Adjustment of Stores of Carbon Dioxide During Voluntary Hyperventilation

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Carbon dioxide is stored in the body in various forms, including carbonic acid, bicarbonate and carbamino compounds. The various stores of CO₂ can be considered to be in the lung, blood, soft tissues and bone. In an adult human, these stores are thought to be in the order of 120 liters of CO₂. It is common knowledge that hyperventilation will reduce the level of CO₂ in the blood; however, only a small fraction of the total CO₂ of the body is in the blood. Questions arise, then, as to how much CO₂ can be eliminated by hyperventilation, from where does it come and in what manner does elimination proceed.

Several studies have been made of exchanges of CO₂ in animals and in man. In 1916, Liljestrand found within the first hour of hyperventilation by men that 0.8 to 1.0 liter of stored CO₂ was eliminated for each 1 per cent decrease in alveolar concentration of CO₂. Brocklehurst and Henderson, and Adolph and associates subsequently derived widely varying values for the CO₂ capacity of the body. Several investigators have studied the CO₂ stores of animals. From hyperventilation experiments on anesthetized dogs, Farhi and Rahn found that alveolar CO₂ tension declined in an exponential manner and calculated that one half of the readily available stores of CO₂ was eliminated in 4 minutes.

The present study was undertaken to obtain additional information on the rate of elimination of CO₂ during hyperventilation in man, which might contribute to an eventual better understanding of clinical conditions such as CO₂ retention and narcosis.

Methods and Procedures

For analysis of the rate of elimination of CO₂, it was necessary that hyperventilation, once begun, be maintained at a constant rate. This could not be achieved with a cuirass respirator used on relaxed humans but was obtained in trained subjects as follows. Compressed air of known composition flowed at a constant rate into a spirometer balanced to prevent outflow except during inspiration (fig. 1). By observing the spirometer and maintaining it at a constant average volume, the subject could keep his respiratory minute volume equal to the constant rate of inflow of compressed air.

The subjects were three trained male physicians. After they sat at rest for 10 to 15 minutes, and in two experiments after an overnight fast, a small mask was applied and normal breathing was continued.

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with the flow of air adjusted to equal the spontaneous normal minute volume. Expired gases were directed through an infrared carbon dioxide analyzer* and an expiratory valve to a series of large-bore stopcocks that permitted collection of gas in any of five evacuated neoprene bags† (fig. 1). After 15 minutes, a forced maximal expiration was made following a 24-second period of breathholding at the end-normal inspiratory level. This permitted equilibration of CO₂ between alveolar gas and mixed venous blood and analysis of the CO₂ tension thereof. Natural breathing was then resumed for 10 to 15 minutes, and expired gases were collected for the last 5 minutes in the distal bag. At a predetermined signal, the flow of compressed air was suddenly increased and the subject breathed at a comfortable rate and depth such as to maintain the spirometer recording between two parallel lines previously drawn on the recording drum. The average minute volume for successive collection periods of 5 to 10 minutes was constant within about 1 per cent for intervals up to 64 minutes. While gases were being collected in one bag, the volume of contents of another was measured in a 120-liter spirometer, and samples were taken for analysis of oxygen and CO₂ by the Haldane apparatus. All expired gases were collected throughout the experiment over individual periods of 5 to 10 minutes. At the end of hyperventilation, breathholding and maximal exhalation were repeated as before, for estimating the mixed venous CO₂ tension. For two subjects, the difference between end-tidal and mixed venous tension esti-

*Liston Becker Model 16 (modified sampling cell), Beckman Instrument Company, Springdale, Connecticut.
†Darex balloons, Dewey and Almy Chemical Company, Cambridge, Massachusetts.

FIGURE 1: Diagram of apparatus for collecting expired gases during constant voluntary hyperventilation.
mated by breathholding agreed within 1 mm. of mercury with the difference as estimated by the more complex indirect procedure of DuBois and co-workers.10

During rest and hyperventilation, end-tidal, or alveolar, CO₂ tension was recorded continuously with the CO₂ meter and an Esterline Angus recorder (0.2-second response time). The meter was calibrated for each experiment with known gas mixtures and provided accuracy within 0.5 mm. of mercury. A volume of 430 ml. was required for complete flushing of the mask and meter. When this was added to the 320 ml. needed to purge the subject’s dead space,10 an expired volume of at least 750 ml. was considered necessary to provide samples in the meter equal in composition to end-tidal gas sampled at mouth level. Experimentally determined correction factors were applied to the recorded CO₂ values for tidal volumes less than 750 ml., which occurred only in some control periods before hyperventilation.

