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.

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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.

DR. CARL A. MOYER (St. Louis): This has been extremely interesting. This phenomenon which you are describing is really a very old one. It was first described by Trube. He termed it pulmonary erection. It is very peculiar that this syndrome, as pointed out by the previous discussor, is not limited to work within the chest. It has actually been recorded to have occurred by such people as Coryllos and Birnbaum in relatively slightly injured people.

Your description of normal to high cardiac outputs, resistant cyanosis, high pCO_2, together with a clear roentgen picture, is very typical of the syndrome that Flegler and Bannister termed congestive atelectasis. We have been interested in this, of course, for now nigh onto twenty years.

Did you make any measurements of the intrapleural pressure changes during each respiratory cycle in these people? One of the characteristics of this syndrome is that the intrapleural pressure changes with each respiration are very large compared to normal, and too, the intrapleural pressure becomes positive at the end of every respiration rather than being still slightly negative.

One of the things that we have recently found, that has produced this in animals with absolute regularity, is very rapidly increasing the concentration of serum albumin while the animals are sodium deficient. Have you any information regarding changes of serum albumin in these cases?

DR. GEORGE CLOWES (Cleveland): I enjoyed very much Dr. Gerbode's clear demonstration of the pulmonary problem following perfusion, which Dr. Gibbon has so well discussed.

I just want to draw to your attention some of the sources of damage of the lung that occur during perfusion in cardiac surgery, some of which are also apropos of other types of surgery. At the meeting held by the NIH in 1958, Open Heart Surgery, Dr. Dewey Dodril of Detroit presented a paper on the focal atelectasis seen following perfusion. At that time Drs. Muller and Damman also drew attention to the congestion and the injury occasioned by high pressure on the venous side of the pulmonary circulation. More recently my associate, Dr. Neville and I, and Dr. Ellison and others have been interested in the matter of this post-perfusion syndrome of the lung.

This phenomenon, I think, explains in part what Dr. Moyer was talking about, as well as the focal atelectasis. It is an intravascular inflammatory lesion in which numerous white cells accumulate on the intima and in the walls of the pulmonary vessels. It is accompanied by peribronchial hemorrhages and not with intra-alveolar heart failure cells. The alveolar septae are engorged and oxygen transmission to the blood passing through the capillaries is reduced.

We think it may be related to denaturation of blood, proteins, and the release of fat. I find, experimentally, that this can also be done with other agents besides blood damage, and I would like to say just a brief word about the corrective matters that Dr. Gerbode brought up. I had the opportunity of reviewing his manuscript and I think that, although he said it is not a paper about therapy, obviously, the implications are terrific.

We agree that the respirator is the answer to this problem if one is so fortunate as to have it. The cardiac output goes down when a patient is relieved of respiratory work by a good respirator. Secondly, there is an improvement of oxygen delivery and pH of the blood, a matter that Dr. Habif brought to your attention so well yesterday.

And, finally, I believe, from our own work on cardiac output, that there is an improvement of the reactivity of the vascular system with particular reference to venous return as pH and oxygen delivery are improved.

DR. FRANK GERBODE (closing): In regard to the remarks of Jack Gibbon, he is, of course, one of the great pioneers in this field and has constantly emphasized the necessity for taking care of the ventilation of the patient not only postoperatively but during operation.

We have found, with regard to respirators, that for many respiratory problems and for assisted respiration in some conditions that the Bird instrument is quite good. However, when we are in real trouble and expect to have a patient on the respirator for a number of days, where the problem is a difficult one requiring a tracheostomy, there is no question in my mind but that the Engstrom unit is more satisfactory. As you know, this is an instrument which runs on controlled volume. You can control the volume of gas delivered to the patient, and it is not necessarily related to pressure.

I appreciate Dr. Hurwitt's remarks. We have in the past, with Maurice Hood, done some of these same studies on patients with ordinary general surgical procedures and have found, for example, that if a patient who has undergone cholecystectomy is not encouraged to breathe postoperatively, certainly the CO_2 will rise and respiratory acidosis will develop.

I was tremendously pleased with Carl Moyer's remarks and will certainly look up the work of Traube in the German literature.

We certainly have measured intrapleural pressure. Dr. Osborn has a paper coming out soon showing how one can apply a formula to this and arrive at an approximation of the amount of respiratory work being done, intrapleural pressure being one of the factors in the formula.

In regard to George Clowes' remarks, we certainly agree with everything that he has had to say and have followed his work very carefully, because we found if we did not we would get behind in this field. I would certainly say, in regard to what he said about respiratory devices, the Engstrom unit, we think in general, is the better one for the most difficult respiration problems.