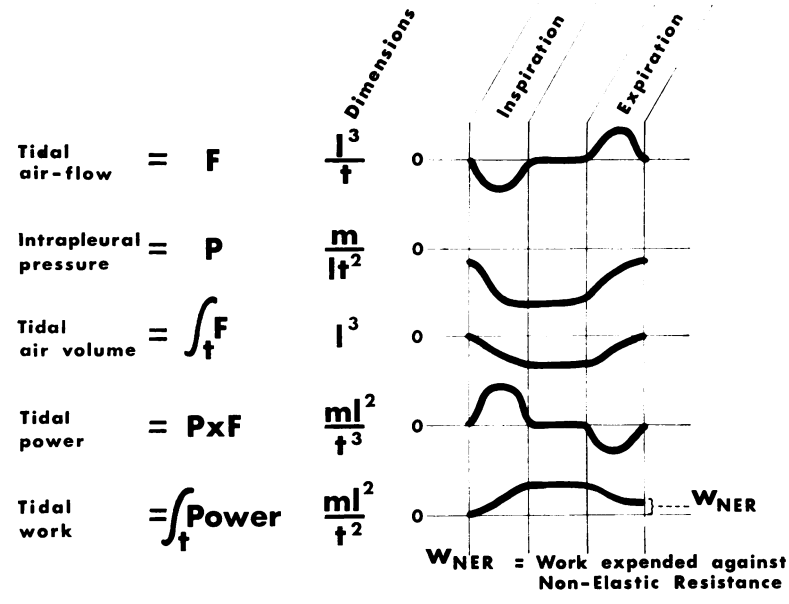

FIGURE 2.

quite a perfect pneumotachygraph, as it gives somewhat a nonlinear response, but its resistance to respiration is extremely low so that it introduces very little artefact and the nonlinear error is relatively small. In the recording and computing apparatus, air flow was simultaneously converted to air volume by electric integration and recorded, giving tidal and minute volume. In many patients, intrapleural pressure was also recorded by tapping into one of the chest drainage tubes, which usually remained open for at least 24 hours, giving a direct measurement of intrapleural pressure. In these patients lung compliance and work of moving the lung (excluding the chest wall) against elastic and against nonelastic resistance could be calculated. De-

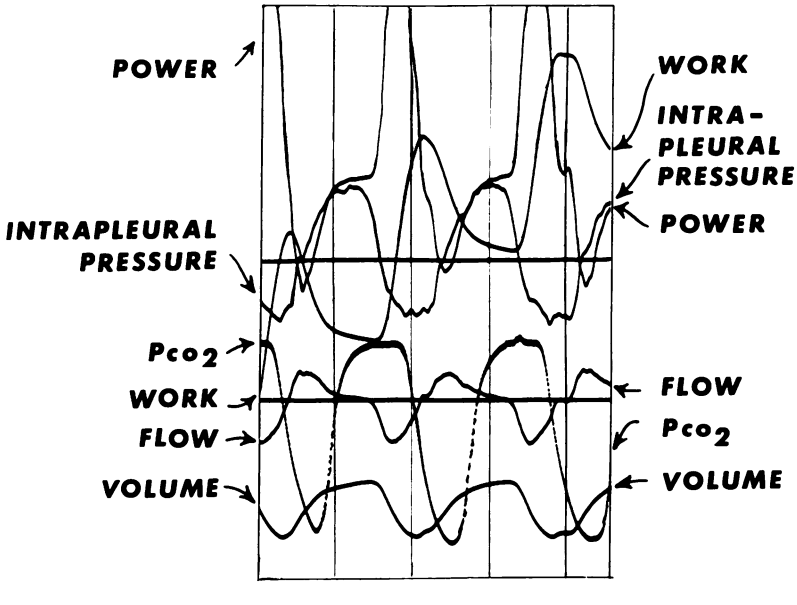

FIGURE 3.

TABLE 1. *"Pure" Hypoventilation—Mitral Insufficiency*

	Arterial PCO_2 mm./Hg	Standard Bicarbonate mEq./L.	Cardiac Index
7 Days postoperative	66	28	3.0
After 6 hours on respirator	31	29	—
After 18 hours on respirator	23	30	—

tails of the method are beyond the scope of this paper, but Figure 2 presents a summary of the units involved and the calculations carried out by the analogue computer, while Figure 3 shows a sample record taken in the recovery room on a patient.

