THE USE OF FREE-FLOWING PERIPHERAL VENOUS BLOOD IN THE ASSESSMENT OF RESPIRATORY ACID-BASE DISTURBANCES

by

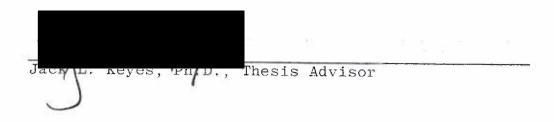
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CHAPTER I

INTRODUCTION

Determination of the arterial blood-gas composition (blood-gas analysis) is essential to the assessment of both cardiopulmonary function and acid-base status in the critically ill patient. Clinically, blood-gas analysis is most often used to measure arterial pH, pCO₂ and pO₂.

Arterial blood has traditionally been used in blood-gas analysis for two reasons: 1) its composition is generally uniform throughout the body, and 2) its oxygen content reflects the efficacy of pulmonary exchange (Slonim & Hamilton, 1976).

Venous blood is generally not used for blood-gas analysis. The primary reason for not using venous blood is that the blood-gas composition varies from one venous sampling site to another. At least two important factors contribute to this variance: 1) metabolic activity varies from one tissue to the next, and 2) blood flow also varies in different tissues and organs. This variance in metabolic activity and blood flow may be more pronounced in tissues with metabolic disorders or with damage to the vascular supply (Goldschmidt & Light, 1925; Harrison & Galloon, 1965; Paine, Boutwell & Soloff, 1961).

Cardiopulmonary and acid-base status of the critically ill patient may change very rapidly. Thus, repeated

sampling of arterial blood is frequently necessary in these cases. However, arterial samples may be difficult to obtain, particularly in infants, young children and those patients with extensive burns (Gambino, 1961; Hofford, Dowling & Pell, 1973; Lilienthal & Riley, 1944; McIntyre, Norman & Smith, 1968). In addition, adult patients who have previously undergone multiple arterial punctures may develop thromboses, further complicating the collection of arterial blood (Gambino, 1961; Nicholls, 1964). Such patients may have acute or chronic oxygenation abnormalities as occurs in severe asthma. obstructive pulmonary disease and congestive heart Patients with chronic metabolic diseases, such failure. as uncontrolled diabetes mellitus, and those with acute metabolic disorders, such as acidosis due to salicylate overdose, may also require repeated arterial punctures.

Complications of Arterial Puncture

Arterial puncture may lead to a variety of complications, depending upon the age of the individual, the site of sampling and the number of samples obtained. The collection of arterial blood is often quite painful, and may cause arterial spasm or damage to the vessel wall (Goldschmidt, 1925; Neviaser, 1976; Stern, Kaplan & Furman, 1973). Ecchymosis and hematoma formation may

occur due to extravasation of blood, particularly with repeated sampling (Cole & Lumley, 1966). Collection of arterial blood from infants may cause excessive blood loss (Hofford, 1973).

Various types of nerve injury have been reported as a result of arterial puncture. According to Pape (1978), brachial artery puncture in both children and adults may lead to median nerve damage. This damage was also observed in low birth-weight infants subjected to repeated brachial artery sampling. Two instances of carpal tunnel syndrome secondary to radial artery puncture in neonates were also documented. Carpal tunnel syndrome is evidenced by nocturnal paresthesias and pain in the fingers, wrist and forearm. It may progress to weakness and complete sensory loss in the affected areas (Wintrobe, M. W.; Thorne, G. W.; Adams, R. D.; Braunwald, E.; Isselbacher, K. J. & Petersdorf, R. G., 1974).

Patients receiving anticoagulant therapy appear to be at high risk of complications to arterial puncture.

Macon & Futrell (1973) reported median nerve neuropathy secondary to spontaneous hemorrhage following brachial artery puncture. Neuropathies of the femoral, obturator and sciatic nerves have also been documented in anticoagulated patients (Macon & Futrell, 1973; Neviaser, 1976). In a study by Neviaser in 1976, brachial and

femoral artery puncture in patients receiving heparin produced hematomas which eventually caused skin sloughing or infection. Incision and drainage, or decompression of infected hematomas may be necessary in such cases.

Neviaser also described ischemic contracture and necrosis of the forearm following brachial artery puncture in patients receiving heparin.

Thus, collection of arterial blood for blood-gas analysis may lead to a number of undesirable complications, particularly when repeated sampling is necessary.

In contrast to arterial blood, venous blood is generally easier to obtain, and in some cases, may even be available from a previously placed intravenous catheter.

Additionally, fewer hazards and complications are associated with venipuncture as opposed to arterial puncture.

It appears that the use of venous blood for blood-gas analysis in certain patients may prove to be safer than the use of arterial blood. The basis underlying the substitution of venous blood for arterial blood is illustrated by the following model.

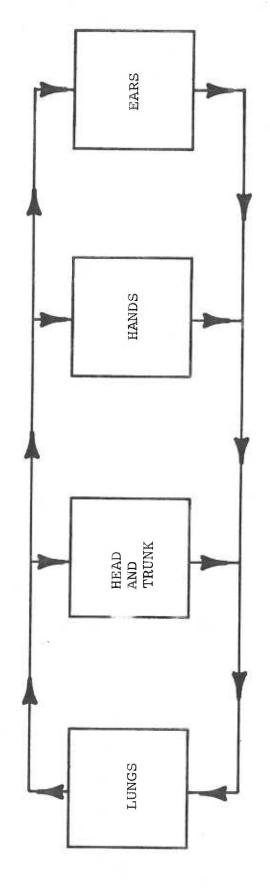
Model

The relationship between arterial and venous blood, as well as the direction of blood flow in each case, may be demonstrated by the following model.

It can be seen from Figure 1 that arterial blood

ARTERIAL BLOOD

Input: to organs and systemic tissues from the lungs



VENOUS BLOOD

from the organs and systemic tissues to the lungs Output:

Head, hands and ears The direction of flow of arterial and venous blood. represent some of the existing parellel circuits. Figure 1.

(Revised from Carveth, 1979)

and venous blood flow in opposite directions in the body. Arterial blood represents an input flow to the organs and tissues from the pulmonary circulation. The organs and tissues can be considered to be parallel circuits within the systemic circulation. Hence, arterial blood flows from the pulmonary circulation to the systemic circulation. In the model, head, hands and ears represent parallel circuits in the systemic circulation.

Venous blood is seen as an output flow from systemic tissues to the lungs, or pulmonary circulation. Arterial blood and venous blood differ in terms of blood-gas composition. In the lungs, ${\rm O_2}$ and ${\rm CO_2}$ are exchanged. Upon leaving the lungs, arterial blood has received ${\rm O_2}$, and had ${\rm CO_2}$ removed. As the arterial blood flows through the parallel circuits of the systemic circulation, ${\rm O_2}$ is taken up by the organs and tissues and ${\rm CO_2}$ is added to the blood. Thus, venous blood flowing back to the lungs generally has a ${\rm pO_2}$ lower than, and a ${\rm pCO_2}$ higher than that of arterial blood.

The composition of arterial blood is generally uniform throughout the various parallel circuits of the
systemic circulation. In contrast, the composition of
venous blood varies in relation to the metabolic activity
and blood flow to the systemic tissues. This relationship

is illustrated by the following equations taken from Carveth (1979):

$$\dot{Q}$$
 $[O_2]_a = \dot{Q}$ $[O_2]_v + \dot{V}O_2$ (1) where \dot{Q} = flow $\dot{V}O_2$ = oxygen consumption, $[O_2]_a$ = the concentration of oxygen in the arteries, and $[O_2]_v$ = the concentration of oxygen in the veins. \dot{Q} $[O_2]_a$ - \dot{Q} $[O_2]_v$ = $\dot{V}O_2$ (2) \dot{Q} $([O_2]_a$ - $[O_2]_v$) = $\dot{V}O_2$ (3) $[O_2]_a$ - $[O_2]_v$ = $\dot{V}O_2$ (4)

Equation 4 shows that as flow (\dot{Q}) increases, the difference between arterial and venous O_2 concentration will decrease. The O_2 uptake is proportional to the metabolic activity of the organ or tissue. It can be seen that the relationship between flow (\dot{Q}) and metabolic activity is an important one. If the venous blood flow is increased above the metabolic activity of a particular tissue, then the O_2 concentration of the venous blood leaving that tissue will more closely approximate that of arterial.

Similarly, this relationship applies to ${\rm CO}_2$ and ${\rm [H+]}$,

as illustrated by the following equations:

$$[co_2]_v$$
 - $[co_2]_a = \frac{\dot{v}co_2}{\dot{o}}$ (5) where

 $\dot{V}CO_2 = CO_2$ production,

 $[CO_2]_a$ = the concentration of CO_2 in the arteries, and

 $[CO_2]_v$ = the concentration of CO_2 in the veins

$$[H^{+}]_{V}$$
 - $[H^{+}]_{a}$ = $\underline{H+ production}$ (6) where

[H⁺]_a = the concentration of acid in the arteries, and

 $[H^{\dagger}]_{V}$ = the concentration of acid in the veins.

It is of interest to note that these same equations apply when mixed venous instead of peripheral venous blood is used. In this instance, the flow term, \dot{Q} , is cardiac output.

In blood-gas analysis, measures which are proportional or related mathematically to the concentration of $\rm O_2$, $\rm CO_2$ and H+ are generally made. These measurements are the $\rm pO_2$, $\rm pCO_2$ and pH, respectively.

Arterialized Venous Blood

As discussed previously, venous blood is generally easier to obtain than arterial blood. In addition, the collection of venous blood has been associated with fewer hazards and complications. Venous blood may prove to be

a safe, relatively reliable substitute for arterial blood in blood-gas analysis. Some investigators have stated that venous blood will more closely approximate arterial blood in terms of blood-gas composition, if the sampling site is "arterialized" prior to collection (Brooks, 1959: Collis & Neaverson, 1967; Goldschmidt & Light, 1925; Harrison & Galloon, 1965). Arterialization may be produced by gently warming the tissues surrounding the venous sampling site. Elevated temperature leads to vasodilation of the vessels, causing increased blood flow (Q) to the area. According to Collis and Neaverson (1967), the temperature must reach 35-45° C. in order for arterialization to occur. The increased flow (Q) in relation to the metabolic activity of the tissue will cause the venous blood composition to be more similar to that of arterial blood.

The first reference regarding the use of arterialized venous blood occurred in 1920 when Meakin and Davies employed the use of a 45° C. water bath to produce arterialization. Similarly, other researchers have also used a water bath, ranging in temperature from 46-47° C.(Collis & Neaverson, 1967; Gambino, 1961; Goldschmidt & Light, 1925; Paine, Boutwell & Soloff, 1961). The hand to be used in sampling was placed into the water bath for approximately 5 to 20 minutes, depending upon the length of time required

to raise the temperature of the site to $35-45^{\circ}$ C.

Three other methods have also been described in the literature as a means of producing arterialization of the venous sampling site: 1) Warming the hand or extremity with a hot towel (Paine, Boutwell & Soloff, 1961).

2) Heating the surrounding tissues with a hot water bottle (Hofford, Dowling & Pell, 1973). 3) Wrapping the extremity in an electric heating pad set at 40 to 60° C.

In the following study, I compared the blood-gas composition of free-flowing peripheral venous blood, both arterialized and non-arterialized, to that of arterial blood in acute respiratory acid-base disturbances. Since the equations in the model described above apply to mixed venous blood (when cardiac output is substituted for \dot{Q}), I have chosen to also compare the blood-gas composition of mixed venous blood to arterialized and non-arterialized peripheral venous blood.

CHAPTER II

REVIEW OF THE LITERATURE

Arterialized Venous Blood

Meakin and Davies (1920) examined the oxygen (0)₂ saturation of hemoglobin in the arterial and venous blood of one patient. Of particular importance in their study was the effect of varying the temperature of the tissues at the sampling site. Paired arterial and venous samples were collected from the forearm under five different temperature conditions: 1) exposed to room air, 2) exposed to cool atmosphere, 3) exposed to cold atmosphere, 4) arm immersed in water bath at 45° C. for 10 minutes prior to sampling, and 5) arm immersed in water bath at 45° C for 20 minutes prior to sampling. It should be noted that neither method of cooling nor the skin temperature at the time of sampling were specified.