From the volume and composition of expired gases, the exchange of oxygen and CO₂ was determined for each period. The total exchange equals the sum of the metabolic exchange and any change of bodily stores. The latter is negligible during the resting steady state preceding hyperventilation. The metabolic production of CO₂ during the later periods of hyperventilation, including that produced by the work of breathing, was defined as that quantity of CO₂ necessary to maintain the exchange ratio (R.Q.) of expired gas equal to that ratio observed prior to hyperventilation. Thus, the rate of oxygen uptake measured during hyperventilation could be multiplied by the initial exchange ratio to estimate the rate of metabolic production of CO₂ for the same period.14,15,7 This was estimated differently for about the first 10 minutes of hyperventilation, because the rate of oxygen uptake increased immediately with the onset of hyperventilation but returned toward the resting value within 10 minutes. The initial increase of oxygen uptake was attributed chiefly to its accumulation by the bodily stores, and the return to near resting values was interpreted as the end of accumulation. During accumulation of oxygen stores, the metabolic uptake of oxygen was considered equal to the values obtained during the 5 to 10-minute period following the apparent end of accumulation. For studies in which hyperventilation was maintained for 10 minutes or less, the metabolic uptake of oxygen was considered equal to the resting value plus 2.6 ml. per liter per minute of excess ventilation.

The change of “whole body” stores of CO₂ was calculated for each period by subtracting the calculated metabolic production of CO₂ from the total quantity of CO₂ that was collected. For total periods of 20 and 60 minutes, this difference was divided into the changes of stores in the lungs, blood and tissues. The change of “lung stores” (strictly, of alveolar gas and not of pulmonary tissue) equaled the measured change of concentration of alveolar gas multiplied by the functional residual capacity, measured separately. The change of “blood stores” equaled the blood volume, assumed to be 80 ml. per kilogram of body weight, multiplied by the change in CO₂ content of blood corresponding to the observed change in CO₂ tension of mixed venous blood, obtained from a standard dissociation curve for oxygenated blood.11 This procedure is
somewhat incorrect because the change of content of arterial blood probably exceeds that of venous blood, but the error is probably less than 10 per cent because of the larger volume of venous blood.

For the hour-long periods of hyperventilation, breathholding measurements of mixed venous tension were made only before and after, but its change after 20 minutes was considered to equal the change of alveolar tension during that time. This was justified by separate observations on two of the subjects in which breathholding measurements were made after 15 or 25 minutes of hyperventilation. The average differences between alveolar and mixed venous tension agreed within 0.5 mm. of mercury whether measured before or after 15, 25 or 60 minutes of hyperventilation. This is, of course, consistent with an increased difference of content of CO$_2$ between mixed venous and arterial blood at the lower values of CO$_2$ tension existing during hyperventilation. The mixed venous tension (Pv) was considered to be the best available, but probably inexact, estimate of "tissue" tension.

Partial dissociation slopes of CO$_2$ were calculated as follows:

"Whole body" dissociation slope = \[
\frac{\text{\Delta total stores}}{\text{\Delta P}_v \text{ (mm. Hg)} \times \text{weight (kg.)}}
\]

"Tissue" dissociation slope = \[
\frac{\text{\Delta total stores} - \text{\Delta lung stores} - \text{\Delta blood stores}}{\text{\Delta P}_v \text{ (mm. Hg)} \times \text{weight (kg.)}}
\]

All volumes for stores are in milliliters at 0° C. and 760 mm. of mercury, dry.

Results

The results of a typical hour-long study are presented in figure 2. From a resting volume of 9.3 liters per minute (body temperature, ambient pressure, saturated), ventilation was increased to 13.5 ± 0.1 liters per minute. Following the onset of hyperventilation, oxygen uptake increased abruptly for 5 or more minutes, and then usually decreased to levels slightly greater than the control. For seven hour-long observa-
tions on three subjects, the average percentage of the control values for oxygen uptake during the periods of 0 to 10, 20 to 30 and 50 to 60 minutes were 107, 103 and 103 per cent, respectively. The initial increase was attributed in part to accumulation of oxygen stores and in part to the metabolic cost of the increased respiratory work. Assuming the values observed at 15 to 30 minutes to represent only the initial metabolic rate plus the oxygen cost of the increased respiratory work, it was calculated that the average oxygen cost of excess ventilation was 2.6 ml. per liter per minute. This value would be expected to be several times the value of 0.5 to 1.0 ml. per liter required at rest\textsuperscript{11}, because the increased ventilation was voluntary and because some respiratory work was expended on the apparatus.

