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RESTORATION OF CARBON DIOXIDE STORES IN MAN AFTER ACUTE MECHANICALLY INDUCED HYPERVENTILATION

DISSERTATION

Presented in Partial Fulfillment of the Requirements for the Degree Doctor of Philosophy in the Graduate School of the Ohio State University

By

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********

The Ohio State University
1961

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I. HISTORICAL REVIEW

There are many interesting studies in the hyperventilation literature which separately concern themselves with plasma and urine composition changes, and cardiovascular, neuromuscular, respiratory and cerebral function changes. Descriptive changes of essential parameters within these body compartments, volumes or tissues have been made in short term and long term experiments and were adequately reviewed by E. B. Brown (1) in 1953. It is surprising, however, how few studies concern themselves in any manner with the carbon dioxide stores of the body.

Several years ago, a study was made in this laboratory which dealt with the loss of carbon dioxide stores during hyperventilation. A natural extension of this study was to inquire into the not well-documented reaccumulation of these stores with particular reference to the degree of recovery within blood and tissue compartments. Such a study might contribute to an eventual better understanding of the problems encountered by some individuals in acute hyperventilation under emergency situations.

Perhaps "carbon dioxide stores" may appear not to be an appropriate term for the quantity of this substance which is found in the body in view of the fact that it is a metabolic product of the various oxidative processes occurring in the cellular elements.
Barcroft (2) evidently did not consider carbon dioxide as a separate part of his "stores which are found in the body," even though he had earlier made the point that in order to maintain some form of constancy within the internal environment, stores were essential. And, one may ask at this point, what greater need is there than for an adequate store of carbon dioxide? Since the function and efficiency of any cellular element is affected by the reaction of its immediate surroundings, and since no greater and more rapid a change can be caused than by altering the volume of carbon dioxide within the internal environment, it seems almost axiomatic that this substance should be classed as part of the variable "stores."

Under normal circumstances, of course, a steady state is said to exist within the body. Applied to carbon dioxide, this means that the production of CO$_2$ is equal to the elimination of CO$_2$. Under these steady state conditions, a measurement of CO$_2$ elimination is a direct means of considering the metabolizing state of the tissues. Usually, this steady state with regard to CO$_2$ is said to exist when the CO$_2$ elimination under resting conditions is relatively constant, or, when, by observing a continuous automatic recording of "alveolar" CO$_2$ tension, this parameter does not change by more than 1 mm. over a 15 minute period (3). Any relatively minor changes as do occur are considered inconsequential. To permit
such constancy to exist, it is necessary to consider the carbon
dioxide of the body as part of the normal "body stores."

To determine the normal volume of this vital store in the human
body would be a difficult task indeed. However, by careful analyses
of entire carcasses of rats and cats, and by equally careful extrapo-
lation to the human body, it has been estimated that the total CO$_2$
stores approximate 120 liters, 17 liters being ascribed to soft
tissues and 100 liters being included in bone structures (3). The
volume of these stores at any time is determined by the state of
equilibrium existing between tissue structures, interstitial fluid,
blood and alveolar air. The largeness of these stores, it would
appear, is understandable when the structure of bone and existing
equilibrium states are considered. In addition, in a general sense,
large volumes (combined in this case with a high solubility) tend to
prevent extreme changes in local areas which could be disastrous
as far as efficient operation of these particular tissues is concerned.
This is not to imply that all tissues contain similar concentrations
of CO$_2$ and that they all possess equal abilities to buffer changes of
CO$_2$. Indeed, because of the differences of vascularity and solubility
existent in the various parts of the body, the opposite appears to be
the case (4).
Changes in the normal volume of the CO₂ stores occasioned by respiratory disturbances are of two types: an increase of stores is obtained by breathing higher than normal concentrations of CO₂ or by intentional hypoventilation, and a decrease of stores is obtained by breathing at a level greater than required by metabolic demands. This discussion will be limited to experiments dealing with hyperventilation.

The Induction of Hyperventilation

Although hyperventilation experiments had been carried on for a number of years prior to the turn of this century, it appears that Boothby (5) was one of the first to become interested in the loss of CO₂ stores during intentional over-breathing. Since he was primarily concerned with the recovery of CO₂ deficits, he merely measured the excess CO₂ loss over a short period of time (3 minutes) by the difference between total CO₂ output and basal CO₂ output. His results, seemingly not accurately controlled by an efficient system for maintaining a pre-set degree of hyperventilation, indicated a loss of 1.0 to 1.5 liters of CO₂ in excess of the normal metabolic production. No reliable method was as yet available for determining blood and tissue losses.

Although adequate analytical methods were soon to be developed, the study of changes in CO₂ stores induced by hyperventilation lay
dormant while investigators became more interested in CO₂ saturation and subsequent desaturation experiments. It was not until 1955 that these investigations were reopened, with dogs as experimental animals. Farhi and Rahn (3) at that time chose to consider the CO₂ stores of the body as being closely related to a single-compartment mechanical model; i.e., a single reservoir (the body stores) was filled by the CO₂ production and emptied by the alveolar ventilation. This model as applied to relatively short term experiments (45 minutes) intentionally ignored the CO₂ stores of bone, which would not change rapidly, and fat, which was poorly perfused. In a steady state condition, of course, the inflow would equal the outflow. In an unsteady state, such as occurs with increased ventilation, the level of the reservoir would decrease to a new steady state level which was estimated in their experiments to occur in approximately 45 minutes. To quote from Farhi and Rahn (3), "With such a model, following a sudden change in outflow resistance (change in alveolar ventilation) the changes in CO₂ stores will be a simple exponential of time. When these changes are shown on a semilogarithmic plot as a fraction of the final change versus time, a straight line is obtained."

This straight line indicated a half-time for the loss of CO₂ stores (defined as one-half of the readily available stores) of 4.2 minutes. The degree of hyperventilation and serially determined blood changes were not available in this instance.
Tomashefski, Carter and Lipsky (6) performed extensive hyperventilation experiments on 10 normal subjects utilizing a mechanical device to induce a moderate degree of over-ventilation. Respiratory changes as well as arterial blood acid-base balance and CO2 stores changes were observed over a 12 minute period of hyperventilation. A short term rather than a long term experiment was chosen for investigation in order to establish an experimental procedure to study this unsteady state in the light of recent evidence that hyperventilation might be the cause of some aircraft mishaps and that some form of adaptation might occur to lessen the decrease in performance capabilities which usually occurred with hyperventilation (35).

All subjects in this investigation were hyperventilated by increasing frequency 50 per cent over the control and increasing the over-all tidal volume 230 per cent over the control level. Thus, minute ventilation was initially increased approximately 3 times normal. The average frequency (15 breathes per minute) was maintained constant during hyperventilation, indicating that the mechanical respirator was the sole determinant of respiratory frequency as was
orlocally intended. However, tidal volumes tended to decrease; an

ttempt to rationalize this change was made in the following quo-

tation:

With respect to tidal volume, hence total ventilation,
absolute constancy was not obtained... Following activation
of the mechanical respirator, there was a gradual decline
in tidal volume in all cases during the first five minutes of
hyperventilation. This was not due to a decrease in the
inflation pressure provided by the respirator as this factor
is not under subjective influence. Several possible ex-
planations can be suggested. This decline in tidal volume
might represent a decreasing compliance of the lung-thorax
system... several possibilities exist. If there were an
increase in the midposition of the lung as a result of the
mechanically induced hyperventilation, then the subject
would be operating higher up on his pulmonary pressure-
volume curve where the compliance... is less than that
characteristic of the normally used mean pulmonary volume.
This would imply, then, that the initial downward trend ob-
served in the ventilation represents an adjustment of mean
lung volume which, once completed, is maintained during
the remainder of the experimental period. Were the
expiratory reserve volume measured during the experi-
mental procedure, it might be possible to confirm or deny
this argument. One might consider mechanical change in
the lung as a basis for decreased compliance; for example,
increased pulmonary blood volume. However, Fenn (has
shown that) pressure breathing actually leads to expression
of blood from the lesser circulation... Since it was observed
that the level-off point for the ventilation roughly corresponded
to the asymptote for the decrement in arterial PCO2 another
explanation (is possible). Increased muscle tone has been
described as an effect of alkalosis and hypocapnia. One can
thus postulate that an increase in tone of the intercostal
muscles resulted in a decrease in thoracic cage compliance.
Another related aspect has to do with ventilatory regulatory
function. Even though ventilatory (regulation) has been
essentially shifted from the medullary centers to a mechanical
respirator, it can be presumed that regulatory impulses emanating from these control centers are still consistent with a state of respiratory alkalosis. Thus, medullary inhibitory impulses, while unable to over-ride the influence of the mechanical respirator, could be expected to augment the tonic state of the respiratory musculature, ...{(6)

These observations on the declining tidal volume are in agreement with the published results of Fenn (7) who noted an increased chest rigidity with hyperventilation induced by intermittent positive pressure, Brown (1) who observed a decrease to normal of minute volumes during sleep regardless of the pressures applied by a respirator, and Peltier (8) who described subjects exhibiting periods of apnea during sleep while being hyperventilated in a body respirator. "All of these mechanisms are at present unexplained but they indicate that some control over respiration is still present with mechanically imposed hyperventilation." (1)

During these short term hyperventilation experiments (6), it was found that the disturbance in acid-base balance followed Gray's "respiratory pathway" (9) (Figure 1) and that the in vivo relationship between arterial pH, BHCO3 and PCO2 followed the in vitro hyperventilation pathway as described by Davenport (10) (Figure 2). Thus, metabolic compensations were not demonstrable. In addition, it was demonstrated that (a) the arterial carbon dioxide tensions as calculated by the Henderson-Hasselbalch relationship
and as directly determined by the Roughton-Scholander technique agree closely during the transient unsteady state of acute hyperventilation, (b) the arterial carbon dioxide tension is determined by the alveolar tension of this gas at any time during the transient stage, and (c), the hydrogen ion concentration of arterial blood reflects the changes occurring in alveolar carbon dioxide. These changes, previously only tacitly assumed, are plotted in Figure 3.

It was also determined (6) that the lung system contributes significantly to the total expired CO₂ volume only during the first two minutes of hyperventilation. Therefore, the blood and tissues combine to contribute the majority of the CO₂ which is expired, with the tissues eventually supplying the largest volume. This was in agreement with Vance and Fowler (11). When the average values for total CO₂ expired per breath are plotted as a function of time (Figure 4), an initial volume of 63 cc per breath rapidly drops to approximately half this value within the first 5 minutes and then proceeds to fall at a greatly reduced rate as it approaches the average basal output of CO₂ (26 cc per breath). A semilogarithmic plot of these variables (Figure 5) indicates that the final shape of this curve is determined by at least two components, a fast component (most probably CO₂ from lung air, lung tissue and the easily available blood reservoir) and a slow component (the blood
and tissue reservoirs). To include the blood reservoir in both components seems to be fitting because the initial disturbance creates a large $PCO_2$ gradient at the lung level, thus enabling the rapid release of a relatively large volume of blood $CO_2$ stores, while a diminishing gradient along with a slowly declining blood $CO_2$ content indicates that the blood stores are still being depleted and thus should be included in the slow component with regard to $CO_2$ elimination. The initial decrease in $CO_2$ elimination, as analyzed on a breath-by-breath basis, might in part be the result of the decreasing tidal volume, but undoubtedly the easily available blood reservoir of $CO_2$ also contributes substantially to this fast component of depletion.

An analysis of Farhi and Rahn's (3) original work seems to indicate (a) that the calculated half-time for $CO_2$ stores loss is based solely on the fast component as observed above and (b) that the single compartment model described by them is inadequate.

Vance and Fowler (11), utilizing voluntary hyperventilation methods, increased ventilation in the order of 50 per cent for 1 hour. Seven subjects were studied. Approximately 1.5 to 2.5 liters of $CO_2$ stores were released, the majority coming from tissue other than lung and blood.
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 (11).

Accordingly, Farhi and Rahn then suggested a multiple compartment model which "differs from the preceding model by the fact that the bulk of the body has been divided into separate compartments, all discharging in parallel into the alveolar space." (4)

The Recovery from Hyperventilation

The work of Douglas and Haldane (12) is commonly used in a classical description of the effect on respiratory activity of a short period of voluntary hyperventilation. Hyperventilation on a self-experimental basis was carried out for two minutes. It was not noted that apnea of two minutes duration followed, after which periodic breathing of the Cheyne-Stokes' type temporarily supervened, this being succeeded by the normal breathing pattern. A study of the partial pressures of alveolar gases during recovery showed that the carbon dioxide tension, reduced to 15 mm.Hg. by the hyperventilation, rose quickly in what appeared to be an exponential fashion so that after 2 minutes it was essentially normal, but oscillating above and below the control level; the
alveolar oxygen tension, forced to approximately 140 mm. Hg. pressure, rapidly diminished so that within 30 seconds it had dropped to 100 mm. Hg., and after 2 minutes of recovery it stood at 40 mm. Hg. At this point, breathing began, thus increasing the PO$_2$ so that the hypoxic stimulus to breathing was removed. The PCO$_2$, in the meantime, dropped slightly with the advent of breathing. Breathing recommenced when the PO$_2$ again fell, as a result of apnea, to stimulatory levels. After repeated periods of breathing, the pressures of these two gases were restored to normal and respiration became regular.

This resultant apnea was first described by Rosenthal (13) in 1864. According to Brown (1), he attributed this phenomenon to "over-oxygenation of the blood with a resultant removal of the stimulus for breathing." Later, Hering (14), Ewald (15), Fredericq (16), Weil (17), and Mosso (18) demonstrated that apnea was accompanied by a decrease in blood CO$_2$ (1). It was also shown (18) that apnea does not occur when over-breathing is performed with 5 to 6 per cent inspired CO$_2$ mixtures.

However, as experiments concerning hyperventilation grew in number, it became apparent that apnea was not the sole phenomenon observable during the recovery phase. Henderson (19) noted that some individuals continued to over-breathe; Boothby (5) also
described how forced breathing was followed by hyperpnea rather than apnea; Mills (20), in a study of 35 random subjects, found that about 25 per cent of the individuals regularly demonstrated continued hyperpnea after forced breathing, while another 25 per cent showed signs of post-hyperventilatory apnea at one time or another during the long series of experiments. These observations have largely been ignored, however, by authors of texts; Fink (21) more recently studied 13 subjects, of whom 7 underwent both mechanical and voluntary hyperventilation; 3 were mechanically hyperventilated only, and the remaining 3 performed forced breathing experiments only. The subjects were selected from a group of anesthesiologists who, through questioning, were found to be unaware of the effects of the experiment on respiration. Fink writes that "the most notable finding (in these) experiments was the complete absence of apnea following over-ventilation (mechanical and voluntary), despite a reduction of (end-tidal CO₂ tension) to below 15 mm. Hg. in some instances. The absence of apnea may be explained by the fact that none of the subjects knew the nature of the study or realized that a period of apnea would classically be expected." This statement was prompted by his observation that self-experimentation formed the basis for the concept of apnea following hyperventilation in conscious man. Fink, as did Mills, suggested that cerebral
activity played an important part in the maintenance of a hyperpneic state after a short period of hyperventilation. Others (5, 22) have suggested that it was more likely the central vasoconstriction resulting in cerebral hypoxia which provided the explanation for hyperpneic breathing during recovery from hyperventilation (20).