Blood Gases. Blood pH and pCO_2 were determined by a micromethod (Astrup).[1] Arterial samples were obtained either by direct arterial puncture or from free flowing blood from a finger or toe stick, after previous warming of the area. Venous blood samples were obtained from a catheter inserted during surgery into the superficial saphenous vein with the tip lying high in the inferior vena cava or in the right atrium.

Cardiac Output, blood volume and the separation of pulmonary and systemic blood volumes were obtained by precordial counting of rapidly injected radioactive Rose Bengal, with the counting head above the origin of the pulmonary artery and aorta.[3]

Approximately 150 patients were studied following open-heart surgery with perfusion, though not all measurements were made on all patients. Most patients underwent some degree of induced hypothermia.

Results

No obvious pattern of abnormality of lung mechanics (such as change in compliance or resistance) was found either in association with particular lesions or length of operation or perfusion. There were large individual variations in these parameters which will require a study of a larger series of patients for statistical analysis. However, when data on ventilation and blood-gas exchange were examined, they could be assembled into three clearly separate categories of patient condition. The first

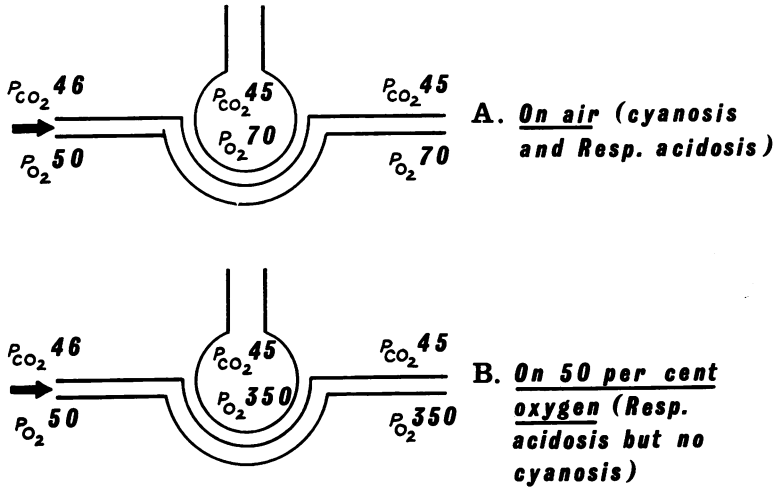

FIG. 4. Hypoventilation with normal circulation—no alveolar-arterial gradient.

two were simple and expected; being the classic syndromes of uniform hypoventilation and of reduced cardiac output. They were also surprisingly rare and so we will describe them only very briefly.

Primary hypoventilation occurred in not more than 5.0 per cent of our patients. It was easily defined by the finding of a high alveolar and arterial PCO_2 with little gradient between alveolar air and arterial blood. Actually, the measurement of expired PCO_2 alone, which took only a few seconds to make, was diagnostic, if the level was above 50 mm. or 7.0 per cent PCO_2. These patients usually had an elevated serum bicarbonate, if they had time to compensate. Table 1 presents data following surgery on a middle-aged woman with mitral insufficiency, pulmonary hypertension and chronic bronchitis. She was greatly improved by assisted positive pressure respiration. It is important to note that she was not cyanotic on 50 per cent oxygen in the oxygen tent, even while acidotic, even before the assisted respiration. Clearing of cyanosis with a moderate rise in inspired oxygen concentration is characteristic of uniform hypoventilation. This is illustrated in Figure 4. Only a moderate increase in inspired oxygen concentration is enough to raise hypoventilating alveoli to a high oxygen tension, though of course, this does not change the slowed rate of CO_2 elimination.

