Journal of Critical Care

VOL VIII, NO 2 JUNE 1993

EDITORIAL

Oxygen Kinetics and the Art of Physiological Monitoring

MOST OF THE studies of oxygen kinetics in critically ill patients involve measuring oxygen consumption ($\dot{V}O_2$) after a change in oxygen delivery ($\dot{D}O_2$). These studies cover only a small portion of the $\dot{V}O_2/\dot{D}O_2$ curve and are subject to mathematical and logistical problems, making interpretation difficult at best.[1] In this issue of the *Journal of Critical Care,* Weissman and Kemper measured $\dot{D}O_2$ after a change in $\dot{V}O_2$, using independent measurements of the variables.[2] This approach has generated interesting data that allows interpretation of several aspects of critical care physiology.

The method was simply to measure $\dot{V}O_2$, $\dot{D}O_2$ and related hemodynamic and respiratory parameters before, during, and after a short period of hypermetabolism caused by mild exercise in the form of chest physical therapy (CPT). The patients were old (average, 67 years), postoperative, ventilated, and not septic (ie, $\dot{V}O_2$ was normal). There was a 50% increase in $\dot{V}O_2$, which would be expected to lead to a 50% increase in $\dot{D}O_2$. However, there was only a 17% increase in $\dot{D}O_2$, and the decreased ratio of $\dot{D}O_2$ to $\dot{V}O_2$ resulted in increased oxygen extraction during hypermetabolism. The authors modestly draw the conservative conclusion that the appropriate integrative response occurred and speculate why the $\dot{D}O_2$ did not increase as much as might be expected (eg, old age, heart disease, cardiac depressant medications, short period of exercise). However there is a wealth of physiological data (and some common errors of methodology) in this elegantly simple experiment that makes this the kind of paper worth spending a few hours studying with a group of students or residents.

The data are replotted in Fig 1A and 1B. Data points 1 and 2 are at rest, 3 and 4 after 1 and 2 minutes of CPT, and 5 and 6 are back at rest. The expected increase in $\dot{D}O_2$ during a 50% increase in $\dot{V}O_2$ is shown as point X. Points worth demonstration and discussion include the following.

1. The metabolic rate of these patients is probably normal (points 1 and 2). Normal oxygen consumption for adults is 3 mL/kg/min or 120 mL/m^2/min. Assuming the patients are normal-sized adults, the oxygen consumption is in the normal range. From this observation, we learn that postoperative patients are not hypermetabolic. We can assume that these patients were not septic. We can also assume that the patients were normothermic and not on significant catecholamine infusions. The $\dot{V}O_2$ was measured by comparison of mixed expired gas analysis to inspired gas analysis. The authors do not tell us if this measurement is expressed as ambient temperature and pressure, saturated with water vapor, or standard temperature and pressure, dry. They correctly used totally independent measurements of $\dot{V}O_2$ and $\dot{D}O_2$. Although they did not tell us what the $\dot{V}O_2$ would have been if calculated by the Fick equation using shared variables with $\dot{D}O_2$, from the data they provide we can make some calculations and discover that the Fick-derived $\dot{V}O_2$ is fairly close to the measured values.
2. In our first conclusion (above), we had to state that the $\dot{V}O_2$ was probably normal and make some other assumptions, because the authors did not tell us the size of the patients and did not account for variable patient size in reporting the physiological parameters. Thus, the standard deviations are quite wide. Gas exchange, hemodynamic, and respiratory physiology data should be normalized to body weight or body surface area thus allowing comparison among and between individual patients and groups of patients. The data would be much tighter if it were properly normalized.
3. Similar to $\dot{V}O_2$, $\dot{D}O_2$ is normal in these patients (ie, 4 to 5 times $\dot{V}O_2$). These patients are anemic with hemoglobin concentration approximately one third less than normal but are compensated because the cardiac index has increased one third above normal (again, making assump-

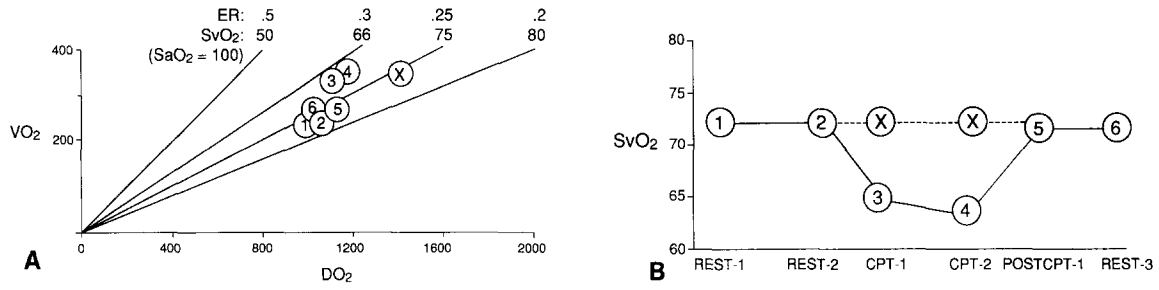

Fig 1. Data from the Weissman and Kemper report[2] replotted for study and discussion. (A) Data on the $\dot{V}O_2/\dot{D}O_2$ diagram. (B) Venous saturation data only. Points 1 and 2, pre-exercise; points 3 and 4, during exercise (chest physical therapy); points 5 and 6, after exercise. *X* represents the expected complete response to exercise, as discussed in the text.

tions about the size of the patients). Because the delivery at rest is about four times consumption, and assuming that arterial saturation is close to 100%, we should expect that 25% of the oxygen is extracted from capillary blood leaving venous saturation of approximately 75%. (The fact that the average venous saturation at rest was 72% probably indicates that the arterial saturation was 97%).