It is thus apparent that recovery from hyperventilation may proceed in the direction of apnea of hyperpnea. However, the mechanism for the continuing hyperpnea is not clearly defined, and apnea as a common result of over-breathing unanesthetized man has been attacked. Mills (20) defines as a third possibility an intermediate group which shows "some continued breathing, though not very vigorously." When each of these pathways of recovery from hyperventilation is examined with respect to the rate of recovery of the CO₂ stores which were lost during hyperventilation, it is natural to expect that the apneic individual would show the greatest initial rate of retention of stores, and that the intermediate and hyperpneic individuals would show slower rates of retention. It is also to be expected that the time for complete restoration of lost CO₂ stores would be considerably longer than the time period over which the excess CO₂ was lost (4). However, Boothby (5), an individual who responded in an hyperpneic manner
to a short period of acute hyperventilation, concluded after self-
experimentation that "in some individuals the loss of CO₂ in
consequence of forced breathing is made up within a few minutes,
though not so rapidly as when apnea occurs."

One of the purposes of this present investigation is to examine
this statement in greater detail with experiments designed to
determine the accumulation of CO₂ in the body stores after acute,
mechanically induced hyperventilation.

The Whole Body and Tissue CO₂
Dissociation Slopes

"The curve which relates the CO₂ concentration in the blood
to that in the air is called the CO₂ absorption curve of the blood
and describes the behavior of the blood towards CO₂" (23). These
absorption curves, or dissociation curves, are in the realm of
common knowledge. It seems not too difficult a task to determine
the volume of CO₂ capable of being carried at a particular PCO₂;
and, having determined a number of these points and having joined
these points, it is again not too difficult a task to imagine what
happens when PCO₂ changes. In a general sense, the arterial
blood level of CO₂ content is determined tonometrically by the
lung level of CO₂ partial pressure. And since the CO₂ pressure
gradient is in the direction of the lungs, the tissue CO₂ content is determined directly by arterial blood partial pressure and hence the partial pressure of carbon dioxide in the lung.

In a normally perfused area, if the metabolic production of CO₂ increases, then the partial pressure of the gas in the area of the cells and interstitial fluids increases; hence, the pressure head toward the perfusate increases and this highly soluble metabolic end-product seeks and finds its equilibrium with the partial pressure in the circulating medium, perhaps with a slight damming effect occurring to increase the volume of CO₂ in the tissues themselves. In a well-perfused area, the CO₂ would be dissipated completely; in a non-perfused area, the CO₂ would build up. If the partial pressure of CO₂ in the arterial blood is increased, the pressure head at the tissue level is decreased and, at the same metabolic rate, tissue CO₂ increases. If in conjunction with this increase there exists a large perfusion area, then it would seem that a new equilibrium would be reached more rapidly than if the area under consideration were poorly perfused. If the partial pressure of CO₂ in the arterial blood is decreased, the pressure head at the tissue level is increased and, at the same metabolic rate, tissue CO₂ decreases. If in conjunction with this decrease there exists a large perfusion area, then it would seem that the new equilibrium
would be reached more rapidly than if the area under consideration were poorly perfused. Therefore, perfusion, metabolic rate, as well as buffering capacity of the tissues are all concerned with the length of time it takes for equilibrium conditions to appear after the steady state of carbon dioxide dynamics is altered.

The carbon dioxide carrying capacity of blood is determined primarily by the unique combination of hemoglobin and carbonic anhydrase. The carbon dioxide loading or unloading capacity of a section of tissue, lacking hemoglobin and carbonic anhydrase, would not necessarily be required to differ from that of blood. Since, under certain conditions, such as decreased perfusion or increased metabolic rate in a localized area, the tissues would be expected to retain by buffering larger than normal quantities of carbon dioxide, it might be expected that the efficiency of the tissue buffering system would be greater than that of the blood. Or, barring a difference in efficiency, the large volume of the tissue and tissue spaces combined with high CO$_2$ solubility might be sufficient to counteract disastrous changes in levels of CO$_2$.

Shaw (24) determined that in 30 per cent of his experiments on cats, the buffering capacity of tissue fluids was equal to or greater than that of the blood, results being expressed as cc of CO$_2$ per kilogram of body or blood weight per mm. carbon dioxide tension
change. Shaw's experiments were based on equilibrium conditions existing after forced breathing of 11 per cent CO₂ for approximately 100 minutes. Whether or not he had attained tissue equilibrium is questionable since it has been suggested that it may take several hours for equilibrium of CO₂ stores to occur (4).

The carbon dioxide absorption or dissociation curve for tissues, then, is a measure of the carrying capacity or storage capacity of the tissues at the particular partial pressure of carbon dioxide prevalent in the environment of the tissues. It has been studied by (a) the saturation method (25), in which case increased concentrations of carbon dioxide are breathed; (b) the saturation method followed by desaturation (25), in which case having attained a level of carbon dioxide saturation the loss of carbon dioxide becomes the measurable quantity; (c) the elimination method (4), in which case hyperventilation is employed to cause a decrease in CO₂ stores; and (d) elimination followed by saturation (26), which is self-evident. In rats, use of these methods has been followed by sacrificing the animals and analyzing the separate tissues for CO₂ (27). In dogs and man (3, 11), methods such as these have been utilized in conjunction with methods for estimating retention or excess elimination of CO₂ to determine whole body and blood retention rates, and whole body CO₂ dissociation slopes. In all of these studies, it is assumed that the
slope of the dissociation curve for tissue is linear between 30 and 80 mm. CO₂ tension, just as the slope of the CO₂ dissociation curve of blood is near-linear over this range of tensions. It has been declared by various means that equilibrium conditions exist after a saturation or elimination has continued for time periods ranging from 1 minute to 100 minutes. Farhi and Rahn (4) have recently stated that "there seems to be a gross relationship between duration of the experiment and slope of the dissociation curve of the body...Vance and Fowler concluded that their results demonstrate the different rates of exchange of alveolar gas, blood, and tissues, and postulate that there are probably multiple sites or pools with various rates of exchange."

Farhi and Rahn (4) have also recently described the effect of increased and decreased muscle perfusion, to which tissue they ascribe the distinction of being the largest buffering pool of the body, on the calculated tissue CO₂ dissociation slope. Utilizing an analogue computer, they arrived at the conclusion that "the crucial point appears to be the effect of muscle perfusion on the functional storage capacity of the body...Even under the most favorable conditions the body CO₂ stores must require several hours to adjust. With few exceptions all the data in the literature must be accepted as representing incomplete experiments in which
time limitations did not allow full equilibration of the body stores".

Table 16, modified from Farhi and Rahn (4), illustrates the slopes of the CO₂ dissociation curves as they are found in the literature.

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It is obvious, after reviewing the literature, that only a paucity of data is available regarding the changes in carbon dioxide stores following an acute bout of hyperventilation. A more complete understanding of the changing stores during recovery appears to be essential in order to institute proper methods to promote a more rapid recovery from the effects of acute hyperventilation. Such an understanding would also pave the way toward further studies involving adaptation responses (35) to repeated hyperventilation.

Since several types of post-hyperventilation respiratory responses have been described, it should be possible to describe these recovery responses in terms of carbon dioxide retention.

The purpose of this paper, therefore, is to clarify the type of recovery encountered in untrained individuals after acute, mechanically induced hyperventilation.
II. EXPERIMENTAL APPROACH

Materials

The experimental setup consisted of a directional breathing valve (Figure 6), a rapid infrared Beckman-Spinco CO₂ analyzer, a Texas Instruments Rectilinear recorder, a Bennett intermittent positive pressure breathing apparatus, a 120 liter Tissot Gasometer, a National Instruments Vol-o-Flow flow meter, and a Statham strain gauge transducer, a Sanborn amplifier and Sanborn Poly-Viso recorder (Figure 7). The plastic directional breathing valve (Figures 6 and 8) was especially constructed so as to allow expiratory flow in either one of two directions. A flap valve was used in one direction (C of Figure 6) for normal and recovery expirations, while a pressure-loaded valve was used in another direction (D of Figure 6) during the hyperventilation. This two-way expiration system offered only a minimum of resistance during the recovery period but permitted use of a pressure-loaded valve during positive pressure ventilation. A three-way valve in the inspiratory line allowed the subject to breathe either room air at ambient pressure or compressed air as directed through the intermittent positive pressure breathing device. A mouthpiece and noseclip, rather than a mask, was used so that dead space could be minimized. The two
expiratory lines led to a common expiration pathway which in turn
directed air to the flow meter and then to the Tissot Gasometer.
This system permitted (a) the collection in the gasometer of normal
and recovery expired air, as well as (b) the continuous monitoring
of individual tidal volumes during the normal, hyperventilation
and recovery periods.

The flow meter was calibrated before and after each experiment
with a Brooks Rotameter which had previously been calibrated by
monitoring flow rates with the gasometer. Differential pressures
were monitored with a Statham strain gauge transducer and instant-
taneous flow velocities were recorded on the Sanborn Poly-Viso
recorder. Calibrations showed a linear relationship between flow
rate and recorded deflections in all ranges of amplification.
Ordinarily, the highest possible range of amplification was used to
record expired air flow velocities.

The detector cell of the CO₂ analyzer was firmly mounted in
such a position so that it was located within a few inches of the
subject's face, thus allowing a 6 inch length of flexible rubber
tubing (3 mm. internal diameter) to easily reach from the detecting
unit to the mouthpiece, the connection at the latter being by way of
a No. 15 hypodermic needle with a flattened point which was
inserted into the mouthpiece so that its tip extended only to the
center of the air stream. The amplifying unit and Texas recorder were mounted on a portable cart to permit flexibility of operation, and calibrating gases were either mounted on the cart or were near enough to the operational area to permit their use. Calibration curves obtained before and after each experiment, showed no significant alterations over the experimental period. A small DeVilbis pump, attached to a one liter bottle, pulled air continuously through a microcatheter cell (volume 0.1cc) mounted in the detecting unit. The reservoir bottle was placed in the circuit to eliminate the pulsating type of flow which was characteristic of the pump. Flow through the detector unit was modified by means of a screw clamp mounted on the tubing connection between the reservoir bottle and the CO₂ detector unit. During preliminary experiments it was determined that (a) volume flow through the detector unit was constant during any one experiment, (b) volume flow was variable on different experimental days due to variations in the screw clamp setting (thus necessitating volume flow calibrations with each experiment), (c) minor changes in flow rates through the detector cell did not measurably distort the readings obtained on a known concentration of CO₂ passing through the microcatheter cell. The output of the amplifying unit was continuously recorded on
a Texas Instruments Rectilinear recorder operating at the fastest chart speed available, 12 inches per minute. Thus a continuous record of CO₂ concentrations at the mouth was available for analysis.

Methods

Fourteen normal, healthy males (medical students, graduate students and project personnel) were used as experimental subjects. All were indoctrinated in the use of intermittent positive pressure breathing on the day prior to the experiment so that (a) apprehension could be alleviated as much as possible, (b) a smooth transition could be made from the normal breathing pattern to the hyperventilation pattern, and (c) the hyperventilation pattern, consisting of an increase in frequency of 50 per cent and an increase in tidal volume of 200-300 per cent could be set in the Bennett IPPB. Otherwise, the subjects were not trained.

On the day of the experiment, an indwelling arterial needle was placed in the brachial artery, after which the subject rested for 30 minutes in a recumbent position. The Tissot Gasometer was rinsed adequately with the subject's resting expired air while he breathed room air normally and a steady state was established. During a three minute period immediately following the flushing procedure, the total expired air was collected in the gasometer while carbon dioxide concentrations at the mouth and expired volumes were
continuously recorded on a breath-by-breath basis by means of the carbon dioxide analyzer and the pneumotachograph systems. An arterial blood sample was drawn during the midminute with the usual precautions observed to prevent glycolysis, clotting, and trapping of air bubbles (28). Gas samples for analysis were withdrawn from the gasometer in mercury tonometers and analyzed by the Scholander micro-gas technique (29). The remaining volume was analyzed with the Beckman CO\textsubscript{2} and O\textsubscript{2} analyzers. The gasometer dead space remained filled with mixed expired air of known gaseous concentrations.

Using the respirator settings obtained the previous day, the subject was then mechanically hyperventilated with compressed air for 12 minutes. Expired volumes and carbon dioxide concentrations were recorded on a breath-by-breath basis. Total mixed expired air was not collected. During the final minute of hyperventilation an arterial blood sample was drawn after which the mechanical ventilation was stopped and the subject was switched to breathing room air during the recovery process. At this time mixed expired air was collected in the gasometer. Tidal volumes and CO\textsubscript{2} concentrations were monitored continuously, and arterial blood samples were collected during the 1st, 2nd, 5th and 11th minutes of recovery. Observations were stopped after 12 minutes. In those cases where
large volumes were expired, a Douglas bag was used in addition to the gasometer for collecting all of the recovery expired air. In all cases conducting lines were adequately washed at the end of the experiment with known volumes of oxygen so as to recover all of the expired air. Mixing was accomplished by transferring the total expired volume into an evacuated Douglas bag. Samples were then obtained for Scholander analysis and a portion of the remaining air was passed through the CO$_2$ and O$_2$ analyzers. Appropriate corrections for the added oxygen were made.

**Measurements**

**Arterial blood.**—Arterial blood analyses were performed by techniques fully described elsewhere (28) from which the following quotation is taken:

pH of whole blood was determined at 37°C. with anaerobic precautions, within 2 minutes after collection, on a Cambridge Research Model pH meter. The vertical glass electrode was filled with mercury; the cork, which was in place at the tip of the 20 gauge needle attached to the syringe containing the arterial specimen to be analyzed, was removed and the first 3-4 drops of blood were allowed to be wasted. The needle was then placed in the lumen of a 1 ml. pipette with the rubbered tip of the pipette pressed firmly against a second cork, which was pierced by the needle and which rested at the base of the needle. Blood was then allowed to enter the pipette with the very minimum of surface exposed to room air. The blood was then quickly transferred to the mercury-filled glass electrode, drawn down, and readings made after an equilibration period of approximately 1 minute.
The partial pressure of CO\textsubscript{2} and O\textsubscript{2} in arterial blood were determined by the Roughton-Scholander technique as modified. Anaerobic precautions in the transference of samples from storage syringe to the R-S syringe were observed which were similar to those employed in the pH study; also, precautions were taken during the admission and evacuation of absorbers to prevent contact with room air. The air sample admitted to the syringe was composed of 6.94% CO\textsubscript{2}, 11.63% O\textsubscript{2} and 81.43% N\textsubscript{2} as determined by the micro-Scholander technique, and thus was close to that of alveolar air, therefore allowing only a minimum of gas transfer to take place between dissolved gases of the blood and the air bubble. Equilibrations and readings of bubble length were made at 37°C, in a constant temperature water bath using a telescopic measuring system.

Whole blood CO\textsubscript{2} content, O\textsubscript{2} content and O\textsubscript{2} capacity were determined as per the methods outlined by Van Slyke.