Results of this study indicated that arterial O_2 saturation remained relatively constant at about 96.1 per cent, despite changes in local temperature. In contrast, venous O_2 saturation varied substantially in response to temperature changes. When the sampling site was exposed to room air, venous O_2 saturation was found to be 56.4 per cent. However, upon exposure to progressively decreasing temperatures of tissues supplying blood to the sampling site, the venous O_2 saturation declined initially to

34.9 per cent, and then to 0.0 per cent. Warming of the sampling site for 10 and 20 minute intervals produced a venous $\mathbf{0}_2$ saturation of 94.2 per cent. Meakin and Davies (1920) concluded that the $\mathbf{0}_2$ saturation of venous blood does not accurately indicate that of arterial blood when the temperature of tissues at the sampling site are lowered. Interestingly, they did not comment on the fact that warming the sampling site of the subject caused the venous $\mathbf{0}_2$ saturation to more closely approximate that of arterial.

Goldschmidt and Light (1925) explored the possibility of using arterialized venous blood rather than arterial blood when determining oxygen content (vol. %), oxygen capacity of hemoglobin (vol. %), O₂ saturation and carbon dioxide content (vol. %). (Terms are those used by authors).

Arterialization of the venous sampling site was achieved by placing the hand and wrist in a 45-47° water bath for 10 minutes. Venous samples were then obtained from the dorsal surface of the hand. Arterial samples were drawn from the radial or brachial arteries.

In this experiment, paired samples of arterial and venous blood from six subjects were compared. Four of the subjects were considered healthy, while the other two were hospitalized patients. Oxygen content of the arterial samples ranged from 14.56 vol. % to 20.33 vol. %.

Arterialized venous values of O₂ content were similar to those of arterial blood, ranging from 16.03 vol. % to 20.08 vol. %, respectively. It is of interest to note that in one subject, arterialized venous blood was found to have a higher O₂ content than arterial blood. The reason given by the investigators for this finding was that the patient had a pulmonary stenosis with a patent ductus arteriosus. In addition, it was observed that the patient was exceedingly disturbed and held her breath at the time of arterial sampling.

Arterialized venous values of CO_2 content also closely resembled those of arterial blood, ranging from 29.7 vol. % to 46.7 vol. %. Arterial CO_2 content ranged from 31.4 vol. % to 47.5 vol. %. Goldschmidt and Light (1925) similarly found the O_2 capacity of hemoglobin and O_2 saturation to be virtually the same as that of arterial blood.

Brooks and Wynn (1959) studied the pH and pCO₂ of arterial and venous blood in respiratory failure and during anesthesia. Both arterialized (warmed) and non-arterialized (unwarmed) venous samples were collected for comparison. Arterialization in this study was performed by loosely wrapping the hand and arm in two heating pads for 15 minutes. When the skin temperature reached between 35°and 38°C., venous blood was collected from the dorsum

of the hand or wrist. Arterial samples were obtained by brachial or femoral artery puncture.

These investigators studied three different groups of subjects: ambulatory patients, patients confined to bed and anesthetized patients. The ambulatory group consisted of five patients. Results of blood-gas analysis for pH and pCO $_2$ in the ambulatory group revealed small differences in pH and pCO $_2$ between arterialized venous blood and arterial blood. The mean arteriovenous difference for pH was 0.018 pH units, while the mean difference for pCO $_2$ was 3.5 mm Hg. (Standard deviations were not reported in this study; insufficient data were available for this computation). Greater arteriovenous differences were found between non-arterialized venous blood and arterial blood. These differences for pH and pCO $_2$ were 0.061 pH units and 9.6 mm Hg, respectively.

The second group included nine patients, all of whom had been confined to bed prior to the test. Arteriovenous differences were found to be even less in this group. A comparison of arterialized venous blood and arterial blood showed a mean pH difference of 0.002 pH units and a mean pCO₂ difference of 0.8 mm Hg. Mean arteriovenous differences between non-arterialized venous blood and arterial blood were 0.045 pH units and 6.7 mm Hg pCO₂.

The anesthetized patients undergoing surgical pro-

cedures comprised the third group. General anesthesia commonly produces vasodilatation and an increase in peripheral blood flow. Hence, the warming procedure to promote arterialization of the venous sampling site was not employed in this group. Skin temperature remained above 35° C. due to spontaneous arterialization. Mean arteriovenous differences for pH and pCO₂ were again negligible: 0.002 pH units and 1.1 mm Hg pCO₂.

Brooks and Wynn (1959) also compared the pO_2 and O_2 saturation of venous blood (arterialized and non-arterialized) to that of arterial blood in 20 of the patients. They reported that significant differences in pO_2 and O_2 saturation were found, although the exact values were not documented. From their findings, these researchers postulated that if the temperature used to produce arterialization were higher, the arteriovenous pO_2 and O_2 saturation difference would be less.

These researchers concluded that under certain conditions, peripheral venous blood may be a reliable substitute for arterial blood in terms of pH and pCO_2 . The required conditions included a skin temperature over the venous sampling site of at least 35° C. and a recumbent subject.

Paine, Boutwell and Soloff (1961) also investigated the use of arterialized venous blood in lieu of arterial blood for measurement of pH and pCO_2 . Venous blood was

arterialized by wrapping the dorsum of the hand in a hot towel or placing it in a container of hot water for 15 to 20 minutes prior to sampling. Temperatures of the water bath and the skin at the sampling site were not reported. All subjects had been recumbent for approximately one and one-half hours before the blood was collected. Arterial blood was drawn as a control at the same time the arterialized venous samples were obtained. Twenty-nine hospitalized patients were used in this study. Three different techniques for sampling were employed. 1) In six subjects, a tourniquet was placed above the elbow and the blood was collected into a 10-ml vacuum tube. 2) In nine of the subjects, a tourniquet was again placed above the elbow prior to sampling, but the blood was collected into heparinized syringes. 3) In the remaining 14 subjects, a tourniquet was applied at the wrist. Venesection was performed to obtain venous samples on half of these subjects. other half were encouraged to flex their fingers prior to collection of venous blood. Arterial samples were all collected into heparinized syringes.

The following results were reported: 1) The use of vacuum tubes for collection of arterialized venous blood yielded the greatest arteriovenous differences in pH and pCO_2 . The mean arteriovenous difference for pH using this

method was 0.050 pH units and the mean pCO_2 difference was 4.9 mm Hg. (Standard deviations were not reported in this study; insufficient data were available for this computation). 2) The second group in which a tourniquet was placed above the elbow and heparinized syringes were used showed mean arteriovenous differences in pH of 0.038 pH units and in pCO_2 of 8.5 mm Hg. 3) The last group with constriction at the wrist and use of heparinized syringes revealed the least mean arteriovenous pH and pCO_2 differences. These mean differences were 0.022 pH units and 2.86 mm Hg pCO_2 .

Paine, Boutwell and Soloff (1961) drew two major conclusions from their investigation.

- 1) Arterialized venous blood as an estimate of arterial pH may be best obtained by: warming the dorsum of the hand for 20 minutes, placing a tourniquet at the wrist and requesting the subject to flex the fingers. Samples of arterialized venous blood obtained by this method are a reliable estimate of arterial pH.
- 2) Arterialized venous blood obtained by the method previously described has, on the average, a pH reading 0.022 pH units and pCO₂ reading 2.86 mm Hg lower than that of arterial blood.

Unfortunately, these investigators failed to draw any

conclusions on the usefulness of arterialized venous blood as an indication of arterial pCO_2 . However, their findings do indicate that arterialized venous blood, when collected with constriction at the wrist, is a reasonable estimate of arterial blood in terms of pCO_2 .

Harrison and Galloon (1965) investigated the use of venous blood as an alternative to arterial blood for the measurement of pCO₂. The pH and pO₂ were not addressed in this study. Careful examination of the previous investigation conducted by Paine, Boutwell and Soloff (1961) led them to believe that causing venous obstruction (by applying a tourniquet prior to venous sampling) might profoundly be affecting the results obtained. Harrison and Galloon studied 13 patients, 12 of whom were undergoing surgery and one patient who was being artifically ventilated.

Venous blood samples were obtained from an indwelling cannula placed on the dorsum of the hand. Arterial samples were drawn from the same extremity within 30 seconds of when the venous samples were drawn. Arterialization of the venous sampling site was accomplished by wrapping the hand in an electric heating pad. This pad had a maximum temperature of 60°C. Venous samples were obtained under the following conditions:

1) Non-arterialized at the beginning of anesthesia:

- (a) with venous obstruction and (b) without venous obstruction.
- Non-arterialized during anesthesia: (a) with venous obstruction and (b) without venous obstruction.
- Arterialized during anesthesia (with the hand at body temperature): (a) with venous obstruction and(b) without venous obstruction.

Arterial samples were drawn concurrently for comparison.

One hundred and forty-six pairs of venous and arterial samples were drawn from the 13 patients.

Results of their study surprisingly showed the smallest arteriovenous differences in pCO_2 to occur in those samples collected at the beginning of anesthesia, without arterialization of the venous sampling site. Venous obstruction was present when these samples were taken. The pCO_2 values of these samples were not reported. These investigators presumed that the small arteriovenous differences in pCO_2 found in this group of samples was caused by a spontaneous arterialization from peripheral vasodilatation accompanying the induction of anesthesia.

Harrison and Galloon (1965) reported the results obtained on 51 pairs of samples collected from 11 of the patients. The venous samples in this group were drawn from a warm hand (temperature was not stated), and venous obstruction was not present. The mean arteriovenous differ-

ence for ${\rm pCO}_2$ was 0.5 mm Hg (at 40 mm Hg ${\rm pCO}_2$) with a standard deviation of 0.7 mm Hg (at 40 mm Hg ${\rm pCO}_2$). It should be noted that there was no arteriovenous ${\rm pCO}_2$ difference greater than 2 mm Hg. From their study, these investigators concluded that venous blood obtained under specified "ideal" conditions will have a ${\rm pCO}_2$ identical to or very similar to that of arterial blood. These "ideal" conditions include: 1) the venous blood must be drawn from the dorsum of the hand, 2) the venous blood must be arterialized by warming the hand to at least body temperature, and 3) there must be no venous obstruction before or during the sampling procedure. When these conditions were met in their study, the arteriovenous difference for ${\rm pCO}_2$ did not exceed 2 mm Hg.

Collis and Neaverson (1967) determined the differences in pH, pCO₂ and O₂ saturation between arterialized venous blood and arterial blood. Venous sampling sites were arterialized by placing the subject's hand in a 45° C. water bath for 5 minutes. Arterialized venous samples were then obtained from a vein on the dorsum of the hand. Venous obstruction was not present. Arterial samples were then taken from the radial artery. All samples were collected anerobically into heparinized syringes and analyzed immediately. Twenty-three conscious, mainly ambulatory patients were studied. Samples were obtained successively; in 10 subjects, the arterialized venous sample was drawn first and in 13 subjects the arterial

sample was drawn first. Hence, a total of 23 pairs of samples were obtained for analysis. Results showed the mean arteriovenous difference in pH to be 0.0052 pH units (SD 0.0075), while the mean difference for pCO_2 was 0.76 mm Hg (SD 0.81). Upon completion of their investigation, Collis and Neaverson compared the two groups in which the order of sampling had been reversed. No significant difference was observed between the two groups. When the 23 pairs of samples were analyzed for pO_2 and O_2 saturation, substantial arteriovenous differences were noted. The mean arteriovenous difference for O2 saturation was 2.53% (SD 1.88). Precise values obtained for pO_2 were not available. However, it was mentioned that some samples differed in pO_2 by as much as 40 mm Hg. At arterial pO_2 values above 60 mm Hg., the arterialized venous pO_2 was of no value in estimating arterial pO2

On the basis of their results, these authors decided that arterialized venous blood may be a useful estimate of the pH and pCO_2 in arterial blood. They also concluded that arterialized venous blood was not a reliable indicator of arterial pO_2 or O_2 saturation.