The second classic syndrome, that of simple reduction in cardiac output with adequate pulmonary function was also rare. Table 2 gives data on such a case and Figure 5 illustrates it diagramatically. The cardiac index is very low, but it is important to note that the arterial blood pH is normal and the arterial blood PCO_2 is low, not high. The venous blood pH is low, however, and its PCO_2 is high, as shown by the large A-V difference. This case illustrates that study of arterial blood alone is of very little use for diagnostic purposes because, in the presence of low output with normal lungs, the small amount of

TABLE 2. *"Pure" Low Cardiac Output—Day After Surgery*

Cardiac index	2.1
Arterial pH	7.39
Venous pH	7.28
Alveolar PCO_2	35
A-V Difference for PCO_2	23

blood trickling through the lungs is entirely well ventilated and, therefore, has a high oxygen saturation and low CO_2 concentration. At the same time, a sample of mixed venous blood (which represents tistue conditions) will be extremely acidotic.

Although our study and apparatus had been designed largely to try to diagnose reduced cardiac output or reduced ventilation early in patients who were getting into trouble and, although such cases did occur occasionally as described above, by far the largest group of patients who exhibited clinical distress showed no reduction in cardiac output or total ventilation. To simplify presentation of these data, we have used a group of patients who underwent repair of uncomplicated atrial septal defect as a control group against which to contrast findings of patients who underwent longer and more difficult perfusion and surgery. For uniformity, findings presented here are those of the day after surgery, though measurements carried out on other days showed the same general

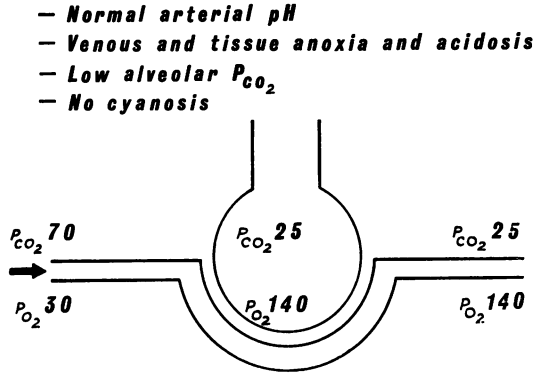

FIG. 5. Reduced circulation with normal ventilation (shock).

TABLE 3.

	IASD	Tetralogy	
		Final Recovery	Died Later
No. cases	12	9	2
Cardiac index	4.0	3.5	4.0
Respiratory min.-vol. L./min./m.²	4.5	14.3	17.3
A-V Difference for PCO_2 mm./Hg	8	14	9
Standard bicarbonate mEq./L.	24	26	21
Arterial pH	7.38	7.4	7.29
Venous pH	7.3	7.35	7.26
Arterial PCO_2 mm./Hg	47	47	54
Venous PCO_2 mm./Hg	53	59	62
Alveolar arterial gradient for PCO_2 mm./Hg	4	6	15

trend. The greatest contrast found was between the patients with atrial septal defect, who underwent rather benign courses, and the patients with tetralogy of Fallot who often had stormy postoperative courses. Selected data on these two groups is presented in Table 3. Among the tetralogy group we have separated off two patients who were so sick that they later died.

The first line of Table 3 lists the mean cardiac index of each group and it is notable that the tetralogies show a mean cardiac index only slightly below that of the atrials, with the two sickest patients (those who later died) having a mean cardiac index equal to that of the atrial group. In the second line, respiratory minute volume is listed (corrected for body surface area). Here the difference is very large. The patients who later died having almost a fourfold increase in corrected minute volume of respiration over the atrials. The data on minute volume for the tetralogy group is based on only three cases with technically complete records on the day after surgery, but it is confirmed by similar measurements on the day of surgery and on many patients with postoperative difficulty, but with other diagnoses. The next two lines contrast the same groups for arteriovenous difference (for PCO_2) and for serum bicarbonate. There is no essential difference except that the two sickest patients do show a moderate metabolic acidosis. Again, in the following lines, there is no difference between the atrial group and the tetralogy group for arterial or venous blood pH or PCO_2 except

TABLE 4. *Means and Standard Deviations for Three Major Diagnostic Groups, Measured the Day After Surgical Repair*

Day After Surgery	Cardiac Index	Ventilation Min.-Vol. L./min/m.²	Alveolar-Arterial Gradient for PCO_2
IASD and/or pure pulmonary stenosis	4.7 ± 1.51	3.95 ± 1.86	0.71 ± 1.86
IVSD	4.67 ± 1.54	6.97 ± 3.14	5.12 ± 2.30
Tetralogy	3.30 ± 2.27	15 ± 2.59	7.42 ± 6.03

that the two sickest patients exhibit moderate acidosis.