Hematocrits were determined by the micro-centrifuge technique using a capillary centrifuge and a capillary meniscus reader. The general procedure of mixing the sample and wasting the first 2-4 drops were followed. The blood sample was then allowed to enter the capillary tube, from a freely hanging drop suspended from the tip of the needle of the blood storage syringe, strictly by capillary action. With one end of each tube flame sealed, at least duplicate samples were centrifuged at approximately 15,500 rpm for 5 minutes.

The blood samples were iced, usually immediately after the pH determination, and removed from the ice bath only for obtaining samples for other determinations. All samples were analyzed at least in duplicate with the accuracy required noted as follows: pH, 0.01 unit; PaCO\textsubscript{2}, 4.0 mm.Hg.; hematocrit, 0.1%; CO\textsubscript{2} content, O\textsubscript{2} content, O\textsubscript{2} capacity, 0.20 vol. %. All blood samples were agitated for 1-2 minutes before aliquots were removed for analysis, thus assuring a uniform distribution of cells. The first few drops of blood from the sample syringe were always wasted to insure that a thoroughly mixed sample was obtained for use. The total analysis time for arterial samples usually approximated 3 hours. (28)
Mixed expired air.-- Analysis of the mixed expired air collected in the gasometer was performed by the Scholander micro-gas method (29). Transfers of samples from mercury tonometers were effected by means of a 0.5 cc. pipette with a mercury drop acting as a floating seal. The results of a pair of tonometers which represent the same mixed sample were required to agree within 0.04 per cent. The average of the two samples was then used for comparison with the results obtained by means of the Beckman CO₂ and O₂ analyzers. CO₂ concentrations determined by the two methods agreed within 0.1 per cent. The oxygen concentrations as determined by these two methods varied to a large extent. Therefore, since the Scholander method for measuring oxygen concentrations appeared to be most reliable, the values as determined by that method were utilized in the results of these experiments, along with the Beckman-Spinco carbon dioxide analyzer values of CO₂ concentrations, which proved to be not significantly different from the micro-gas carbon dioxide determinations.

Total mixed expired gas volumes were measured to the nearest 0.1 mm. and then expressed as a volume by application of the Tissot factor (132.2 cc./mm.). Temperature of the expired air and pressure being known, volumes could then be converted to STPD or BTPS conditions.
**Breath-by-breath volumes and CO₂ concentrations.**

Instantaneous expiratory flow velocities were recorded during the entire normal, hyperventilation and recovery periods at a paper speed of 10 mm. per second. A mean flow rate for each tidal volume was obtained by dividing the planimetered area under each curve by the base length. This was then converted into a volume by multiplying by the duration of the expiration. Since the areas and base lengths to be measured were well-demarcated, no particular difficulties were encountered in determining expired volumes at normal or above normal velocities of flow. The only appreciable errors encountered in this method other than those concerned with measurement, were errors resulting from extremely low velocities of flow which fail to be differentiated by the differential pressure transducer. The latter might result toward the end of any expiration or during the early stages of recovery from hyperventilation when air flow, if present, might have been minimal.

The determination of mean carbon dioxide concentration eliminated with each breath was carried out in a similar manner in that a planimetered area was divided by the base-line length over which expiration occurred. The mean deflection thus obtained was then converted to a mean concentration of CO₂. The area of the CO₂ elimination curve to be included as representative of the
truly expired CO$_2$ was taken as the time during which a flow velocity was actually recorded. The transfer of the duration of each tidal volume was accomplished by measuring time intervals from a point or points common to both volume and CO$_2$ concentration curves.

**Calculations**

Standard methods which had been discussed at length previously (28) were employed to calculate (a) partial pressures of CO$_2$ and O$_2$ from per cent concentration, (b) partial pressure of CO$_2$ from the observed values of pH and total blood carbon dioxide content, (c) bicarbonate content of serum. The basal as well as the total recovery CO$_2$ elimination was calculated from mixed expired gas concentrations and volumes. These were corrected for inspired carbon dioxide and pump withdrawal. Oxygen utilization was determined in a similar manner. Carbon dioxide elimination was also determined by integration of the pneumotachogram and carbon dioxide concentration curves.

This latter procedure was employed for the entire normal and recovery periods but only during the first, second, third, sixth and final minutes of hyperventilation. Total carbon dioxide elimination during hyperventilation was estimated by assuming a linear drop in CO$_2$ elimination per minute between the 3rd and 6th minutes, and
the 6th and final minute of hyperventilation. The excess CO₂ eliminated was determined by subtracting the integrated basal CO₂ elimination from the integrated total CO₂ elimination as measured during the hyperventilation period. Excess CO₂ elimination as measured by these methods compared favorably with the average value obtained strictly by spirometric analyses. The loss of tissue CO₂ stores was determined by subtracting the lung and blood CO₂ loss from the total excess CO₂ loss. The loss of carbon dioxide from lung air was calculated from an assumed FRC of 2200 cc. (30) and the measured PA₃CO₂ difference over the entire hyperventilation period. The blood CO₂ loss was calculated from an assumed blood volume of 80 cc. per kilogram of body weight (11, 31) and the change of arterial carbon dioxide content, assuming that this difference is similar to that occurring in venous blood over the entire hyperventilation period (6). Similar considerations were used to partition the retention of CO₂ into lung, blood and tissue compartments during recovery from hyperventilation.
III. RESULTS

The measurement data for blood and expired air analyses are reported for individual experiments in Tables 1-A to 1-N. Oxygen capacities were measured in the first four runs and were used in the calculation of the partial pressures of arterial carbon dioxide, but since the hematocrit values gave approximately the same results the determination of oxygen capacity was discontinued for the remainder of the experiments and the hematocrit was used instead.

During hyperventilation, respiratory frequency (Table 2) showed an increase of approximately five breaths per minute while the tidal volume showed an initial average increase of 2.6 times the control volume (Table 4). The frequency remained constant but the tidal volumes decreased slowly so that in the final minute of hyperventilation the tidal volume was only twice the average normal tidal volume. The average increase in tidal volume over the entire hyperventilation period was approximately 220 per cent. Thus, the ventilation was increased in a manner similar to that reported previously in a detailed study of the induction of hyperventilation (6). One run was terminated after 9 minutes of hyperventilation because of the development of tetany.
Average respiratory frequencies during recovery indicate a decline below the control level during the first four minutes followed by a gradual increase toward control (Table 3). Average tidal volumes show a sudden drop toward the control level with the termination of the mechanical ventilation but still the mean tidal volume remained slightly above the control for the first four minutes of recovery and then dropped below control levels for the remainder of the recovery period (Table 5).

The comparison of carbon dioxide elimination determined by direct mixed expired air analysis and by summing the individually integrated curves shows no significant difference in the results obtained by the two methods in either the normal or the recovery periods. As indicated in Table 6, the average difference between these two methods was 12 cc. per minute and 10 cc. per minute during normal and recovery periods, respectively. The summed carbon dioxide elimination on a breath-by-breath basis was nearly always higher than that determined by usual methods and in a few instances (specifically refer to subject F.M.) there was found to be an appreciable difference. These results are possible in that sampling was done at the mouth, dead space of the breathing valve (75 cc.) was high with respect to the low tidal volume encountered (366 cc. control tidal volume for subject F.M.), and the very end
of each tidal volume was sampled automatically by the carbon dioxide analyzer. This trapped volume of end-tidal air with its relatively high concentration of CO$_2$ never found its way into the gasometer. Therefore, these comparative results are as expected.

Table 6 also shows that there was a significant decrease, as expected, of 69 cc. per minute in carbon dioxide elimination when integrated control and recovery results are compared. As determined from the mixed expired air, oxygen utilization rose slightly in the recovery period. (This resulted in an apparent rise in CO$_2$ production during recovery, as calculated from the recovery oxygen utilization and the metabolic rate which was assumed to be unchanged from the control.)

Table 7 indicates that the actual respiratory exchange ratio dropped from an average of 0.81 in the control period to 0.45 in the recovery period. This resulted from a diminished CO$_2$ output. In addition, this table shows a significant rise over the control in the respiratory frequency during hyperventilation, the frequency remaining at a constant level during hyperventilation. There was a slight but insignificant drop in frequency during the initial period of recovery when compared to control rates but the control and final recovery rates are essentially the same. Since the tidal volume measurements in the recovery period may be somewhat deceptive in that only one
or two large breaths may have been taken in a given period, the minute ventilation was determined and tabulated in Tables 8-A and 8-B for the hyperventilation and recovery periods, respectively.

End-tidal carbon dioxide tensions and the various arterial blood measurements pertinent to graphic portrayal are presented in Tables 9-A to 9-F where they have been divided into the various collection periods. There is no significant difference in any period between the arterial carbon dioxide tensions as measured directly by the Riley technique and that calculated by the Henderson-Hasselbalch nomogram. However, the difference between peak end-tidal and calculated arterial carbon dioxide tensions are significant in all periods, mean differences being in the range of 4-6 mm. of mercury. This is 1-3 mm. greater than that determined previously in this laboratory and was probably due to the combined factors of a high pump withdrawal rate and a large breathing valve dead space, both of which tend to lower the peak CO₂ tension when tidal volumes are essentially adequate to wash out the dead space of the valve. During the transient stages of considerably reduced tidal volumes which occurred during the recovery period, inadequate valve washout and reduced flow rates tended to obscure the peaks of the individual CO₂ concentration curves and thus made for considerable error in estimating end-tidal CO₂ tension.
The total accumulative CO₂ elimination during the recovery period, the accumulative retention of CO₂ by the body stores, and the per cent recovery of carbon dioxide stores are tabulated in Tables 10 through 12. The loss and gain of carbon dioxide stores, partitioned into blood and tissue reservoirs, is indicated in Tables 13 and 14. Upon close examination of these data, one can see that differences are apparent which tend to place individual experimental results into various groups. This is borne out also by close observation of blood tension analyses as well as respiratory patterns during recovery.
IV. DISCUSSION

The Pathway of Recovery

The first 12 minutes of recovery from the alkalosis of acute, mechanically induced hyperventilation appears to follow the respiratory pathway as depicted by Gray (9) (Figure 9) and Davenport (10) (Figures 10 and 11). Gray's theoretically derived relationship between $[H^+]$ and $P_{ACO_2}$, which shows the pure respiratory pathway of arterial blood changes over a wide range of carbon dioxide pressures, is plotted along with the individual results obtained in these experiments. The $[H^+]$ increases as the carbon dioxide tension increases in a near-linear fashion, following the theoretical line in a reverse direction from that demonstrated previously to occur (6) in a short period of hyperventilation (Figure 1). In addition, the bicarbonate-pH-$P_{ACO_2}$ relationship illustrated in Figure 11 demonstrates the decreasing pH, and increasing bicarbonate and CO$_2$ tension as recovery proceeds. This in vivo recovery path closely follows the in vitro path determined by Henderson (32); however, the mean path lies below the in vitro relationship of Henderson (Figure 11). This is in accordance with the mean pathway for the development of alkalosis (Figure 2) and is the result of different bicarbonate capacities (23).
An Interpretation of Mean CO₂ Retention Data

By plotting the accumulative total CO₂ elimination during recovery as a function of time (Figure 12) and assuming that the mean basic CO₂ production (as determined by integration procedures) remains unchanged during recovery, it becomes apparent that several patterns of CO₂ retention exist. This is in accord with the experiments of Haldane (12) who demonstrated that not all individuals exhibited apnea after short periods of hyperventilation but that indeed some continued to breathe in an hypopneic manner. A graphical representation of mean formation, elimination and retention of CO₂ would therefore tend to be misleading (Figure 13); it would tend to hide evident differences in recovery patterns. From these mean data resulting from short term experiments, it would appear (a) that retention of CO₂ by the body stores occurs rapidly; (b) that, of the mean excess CO₂ stores which are lost during hyperventilation (2630 cc.), one-third of the stores have been reaccumulated within twelve minutes; and (c) that recovery of normal CO₂ stores might be accomplished in 36 to 40 minutes if retention of CO₂ proceeded linearly. A slightly more thorough examination of these data would show, however, that while the accumulated metabolic production of CO₂ increases in a linear manner in keeping with its assumed constancy of production, the
rate of CO$_2$ elimination increases as recovery progresses so that elimination gradually approaches production. When this becomes the case, the two curves (CO$_2$ production and elimination) would become parallel and at that point CO$_2$ retention would cease. It appears that the production and elimination curves are just beginning to approach the parallel situation, and thus the accumulated retention of CO$_2$, which is represented at any time by the difference between these curves, is beginning to plateau. Short term recovery experiments, therefore, might be misleading as far as duration of full recovery is concerned if one merely extrapolated the accumulative retention curve to the point where the lost CO$_2$ stores would be completely refilled.

The Separation of Retention Data into Patterns

Figure 14 illustrates the accumulated sums of CO$_2$ retention for each subject. An attempt has been made to describe three patterns by which recovery of CO$_2$ stores proceeds: Group I (subjects M. C., L. P., J. P., G. H.) shows no retention of CO$_2$ for at least three minutes. At that time, two subjects (L. P. and G. H.) begin to retain CO$_2$ in the body stores. One subject (J. P.) does not begin to show retention of CO$_2$ until approximately 5 minutes have passed, while M. C. does not show any retention through 8 minutes of recovery.
This group of subjects undoubtedly should be classed as individuals who show continued hyperpnea after hyperventilation; Group II (J.S., D.S., D.C., F.M.) shows as flattened S-shaped patterns, a brief period of increased ventilation resulting in little retention followed by a more rapid accumulation of stores and then a slight decrease in accumulation. These individuals might correspond to Mills' intermediate group, neither apneic nor extremely hyperpneic; Group III (E.C., J.T., A.H., K.K., G.L., J.M.) shows an initial sharp rise in CO₂ retention followed by a gradual decrease in accumulation due to a decrease in the rate of retention. This group represents individuals who exhibit apnea or underventilation as an immediate response to hyperventilation. These recovery patterns can be better demarcated when the per cent of the lost CO₂ stores which are restored is plotted as a function of recovery time (Figure 15). It should be stated at this time, however, that individuality with regard to a CO₂ retention pattern is quite apparent.

The three recovery patterns as represented in this paper were chosen by a close consideration of the data in this figure along with an examination of the respiratory data of each individual as he was placed in a particular group. The four subjects comprising Group I posed no separation problems. These individuals all showed a relatively long hyperpneic period and a relatively long delay before
CO$_2$ retention (if it did occur within the experimental time) began. The separation of Groups II and III proved to be more difficult but was finally resolved by placing in Group II those individuals who showed a period of continued hyperpnea longer than 1 minute. Some of the remaining subjects who were placed in Group III did not show signs of continued hyperpneic breathing for several breaths following the onset of recovery but this changes relatively quickly to a pattern which suggested underventilation.

Figure 22 shows that utilization of this highly arbitrary method for obtaining three possible pathways for restoration of lost CO$_2$ stores is sufficiently adequate to produce no overlapping of group standard deviations for the first two-thirds of the recovery period. It is true that with more experimental data, these groupings might not be very apparent. This figure also indicates that a wide degree of separation exists between each group with regard to mean per cent CO$_2$ stores reaccumulated. After 12 minutes of recovery, Group III shows that 51 per cent of the CO$_2$ stores lost during hyperventilation has been restored, while Groups I and II indicate 13 per cent and 30 per cent of the lost stores have been regained, respectively, by the whole body.