Forster, Dempsey, Thomson, Vidruk and DoPico (1972) compared the pH, pCO₂, pO₂ and lactate values of arterial-ized venous blood and arterial blood from human subjects.

Venous samples were obtained from an indwelling "butterfly"

needle inserted into one of the superficial dorsal hand veins. Prior to venous sampling, the entire hand was warmed by an electric heating pad for 10 minutes. Skin temperature varied from 41-43°C. as determined via monitoring the skin temperature. A tourniquet was not used, thus all venous samples were collected from a free-flowing vein. Arterial samples were drawn from an indwelling Teflon catheter placed in a brachial artery. All samples were collected anaerobically into heparinized syringes.

Thirteen male subjects were used in this study. These were composed of five healthy young adults and eight middle-aged patients displaying symptoms of exertional dyspnea. Comparison of arterialized venous blood and arterial blood pH, pCO₂, pO₂ and lactate were made under four different conditions: 1) normal resting state, 2) submaximal and maximal work, 3) CO₂ breathing, and 4) several levels of hypoxia. Work was performed by exercising on a motor-driven treadmill. Thus, samples were obtained when the subject was at rest and during exercise while: 1) breathing room air; 2) breathing a CO₂-enriched gas mixture, and 3) breathing a variety of hypoxic gas mixtures. The precise CO₂-enriched gas mixture and hypoxic gas mixtures employed were not specified. Arterialized venous and arterial samples were drawn simultaneously and immediately

cooled to approximately 1° C. Each pair of samples were analyzed in random order within two hours following collection.

Results of the experiments were reported only in terms of identity plots comparing values from arterialized venous blood (abscissa) and arterial blood (ordinate) during rest and work. Hence, no separate analysis of data could be made. However, the authors did state that the findings during rest and work, while subjects breathed various gas mixtures, showed the same tendencies. Over a 25 mm Hg range of values for pCO₂ (pa_{CO₂} 30-55 mm Hg), arterialized venous pO₂ averaged approximately 1.0 mm Hg higher than arterial pCO₂. Table 1 summarizes the results of this study.

The pO_2 of arterialized venous blood at rest and during work was invariably lower than that of arterial blood. Due to the shape of the oxyhemoglobin dissociation curve, at $\mathrm{pa}_{\mathrm{O}_2}$ values above 70 mm Hg, a large proportion of the O_2 consumed is from that physically dissolved in solution. Thus, a large arteriovenous difference in pO_2 results. However, at $\mathrm{pa}_{\mathrm{O}_2}$ values below 70 mm Hg, or under hypoxic conditions, a greater portion of the O_2 consumed is obtained from that combined with hemoglobin. Hence removing oxygen from hemoglobin at values of pO_2 less than 70 torr (steep portion of oxyhemoglobin dissociation curve)

Table 1
Summary of Results Reported by Forster, et al.

Parameter	n	Pearson's r	Slope	Intercept
рН	82	0.98	0.86	1.041
pCO_2	84	0.95	1.01	-1.27
$pO_2 > 70 \text{ torr}$	33	0.66		
< 70 torr	41	0.92		
Lactate mg/100 ml	42	0.92	0,95	0.01

(Note: Values for pO_2 slope and intercept were not available).

will cause a smaller change in pa_{0_2} than when oxygen is removed at values of $p0_2$ greater than 70 torr (flatter portion of oxyhemoglobin dissociation curve). Finally, the investigators recognized that at all levels of oxygenation in these experiments, the arteriovenous difference and variation from the regression line were of such magnitude that accurate correction of venous to arterial $p0_2$ was not possible. They concluded that arterial $p0_2$ may not be reliably predicted from arterialized venous blood. In contrast, arterialized venous pH, $p0_2$ and lactate may provide an accurate estimate of arterial values.

Summary of Research Using Arterialized Venous Blood

In the studies reviewed involving the use of arterialized venous blood, venous samples were obtained from the forearm or dorsum of the hand. The majority of researchers studied pH and pCO $_2$ (or CO $_2$ content) under varying physiological conditions. The arterial pH range studied varied from 1.267 to 1.484. Arteriovenous differences in pH and pCO $_2$ were found to be small and not clinically significant. Therefore, it was concluded by all investigators that arterialized venous blood may be used in lieu of arterial blood for measurement of pH and pCO $_2$.

Several investigators examined the use of arterialized venous blood for determination of \mathbf{O}_2 saturation and \mathbf{pO}_2 .

Meakin and Davies (1920) and Goldschmidt and Light (1925) found the ${\rm O}_2$ saturation of arterialized venous and arterial blood to be virtually identical. In contrast, Collis and Neaverson (1967) reported large arteriovenous differences in ${\rm O}_2$ saturation. Collis and Neaverson (1967) and Forster, Dempsey, Thomson, Vidruk and DoPico (1972) reported substantial differences in ${\rm pO}_2$ between arterialized venous and arterial blood. These differences were particularly great when the ${\rm pO}_2$ exceeded 70 mm Hg, due to the shape of the oxyhemoglobin dissociation curve. Hence, it was decided that arterial ${\rm pO}_2$ may not be accurately predicted from arterialized venous blood. A summary of all investigators reviewed above is found in Table 2.

Problem Statement

Previous studies employing the use of arterialized venous blood were performed within a relatively narrow pH range (i.e., 7.267 to 7.484). In this study, I investigated the relationship between the blood-gas compositions of arterial and venous blood in respiratory acid -base disorders over a much wider range. As outlined below, healthy dogs instead of human subjects were used. In order to produce respiratory acid-base disturbances, ventilation rate was controlled via a mechanical ventilator.

Up to this point, this study has dealt only with the use of peripheral venous blood. However, mixed venous

Table 2 Summary of Rescarch Using Arterialized Venous Blood

Investigators	Subjects and Numbers	Site of Venous Sampling	Method of Arterialization of Venous Blood	Parameters Measures	Results	
(Meakin and Davies, 1920)	1 human	forearm	arm innersed in 45° water bath for 10 minutes and 20 minutes	$egin{aligned} \mathbb{Q}_2 & ext{saturation of} \\ & ext{hemoglobin (\%)} \end{aligned}$	Arterial O ₂ sat. (%) 96.1	Venous O ₂ sat. (%) 0.0-94.2
(Goldschmidt and Light, 1925)	6 humans	dorsum of hand	hand and wrist immersed in 46-47 ⁰ water bath for 10 minutes	a) O_2 cont.(vol.%) b) O_2 cap. of hemoglobin c) O_2 sat. (%) d) O_2 cont. (vol. %)	a) 14.56-20.33 vol.% b) 17.41-32.15 vol.% c) 45.3-98.1% d) 31.4-49.9 vol.%	Venous 16.03-20.08 vol.% 17.85-32.04 vol.% 50.0-97.4% 29.7-49.6 vol.%
(Brooks and Wynn, 1959)	24 humens dor 3 groups: han 1) 5 ambula- writory patients 2) 9 patients on bedrest 3) 10 anesthetized patients	dorsum of hand or wrist nts on tized	groups 1 and 2: forearm wrapped in heating pads for 15 minutes group 3: no warming used	pH (pH units) pCO ₂ (mm Hg)	Mean Arteriovenous Differences (using arterialized venous blood) Group pH (pH units) pOO ₂ (mm Hg) 1) 0.018 3.5 2) 0.002 0.8 (using non-arterialized venous blood) 1) 0.061 9.6 2) 0.045 6.7 3) 0.002 1.1	Differences venous blood) poo_2 (mm Hg) 3.5 0.8 zed venous blood) 9.6 6.7 1.1

Investigators	Subjects and Number	Site of Venous Sampling	Method of Arterialization of Venous Blood	Parameters Measures	Results
& Soloff, 1961)	29 humans	3 groups 1) 6 subjects constriction above elbow vacuum tubes 2) 9 subjects constriction above elbow heparinized syringes 3) 14 subjects constriction at wrist	dorsum of hand wrapped in hot towel or immersed in hot water bath for 15-20 minutes	pll (pH units) pco_2 (mm Hg) pH (pH units) pco_2 (mm Hg) pH (pH units) pco_2 (mm Hg)	Mean arteriovenous Differences Group pH (pH units) pCO ₂ (mm Hg) 1) 0.050 4.9 2) 0.038 8.5 3) 0.022 2.86
(Harrison and Galloon, 1965)	13 humans	dorsum of hand	hand wrapped in heating pad	pOO ₂ (mm Hg)	Mean arteriovenous Difference $\overline{p}\overline{\omega}_2$ (mm Hg) 0.5 (at 40 mm Hg $p\omega_2$) SD 0.7 mm Hg (at 40 mm Hg $p\omega_2$)
(Collis and Neaverson, 1967)	23 humans	dorsum of hand	hand immersed in 450 C. water bath for 5 minutes	pH (pH units) pO_2 (mm Hg) O_2 sat. (%)	Mean arteriovenous Difference pH (pH units) pCO ₂ (mm Hg) O ₂ sat. (%) 0.0052 0.76 2.53 SD=0.0075 0.81 1.88
(Forster, Dempsey, 13 humans Thomson, Vidruk males and DoPico, 1972)	13 humans mules	dorsum of hand	hand wrapped in heating pad for 10 minutes	$\begin{array}{l} \mathrm{pH}\;(\mathrm{pH}\;\mathrm{units})\\ \mathrm{pOO}_2\;(\mathrm{rm}\;\mathrm{Hg})\\ \mathrm{pO}_2\;(\mathrm{rm}\;\mathrm{Hg})\\ \mathrm{lactate}\;(\mathrm{mg/l00ml}) \end{array}$	Refer to Table 1

blood drawn from the pulmonary artery is sometimes used to assist in the assessment of cardiopulmonary status of critically ill patients. A Swan-Ganz flow directed catheter must be placed in order to obtain mixed venous blood samples. This is an invasive and, in some cases, dangerous procedure. Many of the hazards and complications associated with arterial puncture pertain to the collection of mixed venous blood. A Swan-Ganz flow directed catheter was placed in the pulmonary artery of each of the experimental animals used in this study. Data obtained from mixed venous blood samples drawn from this catheter was also used in a different study by another investigator. Information derived from an analysis of this blood can be compared with that obtained from peripheral venous blood. Therefore, I investigated the relationship between the blood-gas composition of peripheral venous and mixed venous blood. Specifically, the possibility of substituting peripheral venous for mixed venous blood in blood-gas analysis was explored.

This investigator could find no research literature comparing peripheral venous to mixed venous blood. While there has been much interest in comparing arterial to mixed venous blood, this literature does not relate to the present study, and was not reviewed.

In this investigation, the following questions were explored:

- 1) How does the blood-gas composition of arterial and arterialized peripheral venous blood compare in the extreme pH ranges of respiratory acid-base disorders?
- 2) What is the difference between mixed venous and arterialized peripheral venous blood-gas composition in respiratory acid-base disorders?
- 3) How does the blood-gas composition of nonarterialized peripheral venous blood compare to mixed venous and arterial blood in respiratory acid-base disturbances?

Implications for Nursing

The profession of nursing has changed profoundly in the last two decades. Nursing roles, responsibilities and goals have evolved in conjunction with the advancement of medical knowledge and technology. Nurses now participate actively in the assessment and management of critically ill patients. One of the methods frequently used to assess such patients involves determination of the arterial bloodgas composition. Nurses working in critical care areas are frequently responsible for collecting samples to be used in blood-gas analysis. In addition, nurses may also be required to interpret the results of these analyses

and determine their implications for future patient management. The status of critically ill patients may change very rapidly, particularly respiratory function. Changes in respiratory status frequently produce changes in acid-base status which are reflected in blood-gas composition. Nurses are present and observing such patients 24 hours a day. Therefore, they are in an excellent position to assess the acid-base status and intervene appropriately.