The essential clue we believe is found in the last line, in which the gradient between alveolar PCO_2 and arterial PCO_2 is listed for each group. For the atrials, it is almost normal but it is clearly elevated for the tetralogy group as a whole and extremely high for the two who died. In fact, for those two, it is high enough to entirely explain the rise in arterial PCO_2 and fall in pH. Although, we did not have instrumentation to measure PO_2 in these patients, careful notes were made of the degree of cyanosis of individual patients and, whenever possible, arterial blood saturation was estimated by visual colorimetry against known standards. In general, the sick patients in the tretralogy group were somewhat cyanotic with demonstrable unsaturation of arterial blood and this cyanosis did not disappear on administration of high concentrations of oxygen. This was in marked contrast with patients known to have uniform hypoventilation.

Although the largest differences were observed between patients with simple atrial defect and with tetralogy, differences in the same direction, but of somewhat smaller degree were found whenever patients were grouped according to difficulty of surgery or length of perfusion. Table 4 presents data including means and standard deviations on three gross groups, first those with either secundum atrial defect or pure pulmonary stenosis; secondly, those with simple ventricular defect (including several with pulmonary hypertension) and thirdly; those with cyanotic tetralogy of Fallot.

Discussion

From the data above, it appeared that the greatest number of patients who appeared "sick" in the postoperative period suffered neither from primary reduction in cardiac output nor from simple hypoventilation. There are several possible physiological causes for the findings of normal cardiac output, greatly increased ventilation, increased alveolar arterial gradient and cyanosis. Transport of oxygen to the tissues and of CO_2 from tissues to air depends on three general systems, as illustrated in Figure 6. One is the system of air movement or ventilation, a second is the system of blood movement measured as cardiac output, but third is harder to define, consisting of the *transport zone* between blood and air. It depends on the available area of alveolar membrane, on the ventilation-blood flow ratio, on the thickness of the alveolar membrane and on the amount of blood or air shunted past nonfunctioning alveoli.

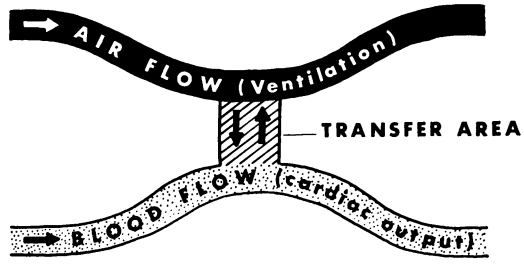

FIG. 6. The three critical parameters of blood-gas transport.

One possible explanation for the findings listed might be overloading of the available capillary bed. This is shown as a diagram in Figure 7. To investigate

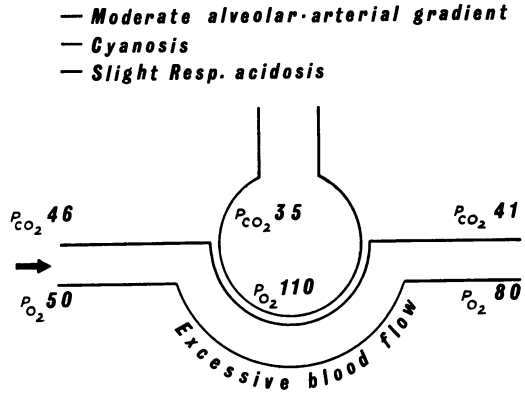

FIG. 7. Pulmonary A-V shunting because of overloaded capillaries.

TABLE 5.

Day After Surgery	No. Cases	Cardiac Index	Ventilation Min.-Vol. L./min./m.²	Alveolar Arterial Gradient for PCO_2 mm./Hg
IASD	12	4.3	4.0	4
Pure pulmonary stenosis	5	6.6	3.8	0

its probability, we have contrasted a group of patients with pure pulmonary valvular stenosis with the patients with tetralogy of Fallot. All these patients are said to have decreased pulmonary blood flow and hypovascularity of the pulmonary bed before surgery, so that if overloading of an underdeveloped capillary network were a valid explanation, one would expect patients with pulmonary stenosis to have the same postoperative findings as those with tetralogy. However, as shown in Table 5, they do not, but are essentially normal after surgery.