Assuming a mean blood volume of 80 cc. per kilogram of body weight (31), and arterial and venous blood CO$_2$ content differences
which are similar both at the end of hyperventilation and at the end of the total recovery period, the CO$_2$ stores regained by the blood and tissues can be estimated as illustrated in the following considerations of the separate groupings.

**Group III (Apnea or Immediate Hypoventilation)**

Of the 51 per cent of the lost CO$_2$ stores regained by this group, roughly one-half is retained by the blood and one-half by the tissue reservoirs (Table 15). The volume of CO$_2$ regained by the blood reservoir in the 12 minute recovery period represents 94 per cent of the total blood CO$_2$ content lost during the hyperventilation period. Thus, the blood CO$_2$ stores which are so readily lost during hyperventilation are rather easily reaccumulated during an apneic or hypoventilatory recovery period. Since the tissues contribute most of the excess CO$_2$ lost during hyperventilation, they show a large deficit after 12 minutes of recovery. The individual and mean CO$_2$ accumulation of Figures 15 and 22, respectively, indicate that the initial rapid rise, representing both blood and tissue CO$_2$ storage, is gradually leveling off. This plateauing effect, which just begins to be apparent at the termination of the experiment, should represent principally the retention of CO$_2$ by the tissue stores. It infers that the rate of CO$_2$ retention by the whole body is diminishing. Extrapolation of the CO$_2$ accumulation curve for this group indicates a
continued rise at the rate of one per cent per minute. It would therefore take at least 60 minutes for this group to return to the normal steady state condition. Undoubtedly it would take a longer time since the accumulation curve would tend to become flatter as the tissue stores approach their normal level. The comparison of Figures 16, 17 and 18, each of which shows the concurrent changes of pH, \( \text{PaCO}_2 \), \( \text{PaO}_2 \) and per cent stores accumulated, gives some indication of the differences existing in the blood reservoir CO\(_2\) accumulation rates in the three groups. Group III shows some sign of a plateau being reached with regard to arterial CO\(_2\) tension as early as 5 minutes after halting mechanical hyperventilation.

Further examination of Figure 18 shows that the pH return rapidly toward normal while the arterial oxygen tension drops precipitously to a low level of 63 mm. Hg. These changes are the result of an initial fall in ventilation to levels well below the control due to a decrease in both frequency and tidal volume (Figure 21). Minute ventilation, frequency and tidal volume all proceed to approach control levels from below, with the tidal volume showing marked variability.

Since the percentage distribution between blood and tissue is approximately equal, it is evident that the resistance of the tissue reservoirs to filling is such as to allow homogeneity to exist in
the early portion of the recovery period between CO$_2$ retention in blood and tissue. As elimination of CO$_2$ increases and retention decreases while production of CO$_2$ remains approximately constant, a point is reached whereby the blood CO$_2$ stores have gained almost all that was lost during hyperventilation; from that point on, retention is principally a balance between elimination and tissue storage, and thus represents the increase of CO$_2$ in the tissue reservoir, the limiting factors being metabolic production, elimination and tissue perfusion (4).

It is to be expected that with an increase of CO$_2$ tension in arterial blood resulting from an increased resistance for CO$_2$ elimination at the lung level (decreased ventilation), the tissues would retain some portion of the CO$_2$ produced by its metabolizing cells, less CO$_2$ passing into the capillary blood along the diminished gradient; i.e., the resistance increases at the tissue level, too. But with the creation of this damming effect, tissue CO$_2$ tension rises and temporarily lowers the resistance for passage of CO$_2$ from tissues to venous capillary blood, allowing venous CO$_2$ tension to increase. The apneic individual, therefore, would show a relatively rapid rise in blood stores and hence tissue stores; with the commencement of irregular breathing, continually new gradients are established at the tissue level resulting in alternating greater and lesser amounts of CO$_2$.
being dumped into the blood; and with elimination of CO₂ now to be considered, the rate of increase of blood CO₂ stores diminished. This accompanies a diminution in the rate of increase in tissue stores. With a more uniform ventilation, the rate of increase of blood CO₂ stores tends to diminish more and more as it approaches its normal content.

However, not until new gradients (high enough to counteract the pulmonary barrier) are erected can CO₂ flow to the outside be restored. The elevation of these internal gradients results in CO₂ retention. Once the ventilation drive is restored, at least partially by the forcing function of the PCO₂, the pulmonary barrier is allowed to drop (an increase in ventilation) and again the CO₂ tensions are altered. Thus alveolar as well as arterial and venous tensions probably oscillate over several millimeters of mercury pressure while the early stage of recovery from hyperventilation is in progress. With apnea, of course, the oxygen tension of arterial blood decreases rapidly toward normal tissue levels, thus becoming a stimulant to respiration through the carotid body mechanism.

The role of chemoreceptors in post-hyperventilation apnea has been investigated by Heymans and Jacob (33). When the carotid and aortic bodies were suppressed, they found a marked prolongation of the apnea, and pointed out that animals deprived of their peripheral
chemoreceptors may die in apnea. It is evident that these data indicate that oxygen tension, as well as CO₂ tension and hence hydrogen ion concentration, plays a part in determining the duration of post-hyperventilation apnea.

**Group II (Delayed Hypoventilation)**

This group, regaining approximately 30 per cent of its lost CO₂ stores, similarly shows an approximate 50 per cent distribution between blood and tissue, the blood reservoir showing less of a percentage gain (76 per cent of the lost CO₂ content) than it does in Group III. Figure 17 indicates that in comparison to Groups I and III, this is an intermediary group with regards to the changes of pH, PaCO₂, PaO₂ and per cent CO₂ retention. The minute ventilation in Group II (Figure 20) shows the same initial sharp drop as the other groups, but it only continues falling to slightly below control levels and then approaches the control ventilation. This results from a gradually declining frequency toward the control level, and a rapid decline in tidal volume to below control level, both of which then proceed to approach their respective control values.

Since the total CO₂ loss from the body stores is somewhat greater, and since the whole body CO₂ stores regained is about the same as in Group III, the total per cent regained by the whole body, and hence
its distribution between blood and tissue, is lower than in Group III. The individuals comprising this group all show the same pattern of recovery in that a short period of hyperpnea follows the end of mechanical hyperventilation, and that this is followed by a tendency toward hypoventilation. The degree of hypoventilation is variable and hence at the end of the experimental recovery period a variety of accumulated sums of CO\textsubscript{2} retention results (Figure 14). This accounts for the similarity between volumes of CO\textsubscript{2} retained in Groups II and III at the end of the recovery period.

**Group I (Lengthy Hyperpnea)**

This group shows a mean total retention of approximately 13 per cent of the lost CO\textsubscript{2} stores with a distribution of 10 percent and 0 per cent between blood and tissue reservoirs, the blood reservoir regaining 49 per cent of the CO\textsubscript{2} content which was lost during hyperventilation. A near-linear rise in mean total CO\textsubscript{2} retention is shown to occur after 4 minutes of recovery (Figure 16) during which a minimum of tissue CO\textsubscript{2} stores are replenished. Examination of the PA\textsubscript{CO\textsubscript{2}} data and the CO\textsubscript{2} retention data (Figure 16) indicates that the arterial tensions do increase during the initial 4 minutes of recovery while no apparent CO\textsubscript{2} retention occurs in the same time interval. This state of affairs may be explained as an error in the assumption that the basic CO\textsubscript{2} production has not changed.
It most probably did increase somewhat as a result primarily of the continued hyperpnea existing during recovery. Hence the CO\textsubscript{2} retention would not be zero during the first four minutes as is indicated in Figure 16, and the Pa\textsubscript{CO_{2}} and CO\textsubscript{2} retention data over this initial period of recovery would be more compatible.

Regardless of this possibility, this would still represent a negligible accumulation of CO\textsubscript{2} in the body stores, and the linearity of the latter portion of the average retention curve would be maintained.

However, an alternate explanation is more likely. With the sudden diminution of tidal volume to below the artificially maintained hyperventilation levels (but still remaining above control values) evidently a situation may be obtained whereby the venous blood does not lose CO\textsubscript{2} at the same rate as it did immediately prior to the beginning of recovery; thus, a resistance (the lowered ventilation) is put into the circuit at the lung level resulting in an abrupt increase in Pa\textsubscript{CO_{2}} at the start of recovery. After this initial sharp rise of arterial CO\textsubscript{2} tension, only a gradual rise in tension prevails during the remaining recovery period. Under these conditions the sudden lowering of the lung volume into which venous CO\textsubscript{2} is free to diffuse results in a rapid damming effect in which venous PCO\textsubscript{2} essentially equilibrates with lung PCO\textsubscript{2} which in turn equilibrates with arterial blood. Thus arterial blood for a short time becomes similar to
venous blood with respect to $P_{CO_2}$, but since it is well oxygenated a shift is made on the blood $CO_2$ dissociation curve from reduced blood to oxygenated blood at a slightly higher level of $PCO_2$ and $CO_2$ content. In these experiments, this group shows within the first minute of recovery a rise in $P_{aCO_2}$ of 3-5 mm. Hg. and an increase in arterial $CO_2$ content of approximately 1-5 vol. per cent. During this early transition period the alveolar $PCO_2$, simulating venous $PCO_2$, thus contains a greater concentration of $CO_2$.

With the hyperpneic character of the ventilation at this point, the elimination of $CO_2$ is thus maintained at a level similar to the production level and the measurable retention of $CO_2$ is negligible.

A similar resistance at the lung level is encountered in apneic and hypoventilatory individuals and accounts for some of the abrupt rise in $P_{aCO_2}$ which is evident in Figures 17 and 18. Since the diminution in breathing is of a greater degree in these instances than in Group I, the initial rise in $P_{aCO_2}$ is greater.

The mean recovery ventilation pattern of Group I (Figure 19) approaches the control level without hypoventilation or apnea occurring within the experimental time. It is the result of a gradually diminishing tidal volume and a slightly less gradually diminishing frequency.
It is interesting to note that when the mean total volume of CO₂ regained (measured by integration procedures) and the mean volume of CO₂ regained by the blood (calculated from CO₂ content differences over the recovery period) are used to calculate the blood CO₂ dissociation slope, values of 5.5 cc./kilogram of blood/mm. change in $P_{aCO₂}$ and 5.2 cc./kg./mm. are obtained. These values compare favorably to a normally accepted value of 4.5 cc./kg./mm. It can therefore be stated that the individual who remains hyperpneic during recovery (a) regains little of the lost CO₂ stores during the 12 minutes of recovery, (b) regains blood CO₂ stores first, and (c) regains CO₂ in the blood and tissue reservoirs at a rate determined by the total ventilation and the CO₂ tension gradient established at the tissue level.

**General Considerations**

It is apparent, therefore, that the flier who becomes cognizant of hyperventilation will most rapidly return his acid-base equilibrium toward normal by breath holding. This has been basic procedure. The duration of breath holding would depend primarily on the volume of available oxygen stores in the whole body. If this period of breath holding is followed by a short period of low tidal volume breathing on either air or oxygen, the rate of fall of pH toward normal will be less marked but will still continue while oxygen
requirements are met. If this is followed by another short period of breath holding, it seems that a recontinuation of the rapid rise of blood CO₂ content, and hence an opposite pH change, should result. As far as the blood picture is concerned, acid-base equilibrium would be returned toward normal quickly in this manner. However, if a considerable volume of tissue CO₂ had been lost during hyperventilation, complete readjustment would probably take several hours (4), especially if several episodes of overbreathing had occurred within a short interval of time.

The obvious problem encountered in this procedure is the abrupt drop in oxygen tension and saturation which could result during breath holding under certain emergency conditions. In apneic and extremely hypoventilatory individuals, the accumulation of CO₂ naturally increases most rapidly, but with the creation of dangerously low arterial oxygen tension levels, the lowest tension recorded in these experiments resulting from post-hyperventilation apnea being approximately 40 mm.Hg. Thus, if confusion due to hyperventilation (34) is compounded by hypoxia, psychomotor function would be seriously impaired.

That the total CO₂ stores of the body are compartmentalized with respect to availability was fully demonstrated recently by Vance and Fowler (11), and partially by Tomashefski, Carter and
Lipsky (6). The carbon dioxide of the functional residual capacity while not truly being part of the carbon dioxide stores is lost first during hyperventilation along with lung tissue carbon dioxide. The blood reservoir follows quickly while the soft tissue stores follow the gradient at a rate which is dependent on their degree of perfusion. Considering that perfusion may change or is different in local areas of this mass of tissue, pools of CO₂ (11) are made available for dynamic interchange between compartments at varying times. Similarly, fat and bone stores follow the established gradient and equilibrate slowly with their environs. The result is that a true steady state of equilibrated stores may not prevail for hours (4) when a moderate degree of hyperventilation is maintained in an individual.

Similarly, after only a brief period of moderate hyperventilation (12 minutes), normal complete recovery of the total body CO₂ stores may occupy several hours, even though an examination of arterial pH and CO₂ content may reveal near-control conditions existing after only 12 minutes. This indicates, therefore, that compensating ion shifts within tissues and interstitial fluid would not be completely corrected for some time; and, as alluded to previously, arterial pH, CO₂ content and CO₂ tension measurements which might be taken during recovery to estimate the return to a steady state
condition, are misleading, as would be the observations on alveolar samples. A complete picture of compartmentalization cannot easily be demonstrated during recovery since retention is dependent not only on availability of compartments but also on the variable leak (ventilation).

Boothby's (5) experiments showed that an apneic response (one subject) to approximately 2 minutes of forced hyperventilation resulted in 91 per cent of the lost CO₂ stores being regained in 12 minutes, while an hyperpneic response (one subject) resulted in approximately 75 per cent retention in 12 minutes. This is a large retention volume in each case in comparison to the results of the present experiments. The reason for this difference probably is that Boothby's hyperventilation time was sufficient for the lung CO₂ and blood stores CO₂ to be adequately depleted, but insufficient for much change to occur in the tissue reservoirs. Since the lost blood stores CO₂ is regained relatively rapidly, this reported accumulation represents a rather large fraction of the total stores loss of CO₂.

The loss and restoration of CO₂ stores during hyperventilation and recovery are depicted in Figure 22. It simply shows that the closed system utilized during hyperventilation forcibly maintains near-uniform losses of CO₂ stores in all subjects of these
experiments, and that the open system utilized during the recovery process results in rather markedly different mean patterns of restoration of the lost CO₂ stores. Individuality with regard to a recovery pattern is to be emphasized, however, even though an attempt has here been made to describe three typical pathways which may be followed during post-hyperventilation retention.

The mean value (1.7 cc./kg./mm.) of the individual whole body CO₂ dissociation slopes (Table 13) compares favorably with that of Vance and Fowler (2.05 cc./kg./mm.) (11) even though their periods of hyperventilation were five times as long as those of the present experiments. The average slope of the curve obtained during the recovery process (0.7 cc./kg./mm.) indicates the slowness of the recovery process.