The collection of arterial blood for blood-gas analysis is associated with many hazards and complications. These include such problems as: hemorrhage, thromboses, hematomas, nerve injury and infection. Additionally, arterial puncture may be quite painful to the patient. Nurses have a responsibility to act as the patient's advocate and to provide comfort as well as safety. Critically ill patients often have an indwelling venous catheter (or heparin lock) placed for administration of medications and fluids. Some patients may also have a Swan-Ganz catheter inserted into the pulmonary artery for sampling mixed venous blood. Peripheral venous blood may prove to be a reliable substitute for arterial and/or mixed venous blood in blood-gas analysis. Hence, this study is of importance in expanding the scientific foundation on which nursing practice is based.

CHAPTER III METHODS

Statement of the Variables

The independent variable was the respiratory acidbase status of the animal at the time of sampling. Respiratory acidosis was produced by administering varying concentrations of ${\rm CO}_2$. Respiratory alkalosis was induced by hyperventilation.

The dependent variables were pH pCO₂, pO₂ and HCO₃ concentrations of arterial, free-flowing peripheral venous and mixed venous blood. The arterial blood-gas values provided control data for the determination of each respiratory acid-base disturbance. See Appendix A for definitions of terms.

Procedures and Controls

In this study, ten healthy mongrel dogs of both sexes weighing from 10.9 to 29.5 Kg were used as experimental subjects. Each dog was anesthetized initially with sodium pentobarbital 30 mg/Kg intravenously. Anesthesia was maintained throughout the experiment by the administration of sodium pentobarbital 30 mg/Kg every 1/2 to 1 hour as necessary. The trachea was cannulated and each animal was then placed on a volume respirator (Mode #607).

The right or left femoral vein was catheterized and used for the infusion of solutions. The right or left-femoral artery was catheterized and attached to a pressure

transducer which, in turn, was attached to a polygraph. Wave forms representing the blood pressure and heart rate were thus obtained and recorded by the polygraph. Arterial blood samples were obtained from this femoral artery catheter. A Swan-Ganz flow-directed catheter was placed in the pulmonary artery for collection of mixed venous blood samples.

In order to obtain muscle paralysis, curare 0.5 to 1.0 ml was administered with the maintenance dose of anesthesia as needed. Muscle paralysis prevents the animals from breathing in opposition, or in addition to, the rate set by the respirator. Curare was never given without anesthetic. Complete anesthesia of the animals was assumed by administering maintenance doses of anesthetic at a frequency equal to or greater than that given to animals that did not receive curare.

A rectal thermometer was placed and a baseline rectal temperature taken. Rectal temperatures were monitored throughout the experiment.

One forepaw was selected to be the non-arterialized (unwarmed) venous site, while the other paw served as the arterialized (warmed) venous site. A vein in each forepaw was catheterized to obtain the venous blood samples. The tip of the catheter was directed distally. Free-flowing venous samples were desired. Therefore, tourniquets were

not used. The temperature of each venous sampling site was obtained from a thermometer placed under the skin of the forepaw near the tip of the catheter.

Venous blood was arterialized according to the following procedure: approximately five minutes prior to sampling, a goose neck lamp with a 100 watt bulb was placed 2-3 cm from the forepaw. The bulb was directed toward the tip of the catheter. Warming in this manner continued until the temperature reached 38-42° C. When this temperature range was attained, arterialized venous samples were drawn.

The samples of arterial, warmed, unwarmed and mixed venous blood were collected within approximately two minutes of each other. Each 1 ml sample was collected anaerobically into a 1 ml glass syringe. Prior to the experiment, the syringes were prepared as follows: 1) Barrels and plungers of the syringes were lubricated with stopcock grease. This prevents gas or blood from leaking around the plungers. 2) Sodium panheparin 1000 units/ml was added to the syringes as an anticoagulant. 3) A small volume of mercury was then drawn into each syringe. This mercury facilitated the mixing of each sample. In addition, the mercury was pushed up into the hub of the syringe just prior to sampling, thereby filling the dead space and removing excess heparin. All samples were drawn at 20 and

60 minutes following the changes of respiratory status. Each sample was placed immediately on crushed ice and analyzed within one hour of collection.

At the beginning of each experiment and preceding the manipulation of respiratory status, initial or control samples of arterial, warmed, unwarmed, and mixed venous blood were drawn. The results from these control samples served as baseline values with which subsequent results were compared.

In this study, two different respiratory acid-base disturbances were induced: respiratory acidosis and respiratory alkalosis. Respiratory acidosis was induced in half of the experimental animals by varying the concentration of CO₂ in the inspired gas mixture. Concentrations used included: 3% CO_2 in O_2 , 5% CO_2 in O_2 , and 10% CO2 in O2. The gas mixtures were obtained from cylinders containing gases of known concentration. (Inspired concentrations were accurate to $^{+}$ 0.5%). The animal was given CO, in ascending concentrations as indicated. Arterial, warmed, unwarmed, and mixed venous samples were collected at 20 and 60 minutes after beginning the administration of each gas mixture. After obtaining the 60-minute blood sample while administering the 10% CO_{2} gas mixture, the fractional concentration of the inspired gas was reduced again in descending order, i.e. from 10% to 5% to 3% and then

room air. Hence, each dog was brought back to the control gas mixture, that of room air. A total of seven sets of samples were obtained from each of these animals.

Respiratory alkalosis was induced in the remaining five dogs by gradually increasing the tidal volume by 150 ml increments. Initial tidal volume ranged from 200-350 ml depending upon the weight of the animal. Arterial, warmed, unwarmed, and mixed venous samples were again collected at 20 and 60 minute intervals following each adjustment in tidal volume. After maximal hyperventilation, the tidal volume was returned to the control setting in 150 ml decrements. A total of seven sets of samples were also collected from each of these animals.

Reliability of Measurements

In this study, a Radiometer BGA3 Blood-Gas Analyzer was used to measure the pH, pCO $_2$ and pO $_2$ of arterial, warmed, unwarmed, and mixed venous blood. This analyzer produces results which are reproducible to $^+$ 0.005 pH units, $^+$ 0.1 mm Hg pCO $_2$ and $^+$ 1.0 mm Hg pO $_2$.

The blood-gas analyzer was calibrated prior to the analysis of each set of samples. Calibration procedure and methods of measurement appear in Appendix B.

CHAPTER IV

RESULTS

General Description

The pH, pCO_2 and pO_2 of arterial blood, free-flowing peripheral venous blood, and mixed venous blood were measured during controlled respiratory acid-base disturbances. The bicarbonate concentration of each sample was calculated from the Henderson-Hasselbalch equation (pKa' = 6.1). Ten healthy mongrel dogs of both sexes, weighing from 10.9 to 29.5 kilograms, were employed as the experimental animals. Table 3 shows the ranges of the blood-gas parameters produced in these experiments. This is a much wider range than that previously reported in the literature.

Table 3
Ranges of Results of Blood-Gas Parameters

Source	рН	pCO2mm Hg	pO2mm Hg	[HCO3]mEq/L
Arterial	7.03-7.74	12.3-113.2	68.3-542.3	14.9-29.0
Warmed Venous	7.01-7.68	14.3-116.2	36.4-430.0	15.1-28.4
Unwarmed Venous	7.01-7.67	13.8-117.9	34.1-405.0	15.1-29.6
Mixed Venous	7.02-7.69	14.7-118.2	27.0-102.3	16.4-30.4

Note: A single set of sample values obtained from Dog 3 was not included in this table because of a respirator malfunction and subsequent alteration in respiratory status. The pCO_2 of this sample was substantially elevated such that it was uncertain whether or not the pCO_2 electrode would respond in a linear fashion.

Body temperature (rectal), warmed venous paw temperature, and heart rate were monitored throughout the experiments and at the time of sampling. A summary of these measurements is shown in Table 4. In addition, unwarmed venous paw temperature was determined in Dogs 6 to 10. Body temperature of the animals ranged from 36.0°C to 39.5°C, with the mean of means being 37.0°C + 0.3. Due to this narrow range of variation in body temperature during the experiments, correction of the blood-gas parameters for temperature change was not necessary. Temperature of the warmed venous paw, or sampling site, ranged from 38.0°C to 43.0°C; while the temperature of the unwarmed venous sampling site ranged from 28.0°C to 38.0°C. Heart rate varied between 96 and 180 beats per minute.

Table 4

Summary of the Mean of Means and Standard Error of the Mean for Body Temperature, Warmed Paw Temperature, Unwarmed Paw Temperature and Heart Rate.

	Body Femperature	Warmed Paw Temperature	Unwarmed Paw Temperature	Heart Rate
Mean of Means	37.0	39.9	34.0	137.0
Standard Error of the Mean	÷0.3	- 0.3	±1.0	+ 4.9

The hematocrit and serum protein concentrations were measured at the beginning and end of each experiment to determine if significant changes in blood volume occurred. No evidence of these changes was found.

Respiratory acidosis was induced in the first five experimental animals. Frequency of ventilation was initially set at 12 per minute, with tidal volumes ranging from 200 to 350 ml, depending upon the animal's weight. (See Appendix C). Control values for each of the blood-gas parameters (pH, pCO $_2$, pO $_2$ and [HCO $_3$]) were obtained for each animal following a 60-minute stabilization period at the initial respirator setting. Increasing hypercapnia was produced sequentially by changing the concentration of CO $_2$ in the inspired gas mixture. Control values for the

blood-gas parameters were obtained again at the end of each hypercapnia experiment except Dog 3. In this experiment, insufficient time was available to allow for this last determination. In addition, it should be noted that the data obtained from Dog 1 have not been included in this study. These data were not included because on post mortem examination, the tip of the Swan-Ganz catheter was located in the inferior vena cava near the right atrium, and not in the pulmonary artery.

Respiratory alkalosis was induced in five experimental animals. Control values during these experiments were obtained with the respiratory frequency set at 12 per minute, with a tidal volume ranging from 200-350 ml depending upon the weight of the animal. Increasing hypocapnia was produced by systematic hyperventilation. Note: Dog 10 was considerably larger than the other animals, weighing 29.5 kilograms. Thus, in this animal it was necessary to increase both tidal volume and respiratory rate in order to attain a comparable degree of alkalosis.

Table 5 contains a summary of sample values obtained at the 60-minute sampling time from both acidosis and alkalosis experiments. A complete summary of all sample values (20 and 60 minute) for each animal appears in Appendix C.

Table 5

60 Minute Sample Values Used for Statistical Analyses

HYPERCAPNIA

	П							-								-					-							
	MV	m	7	6.	8	8	28.7	α.	27.2	7	6	5	0	9	9	ė	7		7	9	က်		ä	5	7.	9		e,
Eq/L	ΔŊ	4	7	9	8	8	28.0	9		9		4.	6	S	9	1:	-	28.2	7	9	5	-	3	4	9	4.	23.2	-
HCO3 m	WV	r,	٠,	5	7	9	28.0	5	9	7	28.4	5	8	5	4.	9	5	27.9	9	5.	4	-	3	4.	5	4.	22.7	1
	A	-	Š.	9	œ	œ	27.5	0		9	6	3	8	4	ı,	-	ģ	27.9	-	5	4	1.	2	4	9	4.	22.7	i
	MV	-	7	80.	2	6	72.3	3	4	02.	106.0	14.	78.	7.	-:	-	œ	102.0	7	3	48.6	5	5	7	3	4	58.7	4.
2mmHg	Δ	59.	.09	0	47.	2	75.0	2.	51.	8	58	ě	86.	76.	67.	81.	45.	136.0	15.	59.	68.	3.	8	9	-	2	405.0	'n.
po ₂ m	MΛ	70.	16.	5	30.	48.	168.3	6	56.	49.	143.0	27.	95.	80.	67.	.90	00.		14.	15.	67.3	73.	43.	72.	75.	7	430.0	4.
	A	90.	83.	8	75.	81,	94	2		69	427.0	77.	58.	83.	5	67.	78.	432.0	36.	51.	9.	3.	59.	74.	68.	30.	542.3	02.
	MV	ထ	5	5	6.	5	66.2	7	4	78.	-	3	03.	79.							54.5		9	0	4	6	65.2	4
Яд	ΔΩ	2.	4.	5	6	ë	9.99	5	3	5	-	9	00	71.	4	3	4	112.2	÷	5	51.5	8	3	0	6	1	58.1	œ
pco ₂ mmHg	WV	3	-	8	4.	ě	62.0	7	2	73.		12.	.96	69.7	2	5	0	6	6.	4.	48.5	8	3.	2	5	1.	57.1	8
	A	5.	6	. 9	2	5	58.9	1:	7	e,	13	8	90.		-	œ	6	8	87.	0	47.2	8	0	9	-	8	55.8	8
	MV	.39	. 31	. 25	.08	. 21	7.259	.39	. 31	.17	0.	.77	.09	7.150	.39	. 26	.18	7.035	.09	.14	.29	.35	. 24	.19	.03	14	7.194	33
	UV I	. 39	. 31	. 25	.07	. 21	7.245	39	. 29	.16	.01	. 79	.09	7.169	38	. 25	.17	N	.08	.16	7.309	.36	. 26	22	.04	16	7.223	36
рн	WV	. 44	.33	.26	.08	.23	7.276	. 40	. 31	.19	.01	. 80	.09	7.175	. 38	. 25	.18	0	.10	.14	7.318	٣.	7	7	0	Ξ.	7.221	
	A	. 45	, 34	. 28	.10	. 25	7.290	.32	u,	1.	0		-	7.185	4	2	.2	0	1	-	7.327	~	2	. 7	0		7.230	~
	Period	C-Rm Air	38	50	10%	5%	38	Rm Air	C-Rm Air	e	2	10	S	38	C-Rm Air	m	17. %	108	S	3%	Rm Air	C-Rm Air	3	- 4º	108	40	3 60	Pm Air
		Dog 2							Dog 3		respirator	malfunc			Dog 4							Dog 5						