4. During mild exercise (CPT), the metabolic rate increased by about 50%. During maximal exercise, the $\dot{V}O_2$ increases by a factor of 5 or 10, so this increase is relatively small. However, this is the level of hypermetabolism associated with severe sepsis. An important difference is that the hypermetabolism of exercise returns quickly to normal when the exercise stops (as shown in this study). The hypermetabolism associated with sepsis continues unabated for hours or days.
5. For this 50% increase in metabolism, we would expect a compensatory increase in $\dot{D}O_2$ until it reaches 4 to 5 times consumption shown as point X in the figure. The authors mention several reasons why the cardiac response was blunted. An additional factor is that the patients were anemic and functioning at above normal compensatory cardiac output levels at rest. The cardiac output at rest was around 7.5 L/min. Because there is no way to acutely increase the oxygenation or the hemoglobin, full compensation for the transient increase in $\dot{V}O_2$ would have required an increase in average cardiac output from 7.5 L/min to 11 L/min, which might have been a problem for these old hearts. A better explanation, however, is that the hypermetabolic stimulus was very short. A longer period of hypermetabolism, as in sustained exercise or sepsis, would probably have resulted in higher sustained cardiac output. What mechanism adjusts cardiac output in response to metabolism? Is it simply circulating catecholamines? Clearly not, because this would not explain the integrative response to changes in temperature, or pyrogens. Where is the chemoreceptor that "turns on" cardiac output in response to hypermetabolism or "turns off" erythropoietin once oxygen delivery is normalized during hypoxia? No one knows.
6. Because the cardiac output increased only 17%, whereas the metabolic rate increased 50%, what made up the difference in oxygen supply? Obviously more oxygen was extracted from each deciliter of flowing blood (ie, the extraction ratio was higher during data points 3 and 4 than the resting measurements). How much can this phenomenon of increased extraction compensate for an increasing $\dot{V}O_2/\dot{D}O_2$ ratio? This is the central question of the discussion of critical delivery levels, below which oxygen consumption becomes supply dependent. This is a fascinating discussion but is not addressed in this particular experiment. In Fig 1A, the relationships between oxygen consumption and delivery are expressed graphically, with isobars representing various extraction ratios. Notice that the "extraction ratio" is the dividend of the consumption to delivery ratio. For each isobar, the corresponding venous saturation is expressed, assuming the idealized condition of arterial saturation equals 100%.
7. Wouldn't it be marvelous if we had a bedside physiological monitor that continuously displayed the $\dot{V}O_2/\dot{D}O_2$ graph shown in Fig 1A. Suppose we could watch the cursor move from point 1 to point 2, then to point 3 and 4. The patient has become hypermetabolic. There has been a compensatory increase in oxygen delivery but less than we would expect (point X). We can visualize the compensation for hypermetabolism achieved by increasing delivery and that achieved by increasing extraction. We can see how close we are to the critical point of 50% extraction ratio. We can easily visualize if treatment is needed, what treatment is appropriate, and what the expected result would be. This idealized monitoring system does not exist, yet. However, we do have a continuous monitoring system that comes close: continuous mixed venous saturation monitoring.
8. The same data points are shown in Fig 1B, in which mixed venous oxygen saturation ($S\bar{v}O_2$) is related to time. The $S\bar{v}O_2$ tells us only the ratio of consumption to delivery and tells us nothing about the absolute values of either one. Nor does it tell us the cardiac output, arterial oxygen, or pulmonary function. Thus, the information from continuous $S\bar{v}O_2$ monitoring is incomplete and interpretation is a bit of an art. However, with just a little extra information, continuous $S\bar{v}O_2$ monitoring is as close as we can come to the ideal physiological monitor at the present time.

In the experiment described in this report, we know that hypermetabolism and cardiac response accounted for the change in venous saturation between data points

1, 2 and 3, 4. But suppose we notice a similar change in a resting paralyzed patient? The consumption to delivery ratio has changed from 1:4 to 1:3. Is this because of increased $\dot{V}O_2$ or decreased $\dot{D}O_2$? Could it be both? Is the change significant enough to need treatment? Can $S\bar{v}O_2$ be used to monitor the effects of treatment? The answers to all of these questions require more data, but data that is easily acquired. More importantly, we are stimulated to ask and answer the questions before any other physiological parameters suggest that a change has occurred.

Suppose the patient is becoming septic and oxygen delivery matches the increased metabolic rate. This is the situation represented by point X. In this example, the venous saturation has not changed, but fever, tachycardia, and leukocytosis suggest that the patient is hypermetabolic. The $S\bar{v}O_2$ tells us that the patient has compensated for hypermetabolism by moving normally up the isobar, rather than by increasing extraction.

With this understanding of the physiology of oxygen kinetics and with the art of interpreting the continuous $S\bar{v}O_2$ monitor, we can use $S\bar{v}O_2$ not only to detect and treat problems before they become serious but also to titrate positive end-expiratory pressure and inotropic drugs, to decide when and how much to transfuse blood, when and how to use temperature or positioning as treatment, and to construct a mental image of the more complete monitor suggested in Fig 1A.

The purpose of this dicussion is not to critique the Weissman and Kemper report. They promise us similar studies on patients with sepsis and cardiac failure that should be very interesting; we hope they give us all the data with appropriate normalization. The purpose is to point out the breadth and depth of the information that they bring to us based on simple bedside information (all without a Western blot or RNA probe). Surely, we have all seen the $S\bar{v}O_2$ drop during CPT, coughing, or painful procedures. But how often do we stop to analyze the complex processes involved? Not only have the authors done that, they did it in an organized fashion in several patients and challenged the rest of us to learn something. Those who take the time to read this paper will be better intensivists, researchers, and teachers.

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