The respiratory exchange ratio increased initially and thereafter decreased slowly; in no experiment did it return completely to the resting value. The end-tidal CO\textsubscript{2} tension decreased rapidly for about 5 minutes and thereafter more slowly. The output of stores of CO\textsubscript{2} was initially at a large rate, decreasing thereafter. Elimination continued throughout the period of 1 hour; in the study illustrated, it amounted to 2.3 liters of CO\textsubscript{2}.

The semilogarithmic plot shows that the rate of output of stores was not that of a single exponential function but that the rate during the later periods was greater than expected from a continuation of the initial rate. That is, the amount of depletion of stores per minute did not represent a constant fraction of the stores remaining at a particular time.

Observations on one subject (JV) showed that the rate of output of stores varied with the degree of hyperventilation (fig. 3). This would be expected to be more strictly true for increased alveolar ventilation than for total ventilation. The divergently large value for output from 0 to 5 minutes in figure 3 represents a study in which tidal volume and thereby alveolar ventilation were in fact relatively greater than they

![Graph](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21337/]

**FIGURE 3:** Relation between rate of output of stores and extent of hyperventilation (increase of minute volume). Values are those observed during periods of 5 minutes' duration.
were for the other observations plotted. After 15 to 20 minutes, the absolute values of rate of output were less affected by the extent of hyperventilation.

Results for seven hour-long studies are presented in figure 4 and table 1. An increase of ventilation of the order of 50 per cent for 1 hour eliminated from 1.5 to 2.5 liters of CO₂ stores. This represents approximately a reduction of the quantity of CO₂ in the lungs by one third, or 50 ml., and in the blood by one sixth, or 50 ml. The major quantity thus came from other tissues. Despite differences of the total output between individuals, no consistent differences between their calculated dissociation slopes were observed. Similarly, when the change of stores during the first 5 minutes was expressed as milliliters of CO₂ per millimeter decrease of alveolar tension per kilogram, average values of 0.56, 0.56 and 0.61 were obtained for the three subjects. The average “tissue” slope for 1 hour was 1.6 ml./mm. kg., or about 80 per cent of the “total body” slope. More than half the elimination for 1 hour was accomplished within the first 20 minutes. During this period, the loss from the blood represented about 30 per cent of the total, whereas it was only 20 per cent of the total for 60 minutes. The values of dissociation slopes, calculated for the 20-minute period of elimination, were in all instances less than the values for the 60-minute period.

Symptoms of acute hyperventilation, such as dizziness or paresthesia, were not noted, nor did apnea follow the end of hyperventilation for 1 hour. Drowsiness was experienced, as were muscular discomfort from prolonged immobilization and other more vague but unpleasant sensations.

![FIGURE 4: Total output of CO₂ stores for three subjects. The concentric circle at 2 minutes is the average value (156 ml.) of seven separate observations in which ventilation was increased an average of 80 per cent.](image)

Comment

The pattern of gas exchange during hyperventilation was in close agreement with that observed by Liljestrand; in his subjects, however, both the degree of hyperventilation and the amount of CO₂ stores eliminated during corresponding periods were greater. With a 300 per cent increase in ventilation for 21 minutes, two of his subjects depleted their stores by about 2.2 liters, whereas our subjects eliminated about 1.2 liters with a 50 per cent increase of ventilation. With constant hyperventilation for 40 minutes, one subject of Liljestrand eliminated about 3.2 liters with a 250 per cent increase of ventilation. It appears that the output of stores may not increase proportionately when ventilation is increased by more than 100 per cent.
<table>
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<th>Subject</th>
<th>Study</th>
<th>Body weight, kg.</th>
<th>Exchange ratio</th>
<th>Terminal</th>
<th>Period of hyperventilation, minutes</th>
<th>Increase in minute volume, per cent</th>
<th>Mixed venous CO₂ tension, mm. Hg</th>
<th>Decrease of stores, ml. (standard temperature and pressure, dry)</th>
<th>Dissociation slopes, ml.</th>
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*After overnight fast.
†Estimated by breathholding after the 60-minute period, and by change of alveolar tension for the 20-minute period.
Farhi and Rahn, in their informative analysis of gas stores, concluded that elimination of the readily available stores of CO₂ in anesthetized dogs proceeded in an exponential manner, with a half time of 4 minutes. However, our results, as well as those of Liljestrand, in man indicate a more complex and prolonged process. Even if one determines a minimal half time from the initial and terminal rates of output, it is at least about 15 minutes. Also, Shaw and Messer's data on cats are probably not consistent with a half time as small as 4 minutes. It appears unlikely that all of the stores would exchange in a manner describable by a single exponential function, as this implies a single bodily pool of CO₂. There are probably multiple sites or pools with varying rates of exchange. The results presented here demonstrate the different rates of exchange of alveolar gas, blood, and "tissues." The much larger dissociation slopes obtained from prolonged observations in animals (table 2) demonstrate the existence of CO₂ in tissues, including bone, that is very slowly exchangeable.