Table 5 (continued) HYPERVENTILATION

	MV	.5 25.5	.3 17.	.6 18.	.9 22.	.4 25.	.4 24.	. 9 21.	.5 20	.7 20.	7 21.1	.4 18.	.4 17.	.5 18.	.9 23.	.6 20.	1 16.	.7 17.	.0 17.8	.6 21.	. 9 18.	.6 17.	1.2 17.8	.7 20.	42	
HCO3 mEq/L	WV UV	24.4 24.19.5 19.	7.2 1	7.2	1.5 2	4.2 2	3.1 2	0.2	18.2 18	0.1 2	20.8 19	7.8 1	5.1	7.7	2.2 2	9.4	7.5 1	6.6	16.2 17 19.1 18	0.2 2	8.4	6.6	18.0 18	8.8		
H	Ą	0.5	6.2	6.9	0.3	3.4	m c	9 6	18.1	0 0	21.3	6.5	5.2	6.3	3	ထံ	- 4		17.0		5 00		17.8	, 00		
	MV	50.2	5:	in c	00	6	90	70	30.7	6.	42.0					α.	. α		41.9	0			41.5	. 0		
2mmHg	ΛΩ	45.9	1.9	8	00	4	e <	, r	45.5	50	59.9	2	0	9 9	2	1.	9 10	0	62.7	7	3 6	0	54.3	, 6		
po ₂ mn	WV	65.9	8 4	0	3.0	3	800	, ė	53.3	7.	0 4	0	.i.	54.8	7	5	מי ע	· ~	76.1	2.	0 6		68.1	100	ednence:	
	A	88	2 2	97.	. 0	6	m' <	* 4	77.5	7.	132.9		8	4.6	4	15.	20.	29.	123.0	4	- 5	8	76.5	7 7	ρ0 02	
	MV	40.6	9 6	œ 1	3.	7	9,0	1 4	28.0	. 8	39.5	. 10	-	m m	0	3	1.	, 0	23.8	-	2, -	00	20.7		the followin	(2)
2mmHg	UV	44.6	ထ က	-	υ. Γ.	3	9.	110	25.7	. w			3	23.4	7	4.	1.0	10	23.4	6	. c	::	21.0	9 6	ged in	n Air) n 3% CO2
pco ₂ mm	ΛM	39.2	m in	-	9.0	2	-1	. 4	24.0	4 .	8 4	. 0	œ .	30.8	0	3		. 9	21.8	8		. 9	20.5	7.	chan	21 (Room 97 (with
	А	37.9	50	9.	3:	7	m u	. 0	22.9	. 6	7. 1	, 0	9	21.7	1.	-	9 0	10	21.8	6			19.8	7 9	Note: FI	000
	MV	7.419	. 68	.61	39	. 26	. 41	.55	7.480	. 17	E. 4	4 4	.54	38	.37	. 54	. 62	. 60	7.515	34	. 45 7. 7.	. 60	7.555	.31	2	
	UV	7.362	.62	.61	.39	. 28	41	. 540	7.478	.17	W <	. 4	.5	7.457	3	. 52	.61	. 60	7.515	. 34	. 4. v.	. 60	7.559	. 32		od
Hď	WV	7.416	.68	. 60	.39	. 28	41	.54	7.502	.18	7.347	5.1	. 55	. 47	.36	.53	.61	. 61	7.520	.34	٠ 4 د م	. 61	7.565	. 32	٦,	nous blc
	A	7.427	.74	. 64	.40	5	4. 1	0.0	7.518	2.4	. 36	.53	.58	7.499	.38	. 56	.64	9 0	7.542	. 35	. 46 76	. 62	7.574	. 32	rial blood	Unwarmed venous blood Mixed venous blood
Period	TV RR	200 12 350 12	50 I	00 1	000	00	100	50 1	500 12	00	250 12	50 1	0.0	50 1	0 1	0 1	0	110	350 12	0 1	500	50 1	650 14	50 1	= Arteri	11 11
1		Dog 6				Dog 7					Dog 8				Вод 9					Dog 10					KEY: A	M V

Specific Parameters

The specific results of this study are organized and discussed under two primary headings:

- Comparisons between arterial blood (A) and peripheral venous blood [warmed (WV) and unwarmed (UV)].
- 2. Comparisons between mixed venous blood (MV) and peripheral venous blood [warmed (WV) and unwarmed (UV)].

Each individual parameter of pH, pCO_2 , pO_2 and $[HCO_3^-]$ is addressed under these two primary headings. Both hypercapnia and hyperventilation (hypocapnia) experiments are included in this discussion of each parameter.

1. Comparisons between arterial blood (A) and peripheral venous blood [warmed (WV) and unwarmed (UV)]

In general, the values for pH, pCO_2 and $[HCO_3^-]$ of peripheral venous blood (both WV and UV) closely correlated with those of arterial blood.

рН

The relationship of the pH of warmed and unwarmed venous blood as a function of the pH of arterial blood is shown in Figures 2 and 3. Inspection of these figures shows that most of the values lie close to, and slightly

below, the identity line. Correlation coefficients

(Pearson's r) for pH were 0.93 or greater for the hypercapnia experiments, and 0.98 or greater for the hyperventilation (hypocapnia) experiments. Refer to Table 6
for specific values.

pCO_2

Figures 4 and 5 show the identity relationship between the pCO_2 of warmed and unwarmed venous blood as a function of the pCO_2 of arterial blood. In both of these comparison figures the observed values lie on, or slightly above the identity line. The r values were always greater than 0.98 for the hypercapnia group, and greater than 0.92 in the hyperventilation (hypocapnia) group. See Table 6 for individual values.

[HCO₃]

Comparisons beween the [HCO₃] of warmed and unwarmed venous blood to that of arterial blood are shown in Figures 8 and 9. The majority of values are found to lie closely scattered about the identity line. The r values for the hypercapnia group always exceeded 0.84. In the hyperventilation experiments, r values were generally greater than 0.92. However, in one animal (Dog 8) the r value comparing arterial blood to unwarmed venous blood was found to be 0.43. Refer to Table 6.

It is apparent from visual inspection of Table 7 that there are no significant differences between the mean slopes and intercepts obtained for pH, pCO₂ and [HCO₃] in the hypercapnia and hyperventilation experiments. Note that this statement applies to comparisons involving both arterial and mixed venous blood with peripheral venous blood.

p02-

The pO_2 values of peripheral warmed and unwarmed venous blood did not correlate as strongly with those of arterial blood. These relationships are shown in Figures 6 and 7. It can be seen that at arterial pO_2 values less than 150 mm Hg, most points lie close to, and slightly below the identity line. However, when arterial pO_2 exceeded 350 mm Hg, the points scattered widely below the identity line. Correlation coefficients for the hypercapnia experiments were 0.67 or greater. In the hyperventilation group, r values varied from -0.53 to 0.61 (Table 6). Mean r values for pO_2 during hyperventilation appears in Table 8.

2. Comparisons between mixed venous blood (MV)

and peripheral venous blood [warmed (WV) and

unwarmed (UV)]

The values obtained for pH, pCO $_2$ and [HCO $_{\overline{3}}$] of peripheral venous blood (both WV and UV) were also found

to correlate closely with those of mixed venous blood.

рН

The relationship between the pH of warmed and unwarmed venous blood as a function of the pH of mixed venous blood is depicted in Figures10 and 11, respectively. In Figure 10, the majority of the points from all nine animals are distributed very close to, and slightly above the identity line. In Figure 11, the points appear to be more equally distributed on either side of the identity line. The r values for pH in the hypercapnia experiments were all greater than 0.99, and greater than 0.98 for the hyperventilation (hypocapnia) experiments. Refer to Table 6 for specific values.

pCO₂

Figures 12 and 13 show the identity relationship between the pCO_2 of warmed and unwarmed venous blood as a function of the pCO_2 of mixed venous blood. Except for two points in Figure 12 (MV vs WV), all of the points fall slightly below the identity line. In Figure 13 (MV vs UV) the observed values lie primarily on, or slightly below, the identity line. The r values for the hypercapnia group always exceeded 0.98, while those for the hyperventilation (hypocapnia) group were always greater than 0.97 (Table 6).

[HCO-_]

Comparisons between the [HCO_3^-] of warmed and un-

warmed venous blood to that of mixed venous blood are shown in Figures 16 and 17. The values from all nine animals are found to distribute primarily on or below the identity line in both figures. Correlation coefficients were 0.73 or greater for the hypercapnia experiments. In the hyperventilation experiments, the r values generally exceeded 0.87. However, in Dog 8 the r value comparing mixed venous blood to unwarmed venous blood was only 0.57. Refer to Table 6 for a summary of these values.

 \underline{pO}_{2-}

The pO_2 values of peripheral warmed and unwarmed venous blood correlated more closely with mixed venous blood than with arterial blood. It can be seen in Figures 14 and 15 that at mixed venous pO_2 values of 50 mm Hg or less, the points clustered tightly above the identity line. At mixed venous pO_2 values greater than 50 mm Hg, the points were found to scatter upward above the identity line. It is of interest to note that in Figure 15 (MV vs UV) these scattered points fell slightly closer to the identity line than in Figure 14 (MV vs WV). As shown in Table 6,r values for the hypercapnia experiments varied from -0.12 to 0.89, and from 0.07 to 0.94 for the hyperventilation experiments. Refer to Table 8 for mean r values for pO_2 during hyperventilation.