Alveolar membrane block or diminished diffusing capacity of the alveolar membrane is also possible and several workers have described diminished diffusing capacity following perfusion.[4, 6, 7] But, if diminished diffusing capacity of the alveolar membrane were the primary disturbance, one would expect a much bigger alveolar arterial gradient for PO_2 than for PCO_2 because of the much greater diffusability of carbon dioxide. Our gradient for PCO_2 among the sicker patients appears much too large for simple reduction of diffusion capacity.

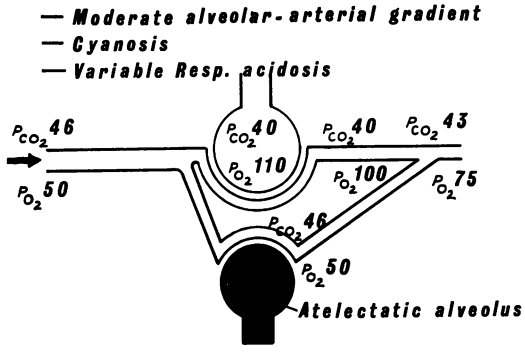

FIG. 8. Pulmonary A-V shunting because of atelectasis.

There is one explanation which fits our data more closely; the concept of pulmonary arteriovenous shunting past atelectatic alveoli. This is illustrated in Figure 8. Of course, if part of the pulmonary blood is shunted past atelectatic alveoli, that part of the blood receives no oxygen. No amount of hyperventilation or administration of concentrated oxygen will prevent cyanosis in the mixed arterial blood. But, hyperventilation can reduce the PCO_2 well below normal, so it is perfectly possible for the normal alveoli to hyperventilate and reduce the PCO_2 of the ventilated blood to the point where the mixed arterial blood is perfectly normal for PCO_2 despite the shunt.

What can the body do to protect itself, if there is a major amount of pulmonary arteriovenous shunting or diffuse atelectasis? It can only do what our sick patients have apparently done—hyperventilate to maintain a normal mixed arterial PCO_2 and increase cardiac output to try to carry the largest amount of oxygen possible with the unsaturated arterial blood. But, such a patient's life depends on his being able to hyperventilate and work his circulation to the limit. When he tires, his compensation topples suddenly and completely. Respiratory and metabolic acidosis which have been held off by a great effort to hyperventilate and pump extra blood develop within minutes, if he tires, and allow arterial PCO_2 to rise or increased tissue

TABLE 6.

Day After Surgery	No. Cases	Length of Perfusion (Min.)	Ventilation Min.-Vol. Day After Surgery L./min./m.^{2}
IASD or pure pulmonary stenosis	17	40	4.0
IVSD	13	80	7.0
Tetralogy	11	110	15.3

anoxia to develop. As ventilation is reduced, the arterial blood becomes acidotic, cardiac output drops, he can no longer maintain the increased work of respiration and circulation. The whole delicate house of cards is down and respiratory and metabolic acidosis develop very fast and at the same time.

We have described a syndrome which includes hyperventilation, normal or increased cardiac output and increased alveolar arterial gradient. We have suggested that it is most likely due to shunting of pulmonary blood through areas of atelectasis and we might add that the atelectasis is almost certainly very diffuse, because of the absence of major areas of atelectasis on x-ray. We do not as yet have data to define the primary cause of this condition. It may be connected with perfusion primarily or with the handling of the lungs during the period of perfusion. Certainly, it shows an association with length of perfusion, as shown in Table 6. Whether it is associated with hypothermia or not, we are unable to determine from our data. Although, it was generally true that patients with longer perfusions were carried to lower body temperatures than those with short perfusions, we were unable to find an significant correlation between lowest body temperature and development of the syndrome described among patients of any given length of perfusion.

We have previously pointed out that the pulmonary congestion syndrome following perfusion may also occur following other procedures.[2] Gardner *et al.*[5] have described an increase in surface tension of alveolar lining material following anoxia or long perfusion, with loss of a specific surface active agent. They have been able to associate this with pathological studies suggesting diffuse atelectasis, as a result of the increase in intra-alveolar surface tension and their experimental findings could be an important explanation for our data.

