The Modern Treatment of Respiratory Failure*

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I suppose it is appropriate that we come to respiratory failure at the end of a longish day. The definition of respiratory failure has an interesting history. Barcroft, 30 to 40 years ago, understood respiratory failure as a tissue phenomenon. He would have described cyanide poisoning to you as an example of respiratory failure. "Ventilatory failure" came into fashion but is not a very good term because total ventilation may be fine but gas exchange may be very poor. Europeans have invented various terms like "global insufficiency," which sounds very impressive in German, but always sounds to me more like a term from the Pentagon than a medical or physiological definition. I prefer to use the term "respiratory failure" to mean everything related to disordered gas tensions; but if you attempt a precise definition, you run into unexpected difficulties. If you define it in terms of blood gases only, you will call patients with an arteriovenous fistula in the lung sufferers from "respiratory failure," which is nonsense. You would also conclude that a man, walking up a mountain, whose arterial oxygen tension was lowered, had "respiratory failure," which isn't strictly true. So you can invent possible definitions only to discard them. I do not think there is much interest in following that line of thought.

I do think it is valuable to distinguish between acute and chronic respiratory failure. We can recognize that there is a state of chronic maladjustment or inadequacy, with compensatory adjustments of polycythemia and bicarbonate retention. We can recognize not only acute and chronic, but acute on chronic; this category includes many of the patients with respiratory failure that we commonly encounter. They have been precipitated into an acute dangerous situation, often having lived satisfactorily for several years with a chronic form of respiratory failure. In table 1 are set out some of the common forms of acute and chronic respiratory failure.

TABLE 1

Respiratory Failure

A. Chronic:
   1. Chronic hypoventilation with normal lungs:
      a. Neuromuscular disorders.
      b. Skeletal deformity.
      c. Primary alveolar hypoventilation syndrome.
   2. Chronic hypercapnia ($pCO_2\uparrow$) and hypoxia ($pO_2\downarrow$) as consequence of chronic lung disease ($\dot{V}/\dot{Q}$ distribution abnormality). Arterial pH usually normal as a result of bicarbonate retention.

B. Acute:
   1. Acute infection superimposed on chronic respiratory failure.
   2. Acute respiratory depression.
   3. Acute status asthmaticus.
   4. Chest injury.
   5. Acute paralysis.
   6. Following cardiac arrest or pulmonary edema.

Insidious Nature of Respiratory Failure

The first point I wish to stress is that progressive hypoventilation is a dangerous condition and very difficult

* Presented as the second of the Seventeenth Annual Stoneburner Lectures at the Medical College of Virginia, March 12, 1964.

sometimes to spot clinically. Let us consider a patient who has just been operated on. He has an oxygen uptake of 300 ml per minute, a respiratory quotient of 0.8, which is average, and an alveolar ventilation of 5 L per minute. His arterial oxygen tension will be 100 mm of Hg on air; his saturation 96%; the arterial CO_2 tension will be 40 mm of Hg; and the pH 7.40. I want you to notice what happens if you just depress his alveolar ventilation, by 1 L per minute, in steps, keeping everything else the same, and assuming that his lung has perfect gas distribution and a normal diffusing capacity. When I drop it 1 L per minute, I have dropped the arterial oxygen tension to 82 mm of Hg. The saturation is still high because of the shape of the dissociation curve; the pCO_2 has gone to 50 mm of Hg and with no bicarbonate adjustment, and the pH will have fallen to 7.32. The next stage of a further liter per minute drop in alveolar ventilation to 3 L per minute results in another fall in arterial pO_2. The saturation is now 87%, which is not detectable as cyanosis, on air. The CO_2 tension will now be about 75 mm of Hg. The patient, from perhaps being a little restless earlier if in pain, is noted by the nursing staff now to be "sleeping quietly." This is due to the anesthetic property of CO_2. The pH is now 7.2 and the patient has reached a critical situation; because if you drop alveolar ventilation another liter per minute, it is evident to everyone that disaster has occurred. The oxygen saturation will be 40%, the arterial oxygen tension is 30 mm of Hg, and the CO_2 is up to 105 mm of Hg. It is this stage that is critical in the postoperative period. In other words, everything may be going well until the patient, perhaps awakening with a lot of pain, is hit with a moderate dose of Demerol. His ventilation is depressed and he now sleeps quietly.