However, the whole body CO₂ dissociation slopes obtained by Vance and Fowler and that obtained in these and many other experiments are wrong, according to Farhi and Rahn (4), because equilibration of CO₂ was not achieved for a specific decrease in PCO₂. They are evidently low values because of the slowness of the equilibration process. The tissue slopes (Vance and Fowler, 1.6 cc./kg./mm.; present study, 1.2 cc./kg./mm.) also must obviously be low, since the tissue loss or absorption of CO₂ during or after hyperventilation lags behind the blood reservoir change.
It is obvious from the literature that investigators have relied on saturation and desaturation experiments for determining the whole body or tissue CO₂ dissociation slope. It is assumed that a linear slope exists from 25 mm. to 80 mm.Hg. CO₂ pressure, and that the starting and end points represent equilibrium conditions. Therefore, measurement of the total loss or retention of CO₂ could be referable to a particular change in blood carbon dioxide tension, arterial or venous, if equilibrium exists.

Table 16 depicts the various whole body CO₂ dissociation slopes existent in the literature. Rahn, in his most recent study, does not furnish another value to enter into this table but does offer the suggestion that "the investigation of the body CO₂ stores presents a real dilemma".

Analysis of the mean CO₂ retention data of Group III indicates that after 12 minutes of recovery, 94 per cent of the blood CO₂ stores have been regained. Therefore, the majority of the CO₂ still to be regained will be retained by the tissues. If the blood reservoir is considered to be almost equilibrated with its normal volume of CO₂, the difference between the total stores loss of CO₂ and the total stores gain after 12 minutes of recovery results in the tissue CO₂ volume still to be regained in order that the control condition might once again be achieved. The difference between
control and final $P_{acO_2}$ represents the change in CO$_2$ tension over which the volume of CO$_2$ retained by the tissues will be made. It can therefore be estimated that a tissue CO$_2$ dissociation slope of 6.7 cc./kg./mm. results from these considerations. It is not considered that this represents a true slope, since (a) the blood stores are not completely filled, and (b) the tissue filling rate does not take into account the lag time present between blood and tissue CO$_2$ retention, so that the change in blood CO$_2$ tension over which the tissue compartment will regain its lost CO$_2$ stores may be quite variable. Otherwise, time is not a factor in this calculation.

Significance and Possible Extension of this Study

The principal significance of this study is that it serves to emphasize the slowness of the return to equilibrium conditions after the tissues have been depleted of several liters of carbon dioxide. In addition, it is submitted that only 2 of 14 subjects exhibited post-hyperventilation apnea, and that these individuals, active in the field for a number of years, were most probably aware of the classic result of acute hyperventilation experiments. Thus, while a low arterial carbon dioxide tension and a high pH tend to inhibit respiration, other factors may be present which stimulate respiration to various degrees over and above the
chemical influences apparent with hyperventilation. These factors have been described as (a) a continued cortical effect on the respiratory centers of the medulla (20, 21), and (b) a build-up of carbon dioxide in the tissues of the medullary reticular area as a result of a decreased perfusion during hyperventilation (5, 22).

It would be of interest to carry out additional experiments of the type described herein, but on a repetitive basis, in order to find out whether or not an individual (a) consistently displays a specific carbon dioxide retention pattern and a specific respiratory response, (b) reaccumulates carbon dioxide in a similar manner when several episodes of hyperventilation occur before recovery is complete, (c) exhibits varying post-hyperventilation responses which are dependent on the volume of stores depletion. Undoubtedly, the results of such experiments would aid in the understanding of recovery from acute hyperventilation.
V. CONCLUSIONS

1. Arterial blood and alveolar air analyses may not be good indicators of the return to a steady state after acute hyperventilation of 12 minutes duration, where tissue CO$_2$ stores have been impinged upon to a considerable degree. The return to a steady state condition may occupy several hours.

2. With a mean loss of approximately two and one-half liters of CO$_2$ from the body stores during 12 minutes of hyperventilation, only one-third of that amount is restored during the ensuing 12 minute recovery period, mostly as result of hypoventilation rather than apnea.

3. It has been shown that acute mechanically induced hyperventilation of individuals, irrespective of their knowledge of the classic respiratory response to hyperventilation, results in fairly distinct patterns of CO$_2$ retention, although individual variations are sizeable. An attempt has been made to place retention data into three groups, dependent upon the immediate respiratory response to hyperventilation. Patterns of retention have been classed as: 
   (a) Group I, continued hyperpnea of relatively long duration,
   (b) Group II, delayed hypoventilation, (c) Group III, apnea or
immediate distinct hypoventilation. After 12 minutes of recovery, Groups I, II, and III indicate a restoration of 13, 30, and 51 percent, respectively, of the CO$_2$ stores which were lost during hyperventilation.

4. It is submitted that two of the 14 subjects exhibited post-hyperventilation apnea, and that these individuals, active in the field for a number of years were most probably aware of the classic result of hyperventilation. These experiments, therefore, tend to support Fink's recent pronouncement that apnea may not be the normal response to short term hyperventilation of unanesthetized man.

5. The lengthy recovery time of tissues CO$_2$ stores, based on the hazardous assumption of a constant CO$_2$ production level, suggests an incomplete restoration of tissue ionic shifts.
APPENDIX
GLOSSARY OF SYMBOLS

B = Barometric pressure
N = Control
EH = End of hyperventilation
s = Serum

\( P_{\text{ACO}_2} \) = Alveolar or end-tidal carbon dioxide tension
\( \text{R-S } P_{\text{aCO}_2} \) = Arterial carbon dioxide tension, Roughton-Scholander technique
\( \text{R-S } P_{\text{aO}_2} \) = Arterial oxygen tension, Roughton-Scholander technique
Calc. \( P_{\text{aCO}_2} \) = Arterial carbon dioxide tension, calculated
\( \text{FECO}_2 \) = Fraction of carbon dioxide, mixed expired air
\( \text{FE}O_2 \) = Fraction of oxygen, mixed expired air
Hcrt. = Hematocrit

BTPS = Body temperature, pressure, saturated

STPD = Standard temperature, pressure, saturated

\( V_{\text{O}_2N} \) = cc. \( \text{O}_2 \) utilized per minute; control
\( V_{\text{O}_2R} \) = cc. \( \text{O}_2 \) utilized per minute; recovery
\( V_{\text{CO}_2\text{SN}} \) = cc. \( \text{CO}_2 \) output per minute; control, spirometer data
\( V_{\text{CO}_2R} \) = cc. \( \text{CO}_2 \) output per minute; recovery, calculated from control exchange ratio and measured recovery \( \text{O}_2 \) utilization
\( V_{\text{CO}_2\text{Int.N}} \) = cc. \( \text{CO}_2 \) output per minute; control, integrated
\( V_{\text{CO}_2\text{Int.R}} \) = cc. \( \text{CO}_2 \) output per minute; recovery, integrated
\[ V_{\text{CO}_2_{\text{SR}}} = \text{cc. CO}_2 \text{ output per minute; recovery, spirometer data} \]

\[ f_N = \text{Respiratory frequency; control} \]

\[ f_{\text{IH}} = \text{Respiratory frequency; initial, hyperventilation} \]

\[ f_{\text{FH}} = \text{Respiratory frequency; final, hyperventilation} \]

\[ f_{\text{IR}} = \text{Respiratory frequency; initial, recovery} \]

\[ f_{\text{FR}} = \text{Respiratory frequency; final, recovery} \]

\[ R_N = \text{Respiratory exchange ratio; control} \]

\[ R_R = \text{Actual respiratory exchange ratio; recovery} \]

\[ \bar{x} = \text{Mean} \]

\[ \bar{d} = \text{Mean difference} \]

\[ s_d = \text{Standard deviation of mean difference} \]

\[ s^2 = \text{Variance of difference means} \]

\[ s_x = \text{Standard deviation} \]
<table>
<thead>
<tr>
<th>TABLE I-A</th>
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<td><strong>SUBJECT M.C.: BLOOD AND RESPIRATION DATA</strong></td>
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B = Barometric Pressure  
N = Control  
EH = End of Hyperventilation  
* = Concentration in Total expired recovery air  
s = Serum
### TABLE I-B

**SUBJECT E.C.: BLOOD AND RESPIRATION DATA**

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**SUBJECT K.K.: BLOOD AND RESPIRATION DATA**

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**SUBJECT J.S.: BLOOD AND RESPIRATION DATA**

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TABLE I-H

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## TABLE I-I

**SUBJECT G.L.: BLOOD AND RESPIRATION DATA**

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<td>Duration of Measured Recovery (Min.)</td>
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### TABLE I-K

**SUBJECT J.M.: BLOOD AND RESPIRATION DATA**

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- FE CO2: 4.88, 3.89*
- FE O2: 15.05, 11.78*
- Paco2: 42.27, 21.31, 26.83, 29.17, 33.63, 33.28
- R-S Paco2: 47.08, 30.45, 38.24, 39.29, 42.83, 43.54
- R-S PacO2: 96.64, 110.09, 92.39, 82.83, 67.97, 90.27
- Calc. Paco2: 44.15, 25.62, 30.60, 31.99, 37.03, 39.72
- CO2 Content: 47.84, 38.91, 42.88, 43.96, 46.04, 45.41
- Hcrt.: 44, 45, 45, 45, 45, 45
- pH: 7.38, 7.55, 7.51, 7.50, 7.45, 7.41
- Dissolved (CO2)s: 2.97, 1.72, 2.06, 2.15, 2.49, 2.67
- (BHCO3)s: 55.35, 48.27, 51.45, 52.67, 54.19, 53.08
- O2 Saturation: 97, 98, 97, 96, 93, 96
### TABLE I-L

**SUBJECT G.H.: BLOOD AND RESPIRATION DATA**

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<td>( Pa_{CO_2} ) (mm.Hg.)</td>
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<td>19.68</td>
<td>21.76</td>
<td>23.70</td>
<td>27.79</td>
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<td>( FE_{CO_2} ) (%)</td>
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<td>2.84*</td>
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<td>( FE_{O_2} ) (%)</td>
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<td>14.48*</td>
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<td>22.40</td>
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### TABLE I-M

**SUBJECT D.C.: BLOOD AND RESPIRATION DATA**

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### TABLE I-N

**SUBJECT F.M.: BLOOD AND RESPIRATION DATA**

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<td>7.53</td>
<td>7.49</td>
<td>7.44</td>
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<tr>
<td>Dissolved ($CO_2$)s (Vol. %)</td>
<td>3.10</td>
<td>1.71</td>
<td>1.95</td>
<td>2.19</td>
<td>2.57</td>
<td>2.94</td>
</tr>
<tr>
<td>($BH_{CO_3}$)s (Vol. %)</td>
<td>55.01</td>
<td>49.16</td>
<td>51.02</td>
<td>52.25</td>
<td>54.63</td>
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<td>$O_2$ Saturation (%)</td>
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<td>97</td>
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<td>94</td>
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</tbody>
</table>
### TABLE II

**Respiratory Frequency During Hyperventilation**

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<th>1-2</th>
<th>2-3</th>
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<th>7-8</th>
<th>8-9</th>
<th>9-10</th>
<th>10-11</th>
<th>11-12</th>
</tr>
</thead>
<tbody>
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</tr>
<tr>
<td>L.P.</td>
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<tr>
<td>J.T.</td>
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<tr>
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<td>11</td>
<td>12</td>
<td>12</td>
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</tr>
<tr>
<td>G.L.</td>
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<td>19</td>
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<td>20</td>
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<tr>
<td>F.M.</td>
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\[
\bar{x} = 11.1 \quad 15.3 \quad 15.6 \quad 15.6 \quad 16.2 \quad 16.4 \quad 16.4 \quad 16.5 \quad 16.1 \quad 15.6 \quad 15.6 \quad 15.9 \quad 15.9
\]

\[
s = +4.5 \quad +3.5 \quad +4.0 \quad +4.3 \quad +4.5 \quad +5.0 \quad +5.0 \quad +5.2 \quad +4.3 \quad +4.4 \quad +5.0 \quad +4.8 \quad +4.5
\]

\(\bar{x}\) = Mean

\(s\) = Standard deviation
## TABLE III

### RESPIRATORY FREQUENCY DURING RECOVERY

<table>
<thead>
<tr>
<th>Subject</th>
<th>Control</th>
<th>0-1</th>
<th>1-2</th>
<th>2-3</th>
<th>3-4</th>
<th>4-5</th>
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<th>6-7</th>
<th>7-8</th>
<th>8-9</th>
<th>9-10</th>
<th>10-11</th>
<th>11-12</th>
</tr>
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<tbody>
<tr>
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<tr>
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<tr>
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<tr>
<td>G.L.</td>
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<td>11</td>
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<td>26</td>
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<td>20</td>
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<td>17</td>
<td>19</td>
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<td>18</td>
<td>17</td>
</tr>
<tr>
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<td>6</td>
<td>7</td>
<td>7</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
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<td>11</td>
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<td>7</td>
<td>8</td>
<td>6</td>
<td>7</td>
</tr>
<tr>
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<td>11</td>
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<td>10</td>
<td>10</td>
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<tr>
<td>F.M.</td>
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<td>23</td>
<td>18</td>
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<td>15</td>
<td>16</td>
<td>18</td>
<td>17</td>
<td>17</td>
</tr>
</tbody>
</table>

| x       | 11.1   | 10.4 | 10.1 | **9.9** | 9.7 | 10.0 | 10.4 | 10.3 | 10.8 | 10.7 | 10.5 | 11.0  | 11.3  |
| s       | ±4.5   | ±6.1 | ±7.0 | ±6.2 | ±5.1 | ±4.6 | ±5.3 | ±3.4 | ±3.8 | ±3.5 | ±3.8 | ±3.7  | ±3.7  |

$x = \text{Mean}$

$s = \text{Standard deviation}$
TABLE IV

HYPERVENTILATION MEAN TIDAL VOLUMES (CC., BTPS)