This is not a paper on therapy, but it is evident that therapy must be directed toward assisted ventilation with particular

TABLE 7. *Effect of Assisted Respiration on Alveolar-Arterial Gradient for PCO_2*

	Venous PCO_2	Arterial PCO_2	Alveolar PCO_2	Alveolar Arterial Gradient
Spontaneous respiration	62	54	42	12
30 Min. positive pressure respirator	59	43	41	2

effort to open up atelectatic alveoli. Positive pressure respiration can, indeed, reduce the amount of shunted blood, as shown in Table 7, compiled from data on an eight-year-old boy before and after one half hour on a respirator. Not only is the arterial PCO_2 reduced, butthe alveolar arterial gradient drops sharply.

Summary

1. Measurements on patients following open-heart surgery with perfusion showed normal or increased cardiac output and increased minute volume of respiration on the part of the patients who were clinically the sickest.

2. Persistent cyanosis and a large alveolar-arterial gradient for PCO_2 strongly suggested diffuse atelectasis as the basic lesion in the commonest postoperative syndrome.

3. There was a positive association between the abnormality of the findings and the length of previous perfusion or difficulty of surgery.

4. Effective positive pressure ventilation was indicated in therapy.

References

1. Anderson, O., Siggaard and Engel & Co.: Scand. J. Clin. Lab. Invest., 12:177, 1960.
2. Baer, D. M. and J. J. Osborn: Amer. Heart Jr., 56:652, 1958.
3. Connolly, J. E., M. B. Bucaner, D. L. Bruns, J. M. Lowenstein and E. Storli: Surgery, 44: 255, 1958.
4. Ellison, L. T., A. R. Nix and R. G. Ellison: Surgical Forum, 12:50, 1961.
5. Gardner, R. E., W. Tooley and T. A. Findlay: Int. Cardiovasc. Soc., Dublin, Sept. 1961 (in press).
6. Kontaxis, A., R. Tomin, B. Wittles, W. Neville and H. Clowes: Surgical Forum, 12:52, 1961.
7. Schramel, R. J., R. Cameron, M. M. Ziskind, M. Adam and O. Creech: J. Thor. & Card. Surg., 38:281, 1959.

Discussion

Dr. John H. Gibbon, Jr. (Philadelphia): Many lives were saved with the recognition that during an operation under general anesthesia, spontaneous respirations were inadequate to prevent the development of respiratory acidosis. Similarly, in recent years, lives are being saved by the recognition that in some patients breathing must be assisted in the postoperative period.

I think the findings here are interesting and somewhat startling, *i.e.*, that only a few patients after open cardiac operations have a diminished cardiac output, and only a few have had very poor pulmonary ventilation.

It is interesting that patients with the tetralogy of Fallot who are quite ill after open-heart operations may have no fall in their cardiac index and that, even when hyperventilated postoperatively, they may show the findings consistent with right to left shunt, presumably through the pulmonary circulation.

Dr. Gerbode and his associates have properly concluded that positive pressure assisted respiration in postoperative periods may be of great value in certain patients.

I am not sure that one can conclude the blood is passing through a completely atelectatic lung, although this may be the case. However, it is difficult to see why this occurs in patients with the tetralogy of Fallot and not in patients with pulmonic stenosis. The other explanation, of course, is that evidence of a right to left shunt may arise from a decreased ventilation-perfusion ratio in certain areas of the lung. Nevertheless, the therapy is clear; you have to help the patient breathe.

Dr. Elliott S. Hurwitt (New York): I wish to comment briefly on the fact that this set of phenomenon is not peculiar to open-heart surgery—that for a large group of general surgeons such as this, the emphasis might properly be placed on the fact that these same phenomena occur in other forms of thoracic surgery and in general abdominal surgery. In a patient population that is increasingly geriatric, undergoing more and more major abdominal surgery, we have found that by doing these determinations repeatedly, many of these same phenomena have been found in very elderly patients. For example, a 90-year-old patient undergoing an operation for an extensive diaphragmatic hernia was found to have a pH of 7.515 in the postoperative period. And we can confirm, of course, the conclusion that Dr. Gerbode has emphasized, namely that assisted respiration can be the critical factor in bringing these patients out of what may well be a terminal course of events.

This is not confined to cardiac surgery and I think that a broader application of this for our general surgical patients will result in considerable salvage.