The change of tension of mixed venous blood has been used to estimate that of the "tissues." The extent to which this is justifiable in an unsteady state is uncertain. In three studies of Liljestrand, hyperventilation was maintained for about 20 minutes and then reduced for about 30 minutes to approach a new steady state; in these studies, there is no consistent difference in dissociation slope whether it is calculated from the stores output and change of alveolar tension at 20 minutes, when the exchange ratio was still increased, or at 50 minutes, when the ratio had returned to control values.

Our values for the "whole body" dissociation slope, averaging about 2 ml./mm./kg., are similar to those previously obtained in man and animals by observations of similar duration (table 2) and pertain to the extent of equilibration obtainable in 1 to 2 hours. With more nearly complete equilibration over periods of many hours or days, much larger values are obtained.

The dissociation slopes calculated from different observations were of similar order but did not agree closely for an individual, whether the change in mixed venous tension or the directly measured change in alveolar tension was used in calculation. Some of the variability may have been caused by changes of metabolic rate during the procedure. The methods used for calculation of change of stores, assuming a constant metabolic R.Q., are apparently justifiable when total metabolic rate is constant during the control and the hyperventilation periods. However, we have some reservations about their exactitude in the situation when a simultaneous increase in metabolic rate occurs during the period of elimination of stores caused by the increased rate of ventilation. It was also noted that in some studies in which the uptake of oxygen increased during the latter part, probably from muscular activity associated with restlessness, the alveolar CO₂ tension remained essentially constant throughout the later periods. Presumably, there was also an increasing metabolic production of CO₂, which offset the continued gradual decrease of alveolar tension that would be excepted from elimination of stores alone. However, the failure of elimination to proceed at a single exponential rate was observed whether or not the rate of oxygen uptake increased.

The quantitative aspects of elimination of stores of CO₂ bear on the problems of therapeutic management of patients with chronic retention of CO₂. An estimate of the quantity of retained CO₂ may be made from the finding that rats exposed to 10 per cent CO₂ for periods up to 4 weeks showed an average increase in total body CO₂ of about 20 per cent. This probably would represent more than 20 liters in adult humans, which is large relative to the 2 liters that were eliminated by hyperventilation for 1 hour. It is uncertain whether similar elimination would be achieved by mechanical hyperventilation of patients with pulmonary emphysema and CO₂ retention.

The arterial CO₂ tension is often used clinically to assess the level of CO₂ in the body. It should be recognized that such an interpretation, if strict, implies the existence of a steady state. The arterial tension, neglecting venoarterial shunts, reflects most directly the alveolar tension, which may be altered much more rapidly than that of the tissues. Following a maintained change of the ratio of alveolar ventilation to metabolic production, as in these studies, prolonged periods are required to approach the new equilibrium. However, the physiologic mechanisms that regulate breathing normally act to maintain constancy of this ratio, thus tending to minimize the extent

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<th>TABLE 2—CO₂ DISSOCIATION SLOPE OF THE WHOLE BODY</th>
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*This report.*
of departures from the steady state. In view of the many factors affecting the production, storage and elimination of CO₂ that are encountered in patients with CO₂ retention, it is not clear to what extent the theoretically necessary steady state can or must be achieved to permit the proper evaluation of tissue levels of CO₂ from those measured in arterial blood. However, it has been observed that coma may persist during recovery from prolonged CO₂ narcosis even after the CO₂ tension in the arterial blood has been reduced to levels not ordinarily associated with alterations of consciousness.15