Table 6

Hypercapnia and Hyperventilation Summary of Correlation Coefficients (Pearson's r):

		Hd	н			pCO ₂ mmHg	mHg			po ₂ mmHg	Нд			HCO3 mEq/L	Eq/L	
	AvsWV	AvsUV	MVVSWV	MVvsUV	AvsWV	AvsUV	MVVSWV	MVvsUV	AvsWV	AvsUV	MVvsWV	MVvsUV	AvsWV	AvsUV	MVVSWV	MVvsUV
HYPERCAPNIA																
Dog 2	0.951	0.928	0.997	0.999	966.0	0.982	0.990	0.995	0.926	0.735	0.886	0.738	0.837	0.901	0.960	0.919
Dog 3	0.996	0.999	0.996	0.997	0.998	0.998	0.997	0.998	0.670	0.707	0.707 -0.118	-0.046	0.978	0.966	0.977	966.0
Dog 4	0.997	0.997	0.996	0.995	0.997	0.999	0.990	0.994	0.749	0.907	0.605	0.730	0.931	0.949	0.731	0.852
Dog 5	0.999	0.999	0.999	0.998	0.993	0.991	0.988	0.992	0.943	0.990	0.801	0.742	0.970	0.993	0.945	0.967
HYPERVENTILATION	ATION															
Dog 6	0.995	0.982	0.998	0.983	0.986	0.984	0.996	0.975	-0.530	-0.244	0.879	0.073	0.930	0.965	0.973	0.962
pod 7	0.989	0.995	0.991	0.998	0.993	0.989	0.993	0.993	0.244	0.104	0.685	0.936	0.980	0.928	0.979	0.948
Dog 8	0,998	0.979	0.989	0.986	0.990	0.916	0.993	0.971	0.342	0.542	0.924	0.908	0.916	0.426	0.974	0.567
Dog 9	0.999	0.999	0.997	0.998	0.995	0.996	0.985	0.977	0.057	-0.502	0.149	0,598	0.939	0.973	0.974	0.940
Dog 10	0.999	0.999	0.999	1.000	0.998	0.995	0.997	0.996	0.608	0.584	0.676	0.879	0.961	0.960	0.878	0.923

A vs WV = Arterial blood versus warmed venous blood A vs UV = Arterial blood versus unwarmed venous blood MV vs WV = Mixed venous blood versus warmed venous blood MV vs UV = Mixed venous blood versus unwarmed venous blood KEY:

49

Mean Values for Slopes and Intercepts: Hypercapnia and Hyperventilation Table 7

pCO ₂ mm Hg	Avsuv Mvswv Mvsuv A	0.98 0.97 0.98	0.05 0.14 0.05 C	5.4 -2.5 -0.4	5.7 10.7 3.0	0.99 0.96 0.91	0.94 0.10 0.90	3.7 -0.4 1.8	5.3 2.8 2.9
	NvsUV AvsWV	 0.98 0.41	0.05 0.20	-0.4 31.8	3.0 14.0	 0.91 0.12	0.90 0.50	1.8 53.7	2.9 41.7
pos mm Hg	AvsUV	0.38	0.22	30.2	15.9	0.10	0.35	46.9	33,7
m Hg	MVS NV	2.88	3.31	-2.4	161.6	0.74	0.42	31.7	21,3
	MVvsUV	2.46	2.82	11.3	127.6	0.86	0.72	19.6	28.4
HC	AVSIV AV	0.82	0.05	4.3	1.6	0.84	0.12	3.1	2.2
HCO3 mEq/L	AvsUV MA	0.74	0.36	7.2	10.0	0.74	0.26	5.1	4.9
	MVVSWV MVVSUV	1.17 0.92	0.56 0.28	-6.3 1.7	16.1 8.1	0.95 0.81	0.22 0.17	0.2 3.1	4.3 3.6

KEY: A vs NV = Arterial blood versus warmed venous blood

A vs UV = Arterial blood versus unwarmed venous blood

MV vs NV = Mixed venous blood versus warmed venous blood

M vs UV = Mixed venous blood versus unwarmed venous blood

Table 8

Mean Values for Pearson's r, Slopes and Intercepts

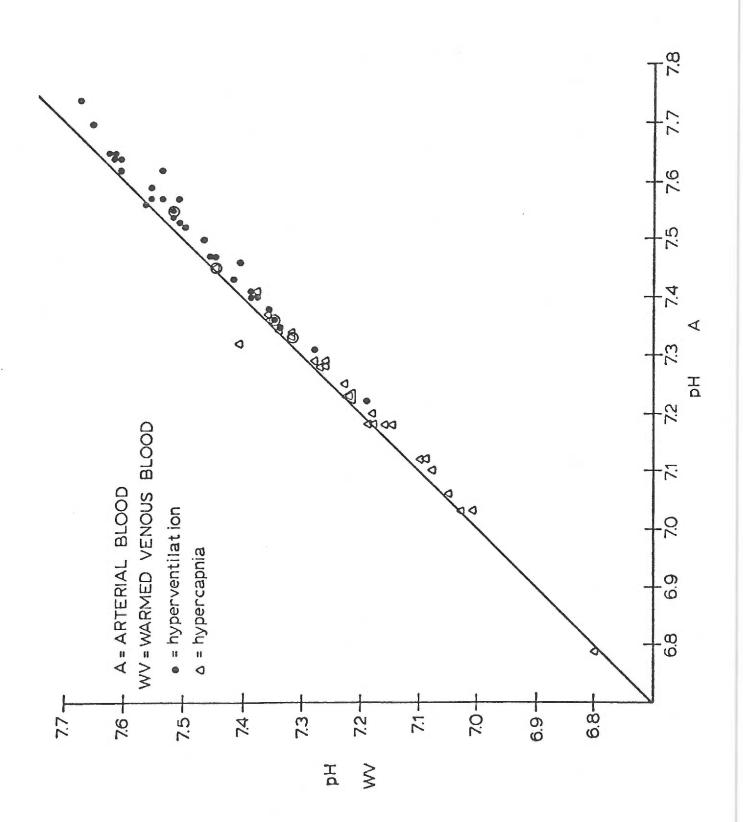
for pO₂ During Hyperventilation (Dogs 6-10)

	A vs WV	A vs UV	
r*	0.38	0.41	
slope	0.12	0.10	
+SD	0.50	0.35	
intercept	53.7	46.9	
+SD	41.7	33.7	
			·
	MV vs WV	MV vs UV	
r*	0.74	0.79	
slope	0.74	0.86	
+SD	0.42	0.72	
intercept	31.7	19.6	
+SD	21.3	28.4	

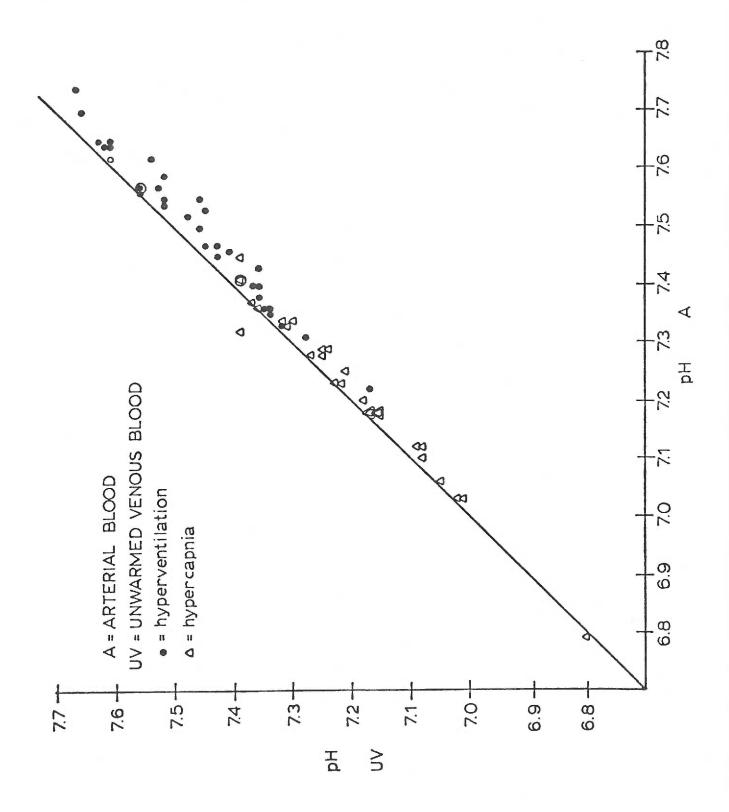
^{*}The mean value for Pearson's r was calculated using Fishers Z transformation. (Note: Slopes and intercepts are repeated here from Table 7 for comparison purposes).

KEY: A vs WV = Arterial blood versus warmed venous blood
A vs UV = Arterial blood versus unwarmed venous blood
MV vs WV = Mixed venous blood versus warmed venous blood
MV vs UV = Mixed venous blood versus unwarmed venous blood

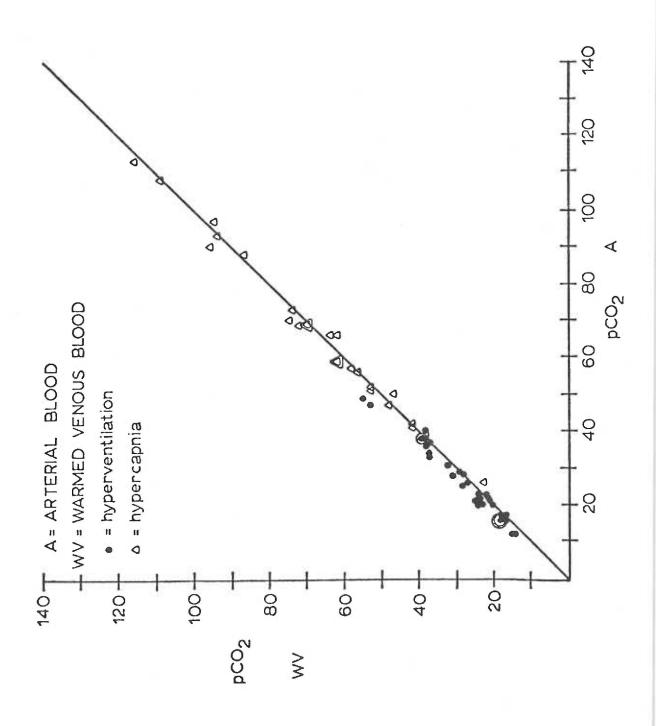
venous pH values obtained from nine animals during respiratory acidosis symbol represents one pair of values. The identity line indicates a Hyperventilation was used to produce hypocapnia. Identity relationship is shown for 60 minute arterial and warmed slope and correlation coefficient of 1.0. and alkalosis. Figure 2.



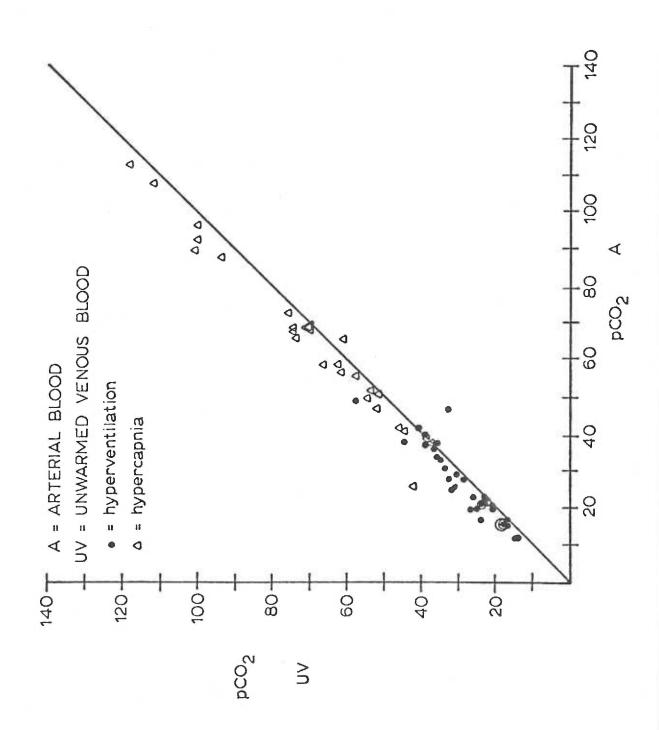
venous pH values obtained from nine animals during respiratory acidosis Each symbol represents one pair of values. The identity line indicates a Identity relationship is shown for 60 minute arterial and unwarmed and alkalosis. Hyperventilation was used to produce hypocapnia. slope and correlation coefficient of 1.0. Figure 3.