<table>
<thead>
<tr>
<th>Subject</th>
<th>Control</th>
<th>0-1</th>
<th>1-2</th>
<th>2-3</th>
<th>5-6</th>
<th>11-12</th>
</tr>
</thead>
<tbody>
<tr>
<td>M. C.</td>
<td>1,141</td>
<td>1,305</td>
<td>1,661</td>
<td>1,748</td>
<td>1,480</td>
<td>1,898</td>
</tr>
<tr>
<td>E. C.</td>
<td>880</td>
<td>1,806</td>
<td>1,623</td>
<td>1,378</td>
<td>1,365</td>
<td>1,347</td>
</tr>
<tr>
<td>L. P.</td>
<td>642</td>
<td>1,944</td>
<td>2,262</td>
<td>2,026</td>
<td>2,423</td>
<td>2,285</td>
</tr>
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<td>J. T.</td>
<td>860</td>
<td>1,326</td>
<td>1,439</td>
<td>1,155</td>
<td>1,160</td>
<td>1,203</td>
</tr>
<tr>
<td>A. H.</td>
<td>350</td>
<td>792</td>
<td>749</td>
<td>611</td>
<td>500</td>
<td>1,003</td>
</tr>
<tr>
<td>K. K.</td>
<td>500</td>
<td>1,772</td>
<td>1,665</td>
<td>1,484</td>
<td>1,133</td>
<td>1,164</td>
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<tr>
<td>J. S.</td>
<td>634</td>
<td>2,333</td>
<td>2,437</td>
<td>2,565</td>
<td>2,136</td>
<td>1,464</td>
</tr>
<tr>
<td>D. S.</td>
<td>658</td>
<td>3,004</td>
<td>3,290</td>
<td>3,611</td>
<td>2,805</td>
<td>2,458</td>
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<tr>
<td>G. L.</td>
<td>695</td>
<td>1,383</td>
<td>1,225</td>
<td>1,124</td>
<td>808</td>
<td>832</td>
</tr>
<tr>
<td>J. P.</td>
<td>530</td>
<td>1,590</td>
<td>1,551</td>
<td>1,360</td>
<td>1,058</td>
<td>922</td>
</tr>
<tr>
<td>J. M.</td>
<td>998</td>
<td>1,959</td>
<td>1,942</td>
<td>2,039</td>
<td>1,857</td>
<td>1,876</td>
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<tr>
<td>G. H.</td>
<td>824</td>
<td>2,237</td>
<td>2,214</td>
<td>2,120</td>
<td>1,302</td>
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</tr>
<tr>
<td>D. C.</td>
<td>758</td>
<td>2,969</td>
<td>2,576</td>
<td>2,343</td>
<td>1,492</td>
<td>1,247</td>
</tr>
<tr>
<td>F. M.</td>
<td>366</td>
<td>815</td>
<td>774</td>
<td>732</td>
<td>679</td>
<td>613</td>
</tr>
<tr>
<td>$\overline{x}$</td>
<td>703</td>
<td>1,803</td>
<td>1,815</td>
<td>1,735</td>
<td>1,443</td>
<td>1,409</td>
</tr>
<tr>
<td>s</td>
<td>± 227</td>
<td>± 680</td>
<td>± 700</td>
<td>± 795</td>
<td>± 662</td>
<td>± 565</td>
</tr>
</tbody>
</table>

$\overline{x}$ = Mean

s = Standard Deviation
<table>
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<tr>
<th>Subject</th>
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<th>0-1</th>
<th>1-2</th>
<th>2-3</th>
<th>3-4</th>
<th>4-5</th>
</tr>
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<tr>
<td>M. C.</td>
<td>1,141</td>
<td>1,381</td>
<td>1,319</td>
<td>1,475</td>
<td>1,298</td>
<td>1,272</td>
</tr>
<tr>
<td>E. C.</td>
<td>880</td>
<td>738</td>
<td>1,015</td>
<td>1,020</td>
<td>662</td>
<td>919</td>
</tr>
<tr>
<td>L. P.</td>
<td>642</td>
<td>2,416</td>
<td>1,894</td>
<td>1,781</td>
<td>1,613</td>
<td>1,422</td>
</tr>
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<td>J. T.</td>
<td>860</td>
<td>354</td>
<td>427</td>
<td>539</td>
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<td>854</td>
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<td>A. H.</td>
<td>350</td>
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<td>1,069</td>
<td>1,247</td>
<td>543</td>
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<tr>
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<td>500</td>
<td>774</td>
<td>337</td>
<td>558</td>
<td>491</td>
<td>497</td>
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<tr>
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<td>887</td>
<td>643</td>
<td>560</td>
<td>560</td>
<td>545</td>
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<td>658</td>
<td>645</td>
<td>436</td>
<td>403</td>
<td>401</td>
<td>419</td>
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<tr>
<td>G. L.</td>
<td>695</td>
<td>408</td>
<td>266</td>
<td>325</td>
<td>389</td>
<td>461</td>
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<td>829</td>
<td>530</td>
<td>520</td>
<td>678</td>
<td>483</td>
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<tr>
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<td>923</td>
<td>633</td>
<td>633</td>
<td>547</td>
<td>566</td>
</tr>
<tr>
<td>G. H.</td>
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<td>1,007</td>
<td>955</td>
<td>867</td>
<td>954</td>
<td>848</td>
</tr>
<tr>
<td>D. C.</td>
<td>758</td>
<td>987</td>
<td>824</td>
<td>647</td>
<td>635</td>
<td>517</td>
</tr>
<tr>
<td>F. M.</td>
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<td>330</td>
<td>278</td>
<td>264</td>
<td>220</td>
<td>227</td>
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</tbody>
</table>

\[ \bar{x} = 703 \quad \pm 227 \]
\[ s = 538 \quad 546 \quad 439 \quad 404 \quad 338 \]

\[ \bar{x} = \text{Mean} \]

\[ s = \text{Standard Deviation} \]
### TABLE V (CONTD.)

**RECOVERY MEAN TIDAL VOLUMES (CC., BTPS)**

<table>
<thead>
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<th>TIME (MINUTES)</th>
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<th>6-7</th>
<th>7-8</th>
<th>8-9</th>
<th>9-10</th>
<th>10-11</th>
<th>11-12</th>
</tr>
</thead>
<tbody>
<tr>
<td>1,247</td>
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<td>1,423</td>
<td>1,336</td>
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<tr>
<td>680</td>
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<td>860</td>
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667, 665, 665, 691, 628, 627, 656

±338, +360, +314, +313, +232, +205, +262
TABLE VI
NORMAL AND RECOVERY CO₂ & O₂ VOLUME MEASUREMENTS

<table>
<thead>
<tr>
<th>Subj.</th>
<th>( \dot{V}_{O₂N} )</th>
<th>( \dot{V}_{O₂R} )</th>
<th>( \dot{V}_{CO₂SN} )</th>
<th>( \dot{V}_{CO₂R} )</th>
<th>( \dot{V}_{CO₂} )</th>
<th>( \dot{V}_{CO₂SR} )</th>
<th>( \dot{V}_{CO₂} )</th>
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<td>261</td>
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<td>112</td>
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\[ \bar{d} = +95.0^* \quad +76.8^* \quad -68.6^* \quad +12.0 \quad +10.36 \]
\[ \bar{s}_d = 22.59 \quad 18.9 \quad 7.84 \quad 4.03 \quad 2.50 \]
\[ s^2 = \frac{\bar{x}_2 - \bar{x}_1}{172.5} \]

* = Significant
**TABLE VII**

**FREQUENCY AND RESPIRATORY EXCHANGE (STATISTICAL ANALYSIS)**

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<th>( f_{IH} )</th>
<th>( f_{IH} )</th>
<th>( f_{FH} )</th>
<th>( f_N )</th>
<th>( f_{IR} )</th>
<th>( f_N )</th>
<th>( f_{FR} )</th>
<th>( R_N )</th>
<th>( R_R )</th>
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<td>1.427</td>
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<td>8</td>
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<td>17</td>
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<td>19</td>
<td>19</td>
<td>15</td>
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<td>0.273</td>
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<td>12</td>
<td>14</td>
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<td>11</td>
<td>12</td>
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<td>11</td>
<td>8</td>
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<td>18</td>
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<td>6</td>
<td>6</td>
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<td>17</td>
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<td>0.374</td>
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<td>0.504</td>
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\( \bar{x} \) 11.1 15.3 15.3 15.9 11.1 10.4 11.1 11.3 0.812 0.445

\( \bar{d} \) +4.21* +0.71 -0.64 0 -0.356*

\( s_{\bar{d}} \) 1.12 0.78 1.48 0 0.083

\( P = 0.05 \)

* = significant
TABLE VIII-A

HYPERVENTILATION MINUTE VOLUME (CC., BTPS)

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<tr>
<th>Subject</th>
<th>Control</th>
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<th>1-2</th>
<th>2-3</th>
<th>5-6</th>
<th>11-12</th>
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<td>20,880</td>
<td>24,915</td>
<td>26,220</td>
<td>22,200</td>
<td>24,674</td>
</tr>
<tr>
<td>E.C.</td>
<td>6,160</td>
<td>23,478</td>
<td>21,099</td>
<td>20,670</td>
<td>19,110</td>
<td>17,511</td>
</tr>
<tr>
<td>L.P.</td>
<td>7,704</td>
<td>23,328</td>
<td>29,406</td>
<td>24,312</td>
<td>26,763</td>
<td>25,135</td>
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<tr>
<td>J.T.</td>
<td>6,880</td>
<td>22,542</td>
<td>24,463</td>
<td>19,635</td>
<td>18,560</td>
<td>21,654</td>
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<td>14,231</td>
<td>12,220</td>
<td>10,000</td>
<td>17,051</td>
</tr>
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<td>24,975</td>
<td>20,776</td>
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<td>18,624</td>
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<td>24,370</td>
<td>25,650</td>
<td>23,496</td>
<td>21,960</td>
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<td>24,500</td>
<td>21,356</td>
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<td>16,640</td>
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<td>30,210</td>
<td>21,020</td>
<td>28,560</td>
<td>24,334</td>
<td>23,050</td>
</tr>
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<td>23,304</td>
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<td>16,884</td>
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<td>32,802</td>
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<td>15,617</td>
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<td>26,427</td>
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<td>20,471</td>
</tr>
<tr>
<td>$s$</td>
<td>$\pm 1,218$</td>
<td>$\pm 6,181$</td>
<td>$\pm 7,503$</td>
<td>$\pm 8,168$</td>
<td>$\pm 6,227$</td>
<td>$\pm 4,393$</td>
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</table>

$\bar{x} = \text{Mean}$

$s = \text{Standard Deviation}$
<table>
<thead>
<tr>
<th>Subject</th>
<th>Control</th>
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<th>1-2</th>
<th>2-3</th>
<th>3-4</th>
<th>4-5</th>
</tr>
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<tr>
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<td>+4,335</td>
<td>+4,391</td>
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<td>+2,645</td>
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</table>

\text{\textbar}{x} = \text{Mean}

\text{s} = \text{Standard Deviation}
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<th>8-9</th>
<th>9-10</th>
<th>10-11</th>
<th>11-12</th>
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<td>+2,556</td>
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<td>+1,901</td>
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# TABLE IX-A

## CONTROL ARTERIAL BLOOD AND END-TIDAL MEASUREMENTS

<table>
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<th>Subject</th>
<th>Time (Min.)</th>
<th>R-S PaO₂ (mm. Hg.)</th>
<th>R-S PaCO₂ (mm. Hg.)</th>
<th>Calc. PaCO₂ (mm. Hg.)</th>
<th>End-Tidal PaCO₂ (mm. Hg.)</th>
<th>pH</th>
<th>(BHCO₃)₅ (mM/L)</th>
</tr>
</thead>
<tbody>
<tr>
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<td>24.47</td>
</tr>
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<td>43.1</td>
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<td>23.43</td>
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<td>22.26</td>
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<td>36.4</td>
<td>7.39</td>
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<td>42.0</td>
<td>7.38</td>
<td>25.15</td>
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<td>(\bar{x})</td>
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<td>44.7</td>
<td>39.0</td>
<td>7.37</td>
<td>24.20</td>
</tr>
<tr>
<td>(s)</td>
<td></td>
<td>± 6.7</td>
<td>± 3.6</td>
<td>± 4.8</td>
<td>± 3.3</td>
<td>± 0.05</td>
<td>± 0.98</td>
</tr>
<tr>
<td>(\bar{S}_{x-\bar{x}})</td>
<td></td>
<td>(\pm 1.2814)</td>
<td>(\pm 1.1934)*</td>
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| P       |             | 0.01                | 0.01                |                      |                          |      | 87

\(P = \) probability level

* = Significant
TABLE IX-B
END-HYPERVENTILATION ARTERIAL BLOOD AND END-TIDAL MEASUREMENTS

<table>
<thead>
<tr>
<th>Subject</th>
<th>Time (Min.)</th>
<th>R-S PaO₂ (mm. Hg.)</th>
<th>R-S PaCO₂ (mm. Hg.)</th>
<th>Calc. PaCO₂ (mm. Hg.)</th>
<th>End-Tidal PaCO₂ (mm. Hg.)</th>
<th>pH</th>
<th>(BHCO₃)ₘ (mM/L)</th>
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</thead>
<tbody>
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<td>93.3</td>
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<td>28.4</td>
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<td>7.50</td>
<td></td>
<td>20.95</td>
</tr>
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<td>27.8</td>
<td>25.3</td>
<td>19.7</td>
<td>7.53</td>
<td></td>
<td>20.07</td>
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<td>26.4</td>
<td>19.1</td>
<td>7.47</td>
<td></td>
<td>18.26</td>
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<tr>
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<td>19.29</td>
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<td>19.7</td>
<td>7.59</td>
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<td>20.38</td>
</tr>
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<td>20.90</td>
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<td>22.08</td>
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<td>± 5.1</td>
<td>± 3.6</td>
<td>± 1.7</td>
<td>± 0.06</td>
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<td>± 1.16</td>
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\[
s_{x_2-x_1} = +1.3980 + 0.5782^* \\
P = 0.01 0.01
\]

EH = End-hyperventilation
TABLE IX-C

RECOVERY PERIOD I: ARTERIAL BLOOD AND END-TIDAL MEASUREMENTS

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<tr>
<th>Subject</th>
<th>Time (Min.)</th>
<th>R-S PaO₂ (mm. Hg.)</th>
<th>R-S PaCO₂ (mm. Hg.)</th>
<th>Calc. PaCO₂ (mm. Hg.)</th>
<th>End-Tidal PACO₂ (mm. Hg.)</th>
<th>pH</th>
<th>(BHCO₃)ₐ (mM/L)</th>
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</thead>
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<td>M. C.</td>
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<td>91.9</td>
<td>27.6</td>
<td>25.9</td>
<td>22.2</td>
<td>7.51</td>
<td>19.58</td>
</tr>
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<td>33.2</td>
<td>27.9</td>
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<td>17.0</td>
<td>7.65</td>
<td>19.60</td>
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<td>7.44</td>
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<td>25.6</td>
<td>22.0</td>
<td>7.53</td>
<td>20.26</td>
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<td>29.9</td>
<td>7.43</td>
<td>22.57</td>
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<tr>
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<td>7.51</td>
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<tr>
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<td>7.56</td>
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<td>7.53</td>
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<td>+4.6</td>
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<td>+1.3000*</td>
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P = 0.01
# TABLE IX-D