What happens next depends on a number of circumstances, e.g., whether he collapses a lobe of his lung, which is a very serious complication at this stage, or whether he gets a slight degree of pulmonary edema, which increases the work of breathing and drops ventilation still further. Thus, the first important concept I want to present to you is the insidious nature of respiratory failure. It is insidious particularly in the postoperative situation. It is so difficult to diagnose early in cases of chest injury that you would be well advised to distrust your own or anyone else's estimates of ventilation. In these cases, a patient may come to the brink of disaster with every observer convinced, until 20 minutes before, when the blood pressure disappeared, that there really hadn't been "too much of a problem." It has been the great contribution of the last 15 years, with the common availability of laboratory methods, that we are getting a much better idea really of what a dangerous enemy respiratory failure may be. And we also are beginning to understand the entity which was very commonly taught about 20 years ago, namely peripheral circulatory failure. The urgency of teaching about respiratory failure consists in recognizing that you can usually reverse it, at least in part, and that by the time the patient has moved into the next stage of severe tissue hypoxia, or of circulatory failure, it may be too late.

Sieker and Hickam (*Medicine* 35: 389–423, 1956), who I think introduced the term "CO_2 narcosis" for the first time, made the point that, as severe respiratory acidosis is treated properly, and the CO_2 has fallen from 100 to 50 (or so) mm of Hg, the patient may still be confused and drowsy. So, just as in the treatment of diabetic coma the return of consciousness does not follow precisely the curve of the blood sugar, the same is true in treating CO_2 narcosis. This occasionally can give rise to anxiety. In one or two people we have treated, we have been fairly convinced the patient must have suffered irreversible cerebral damage, or possibly a cerebral thrombosis, because we have had the pCO_2 at 40 or 50 for 1 hour or so and there has been little return of consciousness; yet full recovery has occurred.

Circulatory Changes in Respiratory Failure

One of the important physiological contributions to this area was the demonstration by Nahas and Cavert (*Am. J. Physiol.* 190: 483–491, 1957) that in the intact dog, giving CO_2 and reducing the pH from normal levels to 7.1, caused a progressive fall in cardiac output, which fell almost linearly with the pH change. This was mainly due to a fall in stroke volume. Patterson (*Proc. Roy. Soc.* (*London*), *Ser. B* 88: 371–396, 1915) had demonstrated as early as 1915 that CO_2, regardless of its pH effect, had a depressant effect on the isolated dog heart. It has also been shown that the body has a protection against this which is the secretion of catecholamines, which goes some way toward counteracting this effect. So my second point is to emphasize that there is, almost at the beginning, an inevitable interaction between respiratory failure and the state of the circulation.

Superimposed Metabolic Acidosis

There is another very important way in which these manifestations interact. When tissue perfusion is reduced below proper levels, even in the presence of a normal arterial oxygen tension, you get a brisk peripheral tissue hypoxia with accumulation of lactic acid. I think we really learned this from early experiments of perfusing dogs with cardiac bypass circuits in which total cardiac output was inadequate, and watching the metabolic acidosis develop. The point was also brought home strongly when we began to restart hearts that had been stopped. We found that after a few moments of cardiac arrest the pH will be down at 6.9. Simply taking patients who have high pCO_2 values or respiratory failure and plotting, to start with, the plasma bicarbonate against the lactate, (and I do not have many observations here as this is work we have in progress), the higher the lactate, the lower the bicarbonate, which is to be expected. And the lactic acidosis, often reaching quite high levels in these patients, is responsible for a considerable part of the "total" acidosis. So this is the second interaction. Failure of the circulation gives rise to problems at the tissue level. When these are severe enough to cause lactic acidosis, you immediately add a metabolic acidosis to the respiratory one. Table 2 summarizes these events sequentially.

Thus the physician's task is obviously 3-fold. The first problem is to ensure oxygenation. The second is to make sure that this is not causing more hypercapnea. If the patient cannot get rid of CO_2, his ventilation must be assisted and the pCO_2 restored to near normal values. Third, and only 1 or 2 minutes later, the physician should worry about the problem of acidosis—not only the respiratory acidosis but the possibility of a quite severe metabolic acidosis. If you face every problem of respiratory failure with these three thoughts in your mind, and add a fourth, that you should worry continuously about cardiac output and renal blood flow as these often will take the patient away from you when you really deserved success, you will realize that the treatment of respiratory failure is not an isolated phenomenon; nor can it be reduced solely to discussion of whether to do a tracheostomy, or which particular tube to use, or what kind of mechanical respirator to prefer.