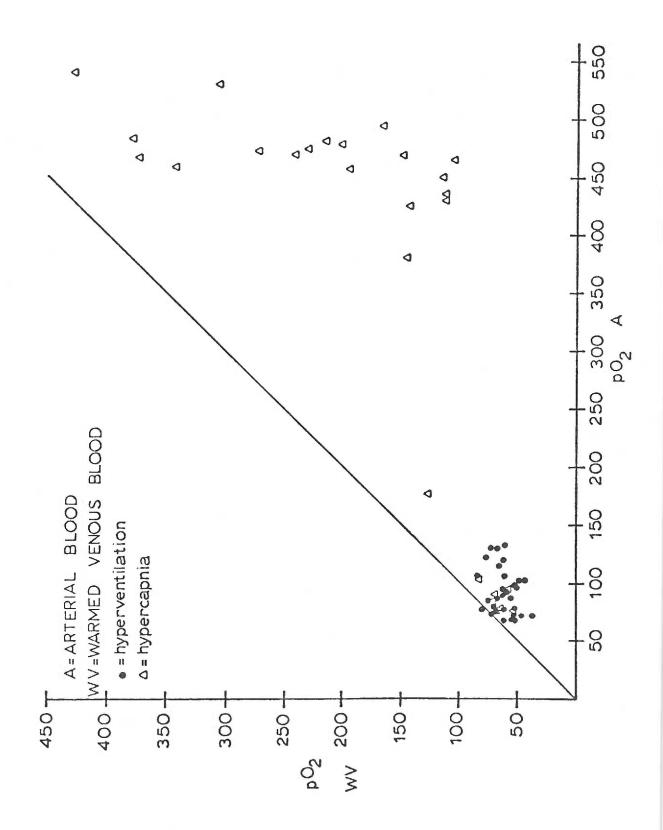
identity line indicates a slope and correlation coefficient of 1.0. Identity relationship is shown for 60 minute arterial and warmed venous pCO_2 values obtained from nine animals during respiratory acidosis and alkalosis. Hyperventilation was used to produce hypocapnia. Each symbol represents one pair of values. Figure 4.



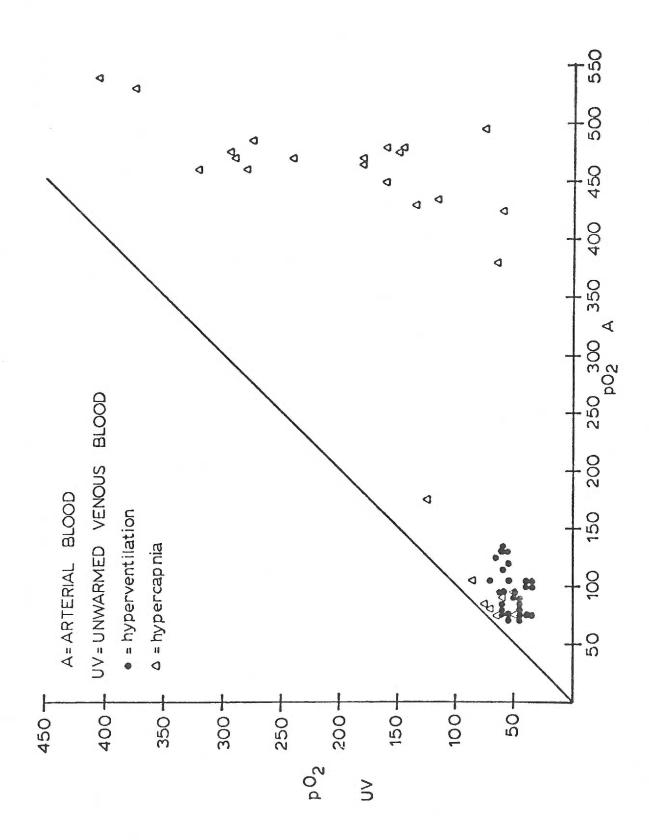
hypocapnia. Each symbol represents one pair of values. The identity Identity relationship is shown for 60 minute arterial and unwarmed venous $p{\rm CO}_2$ values obtained from nine animals during respiratory acidosis and alkalosis. Hyperventilation was used to produce line indicates a slope and correlation coefficient of 1.0. Figure 5.



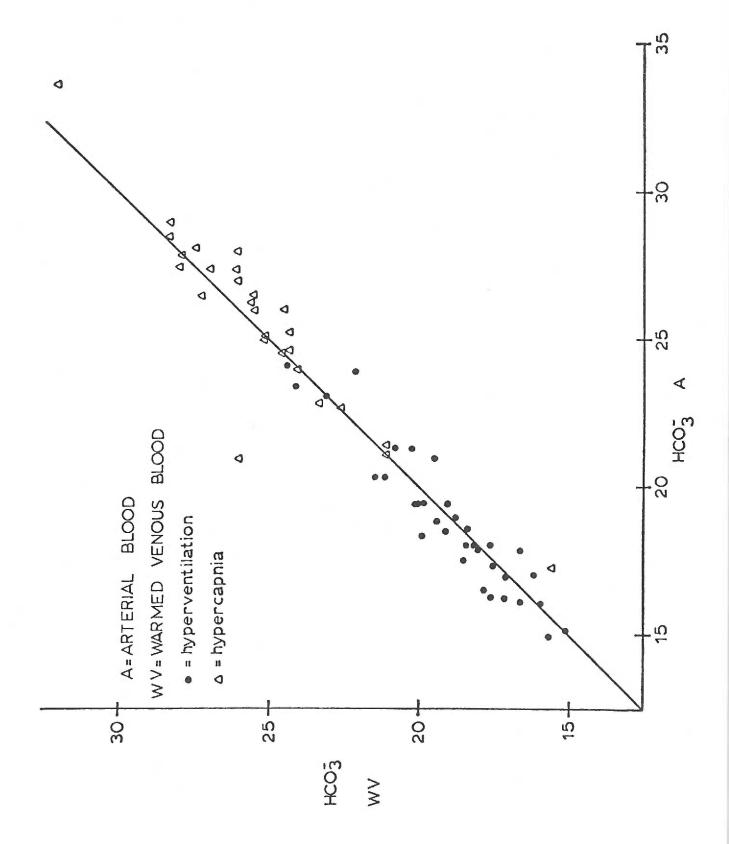
identity line indicates a slope and correlation coefficient of 1.0. Identity relationship is shown for 60 minute arterial and warmed venous $\mathrm{p0}_2$ values obtained from nine animals during respiratory acidosis and alkalosis. Hyperventilation was used to produce hypocapnia. Each symbol represents one pair of values. The Figure 6.



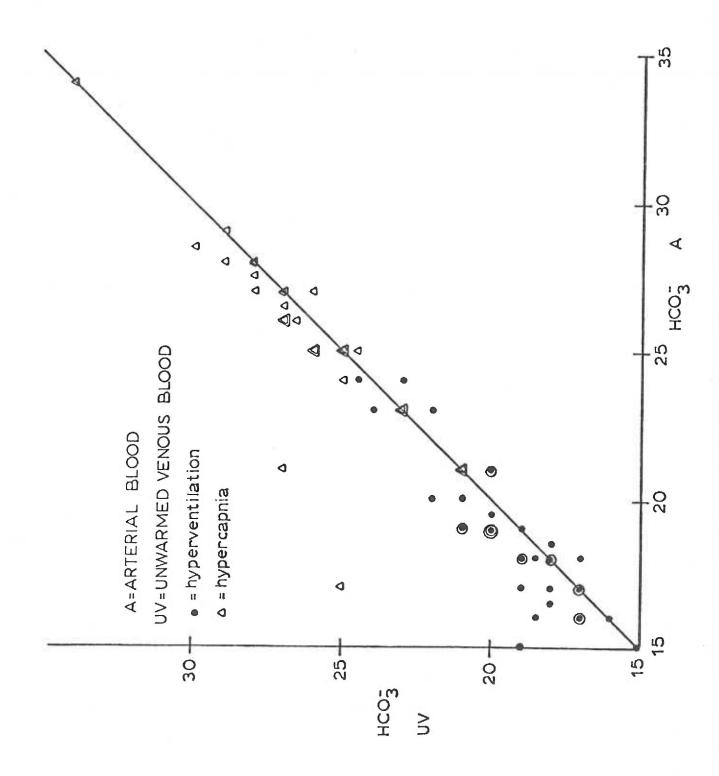
hypocapnia. Each symbol represents one pair of values. The identity Figure 7. Identity relationship is shown for 60 minute arterial and unwarmed venous $\mathrm{p0}_2$ values obtained from nine animals during respiratory acidosis and alkalosis. Hyperventilation was used to produce line indicates a slope and correlation coefficient of 1.0.



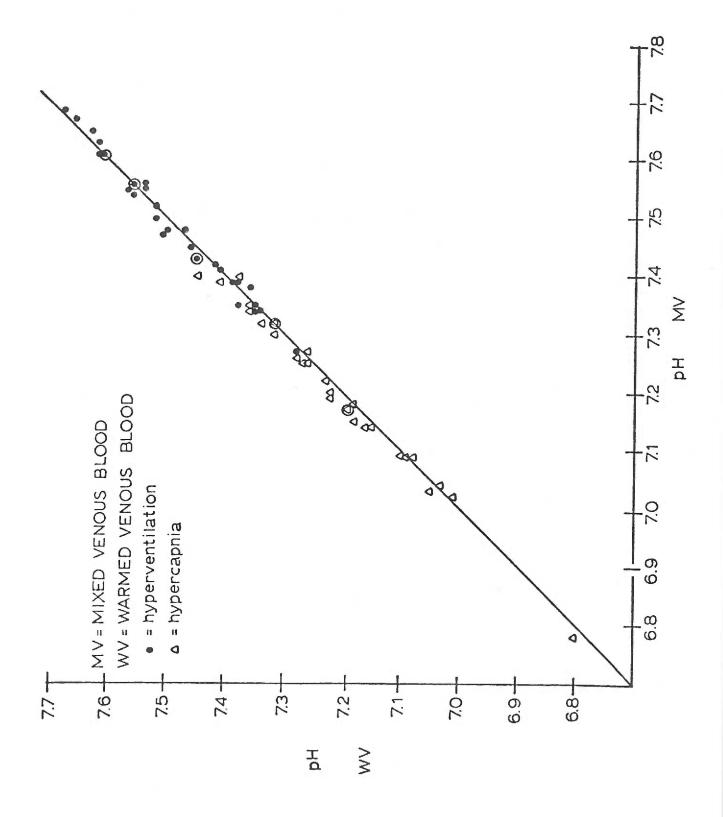
venous HCO_3 concentrations calculated from data from nine animals during respiratory acidosis and alkalosis. Hyperventilation was Identity relationship is shown for 60 minute arterial and warmed used to produce hypocapnia. Each symbol represents one pair of values. The identity line indicates a slope and correlation coefficient of 1.0. Figure 8.



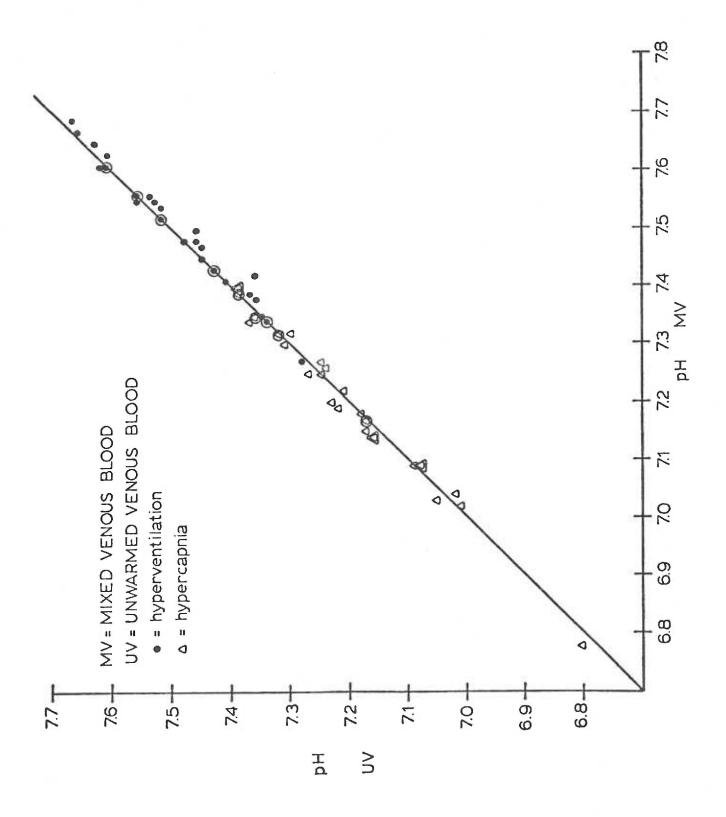
during respiratory acidosis and alkalosis. Hyperventilation was used Identity relationship is shown for 60 minute arterial and unwarmed to produce hypocapnia. Each symbol represents one pair of values. The identity line indicates a slope and correlation coefficient of venous HCO_3 concentrations calculated from data from nine animals Figure 9.