**RECOVERY PERIOD II: BLOOD AND END-TIDAL MEASUREMENTS**

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<th>Subject</th>
<th>Time (Min.)</th>
<th>R-S PaO₂ (mm. Hg.)</th>
<th>R-S PaCO₂ (mm. Hg.)</th>
<th>Calc. PaCO₂ (mm. Hg.)</th>
<th>End-Tidal PaCO₂ (mm. Hg.)</th>
<th>pH</th>
<th>(BHCO₃) s (mM/L)</th>
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<td>19.65</td>
</tr>
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</tr>
<tr>
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<td>40.8</td>
<td>32.3</td>
<td>7.40</td>
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<tr>
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<td>107.1</td>
<td>27.3</td>
<td>30.1</td>
<td>23.9</td>
<td>7.47</td>
<td>20.77</td>
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<td>7.48</td>
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</table>

\[ s_{x} = \pm 0.13 \quad s_{x} = \pm 21.0 \quad s_{x} = \pm 6.3 \quad s_{x} = \pm 6.6 \quad s_{x} = \pm 8.8 \quad s_{x} = \pm 0.07 \quad s_{x} = \pm 1.72 \]

\[ s_{(x_{2} - x_{1})} = \pm 2.9676 \quad s_{(x_{2} - x_{1})} = \pm 2.4292* \]

\[ P = 0.01 \quad P = 0.01 \]
### TABLE IX-E

**RECOVERY PERIOD III: BLOOD AND END-TIDAL MEASUREMENTS**

<table>
<thead>
<tr>
<th>Subject</th>
<th>Time (Min.)</th>
<th>R-S $\mathrm{PaO}_2$ (mm.Hg.)</th>
<th>R-S $\mathrm{PaCO}_2$ (mm.Hg.)</th>
<th>Calc. $\mathrm{PaCO}_2$ (mm.Hg.)</th>
<th>End-Tidal $\mathrm{PaCO}_2$ (mm.Hg.)</th>
<th>pH</th>
<th>(BHCO$_3$)$_s$ (mM/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>M.C.</td>
<td>5.52</td>
<td>78.3</td>
<td>22.4</td>
<td>27.1</td>
<td>23.8</td>
<td>7.50</td>
<td>20.05</td>
</tr>
<tr>
<td>E.C.</td>
<td>5.00</td>
<td>86.6</td>
<td>37.4</td>
<td>40.7</td>
<td>34.2</td>
<td>7.42</td>
<td>25.08</td>
</tr>
<tr>
<td>L.P.</td>
<td>4.91</td>
<td>101.1</td>
<td>31.3</td>
<td>22.7</td>
<td>19.3</td>
<td>7.57</td>
<td>19.66</td>
</tr>
<tr>
<td>J.T.</td>
<td>5.02</td>
<td>83.4</td>
<td>46.9</td>
<td>39.3</td>
<td>34.8</td>
<td>7.41</td>
<td>23.57</td>
</tr>
<tr>
<td>A.H.</td>
<td>5.00</td>
<td>62.0</td>
<td>30.6</td>
<td>42.0</td>
<td>31.9</td>
<td>7.36</td>
<td>22.41</td>
</tr>
<tr>
<td>K.K.</td>
<td>5.00</td>
<td>62.2</td>
<td>42.8</td>
<td>49.1</td>
<td>37.1</td>
<td>7.33</td>
<td>24.25</td>
</tr>
<tr>
<td>J.S.</td>
<td>4.98</td>
<td>92.3</td>
<td>29.4</td>
<td>32.9</td>
<td>26.5</td>
<td>7.44</td>
<td>21.11</td>
</tr>
<tr>
<td>D.S.</td>
<td>4.90</td>
<td>52.7</td>
<td>39.0</td>
<td>39.3</td>
<td>32.0</td>
<td>7.38</td>
<td>21.95</td>
</tr>
<tr>
<td>G.L.</td>
<td>4.95</td>
<td>59.8</td>
<td>40.9</td>
<td>49.9</td>
<td>37.3</td>
<td>7.31</td>
<td>23.73</td>
</tr>
<tr>
<td>J.P.</td>
<td>4.98</td>
<td>90.8</td>
<td>38.0</td>
<td>28.9</td>
<td>25.3</td>
<td>7.49</td>
<td>20.78</td>
</tr>
<tr>
<td>J.M.</td>
<td>5.03</td>
<td>68.0</td>
<td>42.8</td>
<td>37.0</td>
<td>33.6</td>
<td>7.45</td>
<td>24.34</td>
</tr>
<tr>
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<td>30.0</td>
<td>27.8</td>
<td>7.50</td>
<td>22.16</td>
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<td>65.5</td>
<td>37.8</td>
<td>36.9</td>
<td>30.8</td>
<td>7.43</td>
<td>23.12</td>
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<tr>
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<td>67.5</td>
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<td>38.3</td>
<td>34.1</td>
<td>7.44</td>
<td>24.54</td>
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<tr>
<td>$\bar{x}$</td>
<td>5.01</td>
<td>74.3</td>
<td>36.4</td>
<td>36.7</td>
<td>30.6</td>
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<td>22.63</td>
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<tr>
<td>$s_{x}$</td>
<td>$\pm 0.15$</td>
<td>$\pm 14.5$</td>
<td>$\pm 6.4$</td>
<td>$\pm 7.8$</td>
<td>$\pm 5.3$</td>
<td>$\pm 0.07$</td>
<td>$\pm 1.74$</td>
</tr>
</tbody>
</table>

$s^2_{(x_2-x_1)}$ = $3.6570$; $s^2_{(x_2-x_1)}$ = $3.2032^*$

| P      | 0.01       | 0.01                |
# TABLE IX-F

## RECOVERY PERIOD IV: BLOOD AND END-TIDAL MEASUREMENTS

<table>
<thead>
<tr>
<th>Subject</th>
<th>Time (Min.)</th>
<th>R-S PaO₂ (mm. Hg.)</th>
<th>R-S PaCO₂ (mm. Hg.)</th>
<th>Calc. PaCO₂ (mm. Hg.)</th>
<th>End-Tidal PACO₂ (mm. Hg.)</th>
<th>pH</th>
<th>(BHCO₃)ₛ (mM/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>M. C.</td>
<td>12.00</td>
<td>82.5</td>
<td>40.5</td>
<td>41.8</td>
<td>36.9</td>
<td>7.42</td>
<td>25.74</td>
</tr>
<tr>
<td>E. C.</td>
<td>12.21</td>
<td>103.9</td>
<td>32.3</td>
<td>25.2</td>
<td>21.8</td>
<td>7.54</td>
<td>20.41</td>
</tr>
<tr>
<td>L. P.</td>
<td>12.03</td>
<td>89.7</td>
<td>45.5</td>
<td>40.0</td>
<td>33.2</td>
<td>7.41</td>
<td>24.02</td>
</tr>
<tr>
<td>J. T.</td>
<td>12.02</td>
<td>84.2</td>
<td>36.5</td>
<td>46.7</td>
<td>36.1</td>
<td>7.32</td>
<td>22.78</td>
</tr>
<tr>
<td>A. H.</td>
<td>12.00</td>
<td>82.2</td>
<td>44.9</td>
<td>49.7</td>
<td>39.0</td>
<td>7.32</td>
<td>24.27</td>
</tr>
<tr>
<td>K. K.</td>
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<td>96.4</td>
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<td>34.3</td>
<td>28.2</td>
<td>7.43</td>
<td>21.56</td>
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<tr>
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<td>43.5</td>
<td>45.9</td>
<td>37.9</td>
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<td>22.83</td>
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<td>12.03</td>
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<td>49.9</td>
<td>37.7</td>
<td>7.31</td>
<td>23.76</td>
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<td>G. L.</td>
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<td>90.8</td>
<td>38.4</td>
<td>29.6</td>
<td>29.2</td>
<td>7.49</td>
<td>21.28</td>
</tr>
<tr>
<td>J. P.</td>
<td>12.02</td>
<td>90.3</td>
<td>43.5</td>
<td>39.7</td>
<td>33.3</td>
<td>7.41</td>
<td>23.85</td>
</tr>
<tr>
<td>J. M.</td>
<td>11.95</td>
<td>66.9</td>
<td>39.2</td>
<td>35.9</td>
<td>32.6</td>
<td>7.44</td>
<td>23.04</td>
</tr>
<tr>
<td>G. H.</td>
<td>11.63</td>
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<td>40.6</td>
<td>35.0</td>
<td>7.40</td>
<td>23.75</td>
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<tr>
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<td>36.7</td>
<td>43.7</td>
<td>38.9</td>
<td>7.39</td>
<td>25.00</td>
</tr>
<tr>
<td>F. M.</td>
<td>11.98</td>
<td>83.8</td>
<td>39.7</td>
<td>40.2</td>
<td>33.8</td>
<td>7.40</td>
<td>23.25</td>
</tr>
</tbody>
</table>

\[ s_x \pm 0.13 \quad \pm 11.7 \quad \pm 4.1 \quad \pm 7.4 \quad \pm 5.0 \quad \pm 0.07 \quad \pm 1.50 \]

\[ s_{(\bar{X}_2 - \bar{X}_1)} = +2.7732 \quad +3.0928* \]

\[ P = 0.01 \quad 0.01 \]
<table>
<thead>
<tr>
<th>Subject</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
</tr>
</thead>
<tbody>
<tr>
<td>M.C.</td>
<td>258</td>
<td>526</td>
<td>827</td>
<td>1088</td>
<td>1327</td>
<td>1589</td>
</tr>
<tr>
<td>E.C.</td>
<td>120</td>
<td>228</td>
<td>388</td>
<td>498</td>
<td>635</td>
<td>782</td>
</tr>
<tr>
<td>L.P.</td>
<td>262</td>
<td>483</td>
<td>760</td>
<td>992</td>
<td>1201</td>
<td>1423</td>
</tr>
<tr>
<td>J.T.</td>
<td>50</td>
<td>129</td>
<td>205</td>
<td>369</td>
<td>538</td>
<td>730</td>
</tr>
<tr>
<td>A.H.</td>
<td>80</td>
<td>128</td>
<td>218</td>
<td>397</td>
<td>517</td>
<td>612</td>
</tr>
<tr>
<td>K.K.</td>
<td>32</td>
<td>100</td>
<td>249</td>
<td>353</td>
<td>521</td>
<td>701</td>
</tr>
<tr>
<td>J.S.</td>
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<td>394</td>
<td>582</td>
<td>750</td>
<td>934</td>
<td>1106</td>
</tr>
<tr>
<td>D.S.</td>
<td>120</td>
<td>200</td>
<td>270</td>
<td>356</td>
<td>448</td>
<td>535</td>
</tr>
<tr>
<td>G.L.</td>
<td>54</td>
<td>79</td>
<td>118</td>
<td>203</td>
<td>317</td>
<td>473</td>
</tr>
<tr>
<td>J.P.</td>
<td>306</td>
<td>563</td>
<td>810</td>
<td>1061</td>
<td>1273</td>
<td>1508</td>
</tr>
<tr>
<td>J.M.</td>
<td>148</td>
<td>259</td>
<td>351</td>
<td>436</td>
<td>534</td>
<td>645</td>
</tr>
<tr>
<td>G.H.</td>
<td>270</td>
<td>504</td>
<td>650</td>
<td>791</td>
<td>939</td>
<td>1067</td>
</tr>
<tr>
<td>D.C.</td>
<td>280</td>
<td>496</td>
<td>672</td>
<td>872</td>
<td>1030</td>
<td>1194</td>
</tr>
<tr>
<td>F.M.</td>
<td>174</td>
<td>328</td>
<td>455</td>
<td>577</td>
<td>686</td>
<td>788</td>
</tr>
</tbody>
</table>

**TABLE X**

TOTAL ACCUMULATIVE CO₂ ELIMINATION (CC., STPD)
**TABLE X (CONT'D.)**

**TOTAL ACCUMULATIVE CO$_2$ ELIMINATION (CC., STPD)**

<table>
<thead>
<tr>
<th>Recovery Time (Minutes)</th>
<th>Basic Output V$_{CO_2}$ (Int. N.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>7</td>
<td>8</td>
</tr>
<tr>
<td>1868</td>
<td>2134</td>
</tr>
<tr>
<td>928</td>
<td>1084</td>
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<tr>
<td>1636</td>
<td>1815</td>
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<tr>
<td>942</td>
<td>1166</td>
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<tr>
<td>699</td>
<td>813</td>
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<tr>
<td>845</td>
<td>1014</td>
</tr>
<tr>
<td>1299</td>
<td>1514</td>
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<td>1363</td>
<td>1551</td>
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<tr>
<td>922</td>
<td>1041</td>
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</table>
**TABLE XI**

**TOTAL ACCUMULATIVE CO\textsubscript{2} RETENTION (CC., STPD)**

<table>
<thead>
<tr>
<th>Subject</th>
<th>Recovery Time (Mins.)</th>
</tr>
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<td></td>
<td>1</td>
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<tr>
<td>M.C.</td>
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<tr>
<td>E.C.</td>
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<tr>
<td>L.P.</td>
<td>-19</td>
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<tr>
<td>J.T.</td>
<td>201</td>
</tr>
<tr>
<td>A.H.</td>
<td>121</td>
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<tr>
<td>K.K.</td>
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<tr>
<td>J.S.</td>
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<tr>
<td>D.S.</td>
<td>79</td>
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<tr>
<td>G.L.</td>
<td>137</td>
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<td>J.P.</td>
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<td>J.M.</td>
<td>89</td>
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<td>G.H.</td>
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<td>D.C.</td>
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<td>F.M.</td>
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<td>Recovery Time (Minutes)</td>
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<tr>
<td>-------------------------</td>
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<td>Subjects</td>
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<td>E. C.</td>
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</tr>
<tr>
<td>J. T.</td>
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</tr>
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<td>K. K.</td>
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<td>G. L.</td>
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<td>F. M.</td>
<td>1.6</td>
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<td>TIME (MINUTES)</td>
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<tr>
<td>---------------</td>
<td>-----</td>
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</tr>
<tr>
<td>12</td>
<td>18.9</td>
</tr>
<tr>
<td></td>
<td>24.9</td>
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</table>
TABLE XIII