SUMMARY

Adjustments of body stores of carbon dioxide were studied during voluntary hyperventilation for 1 hour at a constant rate by trained subjects. Healthy men eliminated from their stores an average of 161 ml. of CO₂ per mm. of mercury decrease in mixed venous tension, corresponding to a partial dissociation slope for body stores of 2.05 ml./mm./kg. Increasing the respiratory minute volume by about 50 per cent for 1 hour produced elimination of 1.5 to 2.5 liters of CO₂ in excess of the metabolic production, which is a small part of the 100 or more liters estimated to be present in the body. The elimination of stores during a period of 1 hour did not appear to proceed at a single exponential rate, which is consistent with the presence of multiple storage sites or pools that exchange at different rates. In unsteady states, such as may exist during development of or recovery from CO₂ retention and narcosis, the arterial CO₂ tension may not adequately reflect the tissue levels.

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RESUMEN

Se estudiaron los ajustes de la reserva de dióxido de carbono en el cuerpo por medio de la hiperventilación voluntaria por una hora con ritmo constante en los sujetos. Los hombres sanos eliminan de sus reservas un término medio de 161 ml. de CO₂ por mm. de decremento de mercurio en tensión venosa mixta, correspondiendo a una disociación parcial en decremento de sererva corporal de 2.05 ml/mm./Kg. Aumentando el volumen-minuto aproximadamente el 50 por ciento por una hora produjo eliminación de 1.5 a 2.5 litros de CO₂ en excesos de la producción metabólica que es una pequeña parte de los 100 o más litros calculados como presentes en el cuerpo.

La eliminación de reservas durante el período de una hora no aparece seguir en una proporción exponente, lo que está de acuerdo con el hecho de que existen múltiples focos de reserva o lagunas de reserva que sufren el cambio con ritmos diferentes.

En las condiciones no estables tales como la que puede existir durante el desarrollo o en la recuperación de retención de CO₂ después de narcosis, la tensión arterial de CO₂ puede no reflejar adecuadamente los niveles en los tejidos.

RESUMÉ

Les auteurs ont étudié les ajustements des réserves de l'organisme en gaz carbonique pendant une hyperventilation volontaire d'une heure à taux constant sur des sujets entraînés. Les hommes en bonne santé éliminent de leurs réserves une moyenne de 161 ml. de gaz carbonique sur la tension veineuse, ce qui correspond à une dissociation partielle des réserves de 2.05 ml/m. par mm. de mercure et par kilogramme. L'augmentation du volume respiratoire minute de 50% en une heure produisit une diminution de 1.5 à 2.5 litres de gaz carbonique en excès dans le métabolisme, ce qui est une petite partie des cent et quelques litres qu'on estime présents dans le corps. L'élimination de réserves durant une période d'une heure ne sembla pas procéder d'un simple taux exponentiel compatible avec la présence de points de stockage ou de réserves qui se modifient selon la variation des taux. Dans les états instables, il est possible qu'il n'y ait pas d'examen du développement de la cessation de la rétention de gaz carbonique dans l'anesthésie, la tension artérielle de CO₂ peut ne pas refléter exactement le taux des tissus.

ZUSAMMENFASSUNG

Es wurde der Ausgleich der CO₂-Vorräte des Körpers während freiwilliger Hyperventilation von einer Stunde Dauer mit konstanter Zahl bei trainierten Personen geprüft. Gesunde Männer schieden von ihren Reserven durchschnittlich 161 ccm CO₂ pro Millimeter Quecksilberabfall aus, bei gemischter venöser Spannung; das entsprach einem teilweisen Dissoziations-Abfall für Körperreserven von 2.05 ccm/mm/kg. Eine Zunahme des Atemminutenvolumens von ungefähr 50% eine Stunde lang führte zu einer Ausscheidung von 1.5 bis 2.5 Litern von CO₂ aus dem Überschuss des intermedären Stoffwechsels; dies ist ein kleiner Teil der 100 oder mehr Liter, auf die die im Körper vorhandenen Vorräte geschätzt werden. Die Ausschüttung der Reserven während der Zeit von einer Stunde schien nicht nach einer bestimmten Exponentialzahl zu erfolgen, die auf das Vorliegen multiplier Reservoire schliessen lässt, die in verschiedenem Tempo ausgewechselt werden. Bei gestörtem Gleichgewicht wie es
vorkommt bei der Entstehung oder Rückbildung von CO₂ Retensionen und Narkose, kann es geschehen, dass die arterielle CO₂-Spannung kein adäquates Spiegelbild der Werte für das Gewebe darstellt.

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