TABLE 2

Sequence of Events in Acute Respiratory Failure

1. Acute elevation of pCO_2 .
2. Acute respiratory acidosis (pH low).
3. Secondary circulatory depression with fall in cardiac output and later cardiac arrest.
4. Secondary superimposed metabolic acidosis.
5. Final severe combined metabolic and respiratory acidosis.
6. Cardiac arrest/cerebral damage and central respiratory failure.

Death

The physician who treats respiratory and circulatory failure well is not one man usually, but at least two and sometimes three, who are accustomed to think together when faced with these problems, so that no aspect of the total care is ignored. The best management of such patients requires a group of people accustomed to working together without treading on each other's toes, and accustomed to thinking in terms of several systems in the body at once. This is particularly true after cardiac surgery, when the management of some of these patients certainly requires a team effort of the kind I am sketching.

In table 3, I have summarized the sequence of decisions the physician may make in managing respiratory failure.

I deliberately chose this topic not only because it is something I am involved in and feel strongly about, but because I wanted to concentrate on a field in which I believe there have been rather striking practical advances. The reasons for these are historically interesting. But, I do not think anyone would deny or doubt that the ready availability of arterial blood gas analysis, which must be available in an intensive care unit, at least, 24 hours a day, has really played a major part. I cannot imagine how you can deal with many of these patients without knowing what you are doing. The more centers there are using routine blood gas analysis, the more people have learned that there really is no substitute for this particular kind of analysis in particular cases. This whole field is at a stage when many people are involved in it, some willingly and some unwillingly. I think we are all learning of the situa-

tions in which we do very well and those in which, for some reason or another, we do very poorly. I have concentrated on it because I am quite firmly convinced that this is properly the province of the chest physician, which I am. Although I have been on occasion accused of being a pathologist, and described as a physiologist, I am really a physician. If the modern chest physician does not accept responsibility and take an interest, and I would add, a devoted interest, in this kind of management, he is going to find that it is taken over by others. Of course there are some people, anesthesiologists usually, who feel this ought to be exclusively their province. But I think, in the kind of patient I have been showing you, that these are often problems for the chest physician, who will certainly need help, and assistance, and guidance from others. We found it very useful in our intensive care unit to have the cardiac group and ourselves side by side. Our nurses are as competent to deal with pacemaker-monitor problems and coronary thrombosis patients as they are with respirators. The chest physician must work very closely with cardiologists, certainly with surgeons, and certainly with anesthesiologists. But I would not welcome a situation in which the essential problem of controlling breathing over long periods of time, and of treating people in these categories I have been showing you, is turned over exclusively to people other than the chest physician. With the decline of tuberculosis and the shift of interest to emphysema, the chest physician has to re-evaluate what he is doing. And he has to make up his mind, I believe, to do at least two things: to be able to apply simple physiological tests of function to assist other physicians and himself in the diagnosis and management of lung disease, and to become an expert in managing respiratory problems. The thought I leave with you is that if he does not do both of these things, he will find that his specialty has disappeared.

TABLE 3

Sequence of Decisions in Management of Respiratory Failure

A. Accurate diagnosis: usually necessitating arterial blood gas analysis, x-rays, ECG, etc.
B. Re-establish ventilation plus O_2 administration.
C. Sequence of decisions on assisted ventilation.
   1. Simple assistance via mask plus respirator, plus possibly stimulants. Use of 40% O_2 .
   2. Endotracheal tube if
      a. Prognosis probably hopeless.
      b. Need of assistance probably of short duration.
   3. Cuffed tracheostomy tube
      a. If secretions are a problem.
      b. If long term assistance (more than 4 days) likely.
      c. If pCO_2 is higher than 75 mm for 3 hours or so in spite of simpler measures.
   4. Assisted ventilation plus muscle relaxants
      a. In status asthmaticus.
      b. In tetanus.
      c. In lung injury.
D. Treatment of metabolic acidosis (immediate if cardiac arrest has occurred).