CO₂ STORES LOSS DURING HYPERVENTILATION

<table>
<thead>
<tr>
<th>Subj.</th>
<th>Total CO₂ Stores</th>
<th>Lung CO₂ CO₂ Loss</th>
<th>Blood CO₂ CO₂ Loss</th>
<th>Tissue CO₂ CO₂ Loss</th>
<th>Body Weight Kg</th>
<th>PaCO₂ mm. Hg.</th>
<th>Whole Body Dissociation Slope</th>
<th>Tissue Dissociation Slope</th>
</tr>
</thead>
<tbody>
<tr>
<td>M.C.</td>
<td>2199 cc</td>
<td>63 cc</td>
<td>698 cc</td>
<td>1438 cc</td>
<td>70.5 Kg</td>
<td>19.8 mm. Hg.</td>
<td>1.57 cc/kg/mm.</td>
<td>1.03 cc/kg/mm.</td>
</tr>
<tr>
<td>E.C.</td>
<td>2542 cc</td>
<td>60 cc</td>
<td>705 cc</td>
<td>1777 cc</td>
<td>79.5 Kg</td>
<td>16.1 mm. Hg.</td>
<td>1.98 cc/kg/mm.</td>
<td>1.39 cc/kg/mm.</td>
</tr>
<tr>
<td>L.P.</td>
<td>3007 cc</td>
<td>47 cc</td>
<td>609 cc</td>
<td>2351 cc</td>
<td>59.1 Kg</td>
<td>24.5 mm. Hg.</td>
<td>2.08 cc/kg/mm.</td>
<td>1.62 cc/kg/mm.</td>
</tr>
<tr>
<td>J.T.</td>
<td>1939 cc</td>
<td>57 cc</td>
<td>667 cc</td>
<td>1215 cc</td>
<td>78.6 Kg</td>
<td>17.8 mm. Hg.</td>
<td>1.39 cc/kg/mm.</td>
<td>0.87 cc/kg/mm.</td>
</tr>
<tr>
<td>A.H.</td>
<td>1211 cc</td>
<td>56 cc</td>
<td>518 cc</td>
<td>637 cc</td>
<td>62.7 Kg</td>
<td>22.2 mm. Hg.</td>
<td>0.87 cc/kg/mm.</td>
<td>0.46 cc/kg/mm.</td>
</tr>
<tr>
<td>K.K.</td>
<td>1991 cc</td>
<td>60 cc</td>
<td>506 cc</td>
<td>1425 cc</td>
<td>75.0 Kg</td>
<td>19.7 mm. Hg.</td>
<td>1.35 cc/kg/mm.</td>
<td>0.97 cc/kg/mm.</td>
</tr>
<tr>
<td>J.S.</td>
<td>2590 cc</td>
<td>59 cc</td>
<td>717 cc</td>
<td>1814 cc</td>
<td>84.1 Kg</td>
<td>19.3 mm. Hg.</td>
<td>1.59 cc/kg/mm.</td>
<td>1.11 cc/kg/mm.</td>
</tr>
<tr>
<td>D.S.</td>
<td>4576 cc</td>
<td>86 cc</td>
<td>1017 cc</td>
<td>3473 cc</td>
<td>88.2 Kg</td>
<td>36.9 mm. Hg.</td>
<td>1.41 cc/kg/mm.</td>
<td>1.07 cc/kg/mm.</td>
</tr>
<tr>
<td>G.L.</td>
<td>2164 cc</td>
<td>68 cc</td>
<td>541 cc</td>
<td>1555 cc</td>
<td>72.7 Kg</td>
<td>25.4 mm. Hg.</td>
<td>1.17 cc/kg/mm.</td>
<td>0.57 cc/kg/mm.</td>
</tr>
<tr>
<td>J.P.</td>
<td>2877 cc</td>
<td>40 cc</td>
<td>601 cc</td>
<td>2236 cc</td>
<td>90.9 Kg</td>
<td>14.7 mm. Hg.</td>
<td>2.15 cc/kg/mm.</td>
<td>1.67 cc/kg/mm.</td>
</tr>
<tr>
<td>J.M.</td>
<td>2300 cc</td>
<td>65 cc</td>
<td>520 cc</td>
<td>1715 cc</td>
<td>72.7 Kg</td>
<td>18.5 mm. Hg.</td>
<td>1.71 cc/kg/mm.</td>
<td>1.27 cc/kg/mm.</td>
</tr>
<tr>
<td>G.H.</td>
<td>3685 cc</td>
<td>53 cc</td>
<td>575 cc</td>
<td>3057 cc</td>
<td>73.6 Kg</td>
<td>20.1 mm. Hg.</td>
<td>2.50 cc/kg/mm.</td>
<td>2.07 cc/kg/mm.</td>
</tr>
<tr>
<td>D.C.</td>
<td>3564 cc</td>
<td>66 cc</td>
<td>636 cc</td>
<td>2862 cc</td>
<td>77.3 Kg</td>
<td>20.5 mm. Hg.</td>
<td>2.25 cc/kg/mm.</td>
<td>1.81 cc/kg/mm.</td>
</tr>
<tr>
<td>F.M.</td>
<td>2171 cc</td>
<td>51 cc</td>
<td>345 cc</td>
<td>1775 cc</td>
<td>56.8 Kg</td>
<td>20.7 mm. Hg.</td>
<td>1.84 cc/kg/mm.</td>
<td>1.51 cc/kg/mm.</td>
</tr>
</tbody>
</table>

\[ \bar{x} = \text{Mean} \]

\[ \bar{x} = 1.7 \quad 1.2 \]
### TABLE XIV

**CO₂ STORES GAIN DURING RECOVERY**

<table>
<thead>
<tr>
<th>Subj.</th>
<th>Total Stores CO₂ Gain</th>
<th>Lung CO₂ Gain</th>
<th>Blood CO₂ Gain</th>
<th>Tissue CO₂ Gain</th>
<th>Body Weight Kg</th>
<th>PaCO₂ mm. Hg.</th>
<th>Whole Body Dissociation Slope cc/kg/mm.</th>
<th>Tissue Dissociation Slope cc/kg/mm.</th>
</tr>
</thead>
<tbody>
<tr>
<td>M. C.</td>
<td>17</td>
<td>208</td>
<td>70.5</td>
<td>8.4</td>
<td>0.76</td>
<td>0.10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>E. C.</td>
<td>51</td>
<td>652</td>
<td>79.5</td>
<td>13.4</td>
<td>0.61</td>
<td>0.22</td>
<td></td>
<td></td>
</tr>
<tr>
<td>L. P.</td>
<td>16</td>
<td>168</td>
<td>59.1</td>
<td>8.0</td>
<td>0.78</td>
<td>0.34</td>
<td></td>
<td></td>
</tr>
<tr>
<td>J. T.</td>
<td>42</td>
<td>567</td>
<td>78.6</td>
<td>14.7</td>
<td>0.90</td>
<td>0.37</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A. H.</td>
<td>54</td>
<td>519</td>
<td>62.7</td>
<td>20.3</td>
<td>0.80</td>
<td>0.35</td>
<td></td>
<td></td>
</tr>
<tr>
<td>K. K.</td>
<td>60</td>
<td>589</td>
<td>84.1</td>
<td>12.1</td>
<td>0.53</td>
<td>0.12</td>
<td></td>
<td></td>
</tr>
<tr>
<td>J. S.</td>
<td>31</td>
<td>388</td>
<td>88.2</td>
<td>25.5</td>
<td>0.51</td>
<td>0.13</td>
<td></td>
<td></td>
</tr>
<tr>
<td>D. S.</td>
<td>66</td>
<td>793</td>
<td>90.9</td>
<td>9.8</td>
<td>0.39</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>G. L.</td>
<td>54</td>
<td>517</td>
<td>72.7</td>
<td>24.0</td>
<td>0.50</td>
<td>0.17</td>
<td></td>
<td></td>
</tr>
<tr>
<td>J. P.</td>
<td>33</td>
<td>423</td>
<td>90.9</td>
<td>9.8</td>
<td>0.91</td>
<td>0.51</td>
<td></td>
<td></td>
</tr>
<tr>
<td>J. M.</td>
<td>37</td>
<td>378</td>
<td>72.7</td>
<td>14.1</td>
<td>0.63</td>
<td>0.19</td>
<td></td>
<td></td>
</tr>
<tr>
<td>G. H.</td>
<td>41</td>
<td>398</td>
<td>73.6</td>
<td>13.5</td>
<td>0.89</td>
<td>0.50</td>
<td></td>
<td></td>
</tr>
<tr>
<td>D. C.</td>
<td>44</td>
<td>457</td>
<td>77.3</td>
<td>16.5</td>
<td>0.86</td>
<td>0.47</td>
<td></td>
<td></td>
</tr>
<tr>
<td>F. M.</td>
<td>48</td>
<td>350</td>
<td>56.8</td>
<td>18.2</td>
<td>0.7</td>
<td>0.3</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

\[x = \text{Mean}\]
# TABLE XV

## PARTITION OF CO$_2$ RETENTION, BLOOD AND TISSUES**

<table>
<thead>
<tr>
<th>Group</th>
<th>CO$_2$ Loss from body stores*</th>
<th>CO$_2$ Stores regained by body*</th>
<th>CO$_2$ Stores regained by blood</th>
<th>CO$_2$ Stores regained by tissues</th>
<th>% of Total CO$_2$ regained (by blood)</th>
<th>% of Total CO$_2$ regained (by tissues)</th>
<th>% of Lost Blood CO$_2$ Content Regained</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>2891</td>
<td>289 (10%)</td>
<td>299 (10%)</td>
<td>0 (0)</td>
<td>100%</td>
<td>0</td>
<td>49%</td>
</tr>
<tr>
<td>II</td>
<td>3159</td>
<td>881 (28%)</td>
<td>497 (16%)</td>
<td>384 (12%)</td>
<td>56%</td>
<td>44%</td>
<td>76%</td>
</tr>
<tr>
<td>III</td>
<td>1964</td>
<td>917 (47%)</td>
<td>537 (27%)</td>
<td>380 (19%)</td>
<td>59%</td>
<td>41%</td>
<td>94%</td>
</tr>
</tbody>
</table>

* Excluding Lung Air

** Calculations From Mean Data
TABLE XVI

CO$_2$ DISSOCIATION SLOPES OF THE BODY (4)

<table>
<thead>
<tr>
<th>Species</th>
<th>Duration of Experiment (min.)</th>
<th>Slope (cc/kg/mm.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Man</td>
<td>1-5</td>
<td>0.46</td>
</tr>
<tr>
<td>Man</td>
<td>3-8</td>
<td>0.40</td>
</tr>
<tr>
<td>Man</td>
<td>2-3</td>
<td>0.50</td>
</tr>
<tr>
<td>Man</td>
<td>33</td>
<td>2.10</td>
</tr>
<tr>
<td>Man</td>
<td>30</td>
<td>3.8</td>
</tr>
<tr>
<td>Man</td>
<td>20</td>
<td>1.30</td>
</tr>
<tr>
<td>Man</td>
<td>60</td>
<td>2.05</td>
</tr>
<tr>
<td>Dog</td>
<td>30-45</td>
<td>1.50</td>
</tr>
<tr>
<td>Cat</td>
<td>30-90</td>
<td>1.60</td>
</tr>
<tr>
<td>Cat</td>
<td>100</td>
<td>1.80</td>
</tr>
<tr>
<td>Rat</td>
<td>Several Weeks</td>
<td>Up to 11.6</td>
</tr>
</tbody>
</table>
Figure 1. Dashed line is Gray's respiratory pathway of acid-base disturbance (9). The dots represent the data of Tomashefski, Carter and Lipsky (6) obtained from 10 subjects during 12 minutes of hyperventilation. Read from right to left.

Figure 2. The pH-bicarbonate diagram with PCO2 isobars. Mean data of 10 subjects, with standard deviations obtained during 12 minutes of hyperventilation. The in vitro buffer line of Henderson (32) for oxygenated blood lies above the in vivo pathway. Data of Tomashefski, Carter and Lipsky (6).

Figure 3. The mean values of calculated and measured arterial CO2 tensions, end-tidal CO2 tension and [H+] obtained during 12 minutes of hyperventilation. Data of Tomashefski, Carter and Lipsky (6).

Figure 4. Mean CO2 elimination per breath during 12 minutes of hyperventilation. X—X represents the mean basic CO2 elimination per breath. Data of Tomashefski, Carter and Lipsky (6).

Figure 5. Semi-logarithmic plot of mean CO2 elimination per breath during 12 minutes of hyperventilation. Data of Tomashefski, Carter and Lipsky (6).
Figure 1.

Figure 2.

Figure 3.

Figure 4.

Figure 5.
Figure 6. Schematic diagram of directional breathing valve. A, inspiratory line flap valve; B, mouthpiece; C, expiratory line flap valve; D, expiratory line pressure-loaded valve.

Figure 7. Schematic diagram of apparatus. By appropriate positioning of the 3 directional valves, inspirations and expirations can be made to proceed either from A (room air) to B (low resistance flap valve) to C (flowmeter), or from D (compressed air) to E (pressure-loaded valve) to C (flowmeter).
Figure 6.

Figure 7.
Figure 8. The directional valve used to direct expired air in either of two directions by means of a three-way stopcock arrangement.
Figure 9. Gray's respiratory pathway equation is illustrated by the dashed line. Individual results of arterial blood analyses of (H+) and Pa$_{CO_2}$ are shown to group themselves around the theoretical line.
Figure 10. The pH-bicarbonate diagram with $\text{PCO}_2$ isobars. The dashed line indicates in vitro data for oxygenated true plasma, by Henderson. The solid lines denote the in vivo experimental results obtained during 12 minutes of recovery from hyperventilation. Recovery proceeds from right to left.
Figure 11. The average results of Figure 10, with standard deviations. The • represents the average control point, and the average experimental results are denoted by ▲. Read from right to left.
Figure 12. The total CO₂ elimination of 14 subjects during 12 minutes of recovery from acute hyperventilation. The dashed line indicates the mean integrated basic CO₂ production under resting conditions.

Figure 13. The mean resting CO₂ production over 12 minutes (A); the mean accumulative total CO₂ elimination during recovery (B); and the mean accumulative total CO₂ retention during recovery (C).
Figure 14. Whole body stores retention of CO$_2$ during 12 minutes of recovery from hyperventilation; individual results of 14 subjects.
Figure 15. The per cent of the whole body stores lost during 12 minutes of hyperventilation which are regained during 12 minutes of recovery (14 subjects).
Figure 16. Group I. The changes in arterial pH, oxygen tension and carbon dioxide tension, and the percent of the lost CO₂ stores which are regained during recovery from hyperventilation. The dashed extension of the CO₂ stores curve indicates the most probable position of this curve, considering that an extremely low contribution to this group was dropped after 8 minutes of recovery. N = Control; O = end of hyperventilation.
Figure 17. Group II. The changes in arterial pH, oxygen tension and carbon dioxide tension, and the per cent of the lost CO₂ stores which are regained during recovery from hyperventilation. N = Control; O = End of hyperventilation.
Figure 18. Group III. The changes in arterial pH, oxygen tension and carbon dioxide tension, and the percent of the lost CO₂ stores which are regained during recovery from hyperventilation. N = Control; O = End of hyperventilation.
Figure 19. Group I. The dashed lines indicate frequency, tidal volume, and minute ventilation changes from control to end-hyper-ventilation; the solid lines denote the behavior of these variables during recovery. ($V_E$ in BTPS).
Figure 20. Group II. The dashed lines indicate frequency, tidal volume, and minute ventilation changes from control to end-hyper-ventilation; the solid lines denote the behavior of these variables during recovery. ($V_E$ in BTPS).
Figure 21. Group III. The dashed lines indicate frequency, tidal volume, and minute ventilation changes from control to end-hyper-ventilation; the solid lines denote the behavior of these variables during recovery. ($V_E$ in BTPS).
Figure 22. The loss and restoration of CO$_2$ stores are represented as a percent of the total stores which were lost. No attempt is made to identify a group with the loss of CO$_2$ during hyperventilation, although each group is plotted separately. The maximum extent of the standard deviations are indicated by the dashed lines. During the recovery phase, means and standard deviations of each group are plotted. The dashed portion of the Group I curve represents the most probable extension of the mean data, since an extremely low contribution to this group was lost after 8 minutes of recovery.
Figure 22.
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I, Joseph Albin Lipsky, was born in Glen Lyon, Pennsylvania, March 31, 1930. I received my secondary-school education in the public schools of Newport Township, Pennsylvania, and my undergraduate training at Lycoming College and Pennsylvania State University, the latter granting me the Bachelor of Science degree in 1951. From the Ohio State University, where I specialized in physiology, I received the Master of Science degree in 1959. I was appointed to various laboratory teaching assistantships in the Department of Physiology, and I was research assistant to Dr. Joseph F. Tomashefski, Ohio Tuberculosis Hospital. I held these positions for three years while completing the requirements for the Doctor of Philosophy degree.