The identity line indicates a slope and correlation coefficient of respiratory acidosis and alkalosis. Hyperventilation was used to produce hypocapnia. Each symbol represents one pair of values. and warmed venous pH values obtained from nine animals during Identity relationship is shown for 60 minute mixed venous Figure 10.

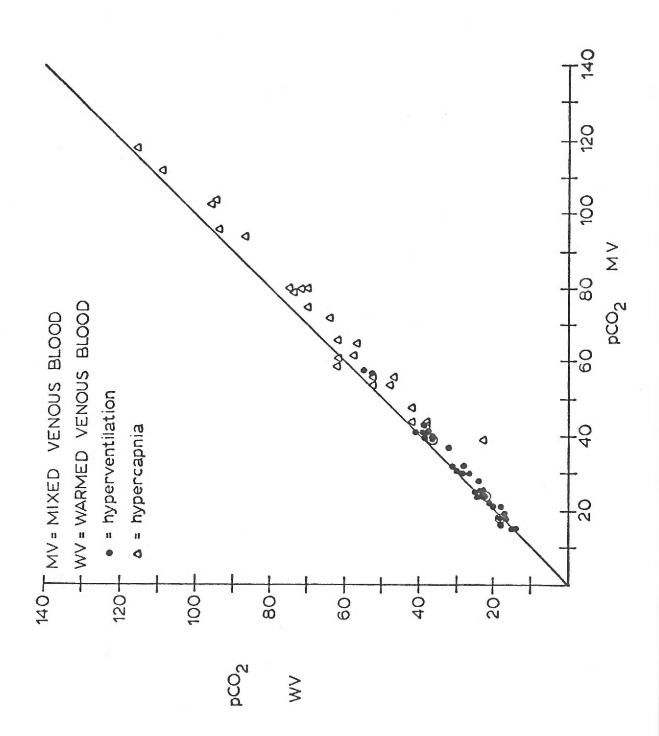


during respiratory acidosis and alkalosis. Hyperventilation Each symbol represents one Identity relationship is shown for 60 minute mixed venous and unwarmed venous pH values obtained from nine animals pair of values. The identity line indicates a slope and was used to produce hypocapnia. correlation coefficient of 1.0. Figure 11.

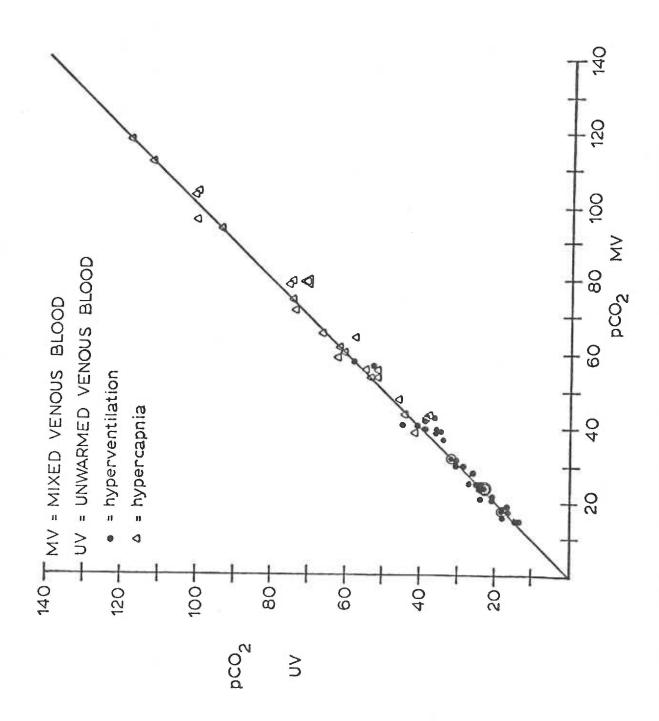


to produce hypocapnia. Each symbol represents one pair of values. The identity line indicates a slope and correlation coefficient respiratory acidosis and alkalosis. Hyperventilation was used Identity relationship is shown for 60 minute mixed venous and warmed venous pCO_2 values obtained from nine animals during Figure 12.

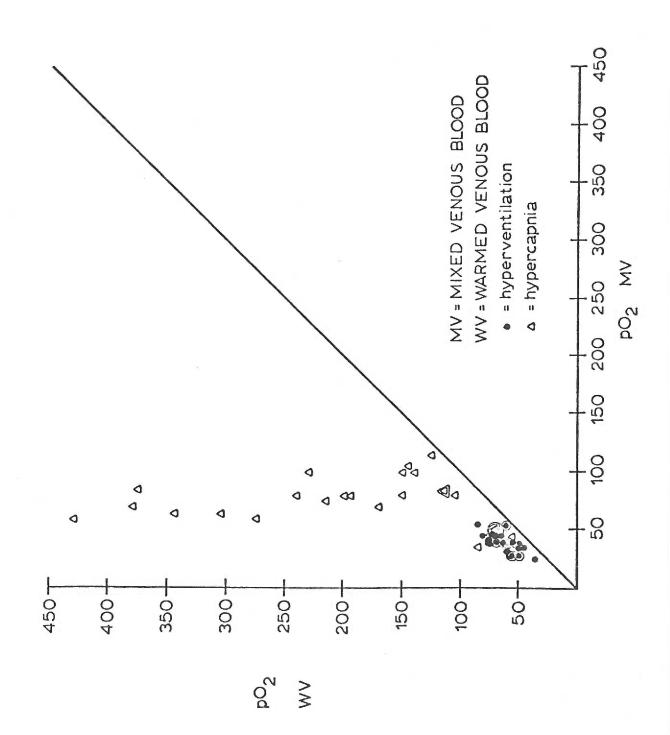
of 1.0.



to produce hypocapnia. Each symbol represents one pair of values. The identity line indicates a slope and correlation coefficient respiratory acidosis and alkalosis. Hyperventilation was used unwarmed venous $p{
m CO}_2$ values obtained from nine animals during Identity relationship is shown for 60 minute mixed venous and of 1.0. Figure 13.

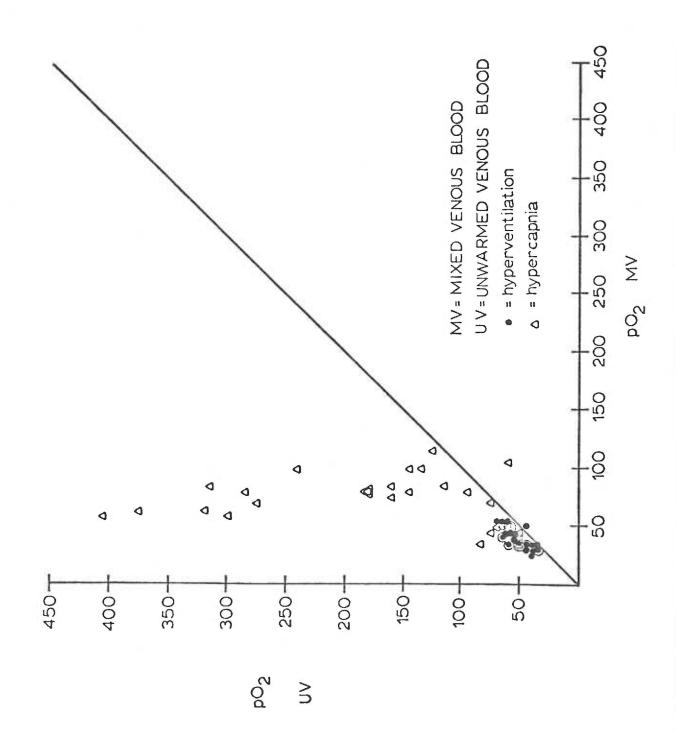


respiratory acidosis and alkalosis. Hyperventilation was used to produce hypocapnia. Each symbol represents one pair of values. The identity line indicates a slope and correlation coefficient Identity relationship is shown for 60 minute mixed venous and warmed venous pO_2 values obtained from nine animals during Figure 14.

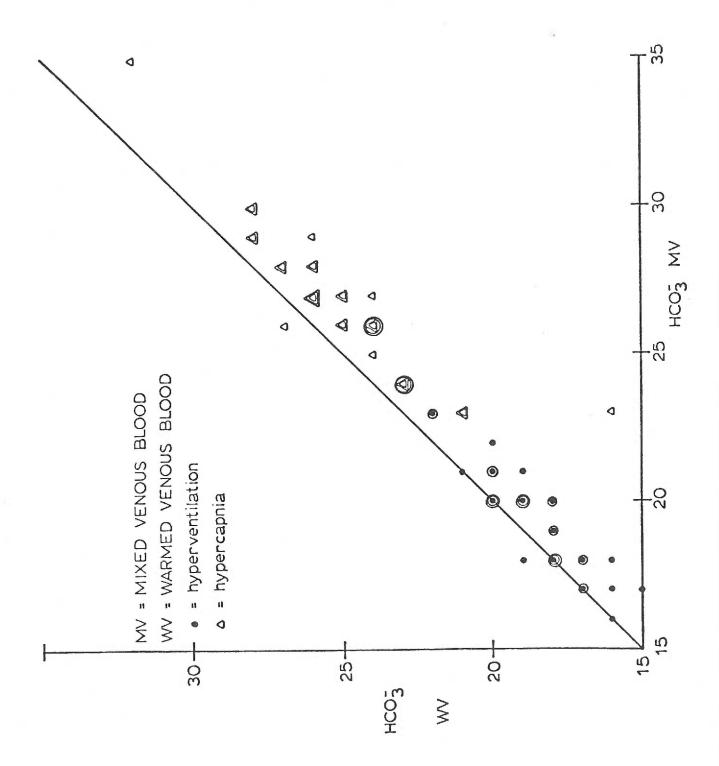


Identity relationship is shown for 60 minute mixed venous and unwarmed venous pO_2 values obtained from nine animals Figure 15.

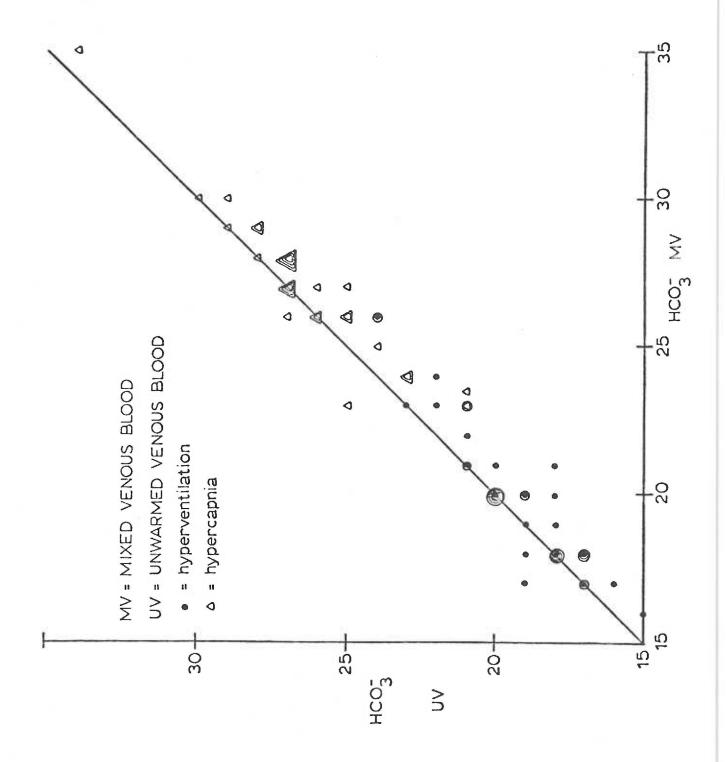
during respiratory acidosis and alkalosis. Hyperventilation was used to produce hypocapnia. Each symbol represents one pair of values. The identity line indicates a slope and correlation coefficient of 1.0.



animals during respiratory acidosis and alkalosis. Hyperventilation was used to produce hypocapnia. Each symbol represents one pair of warmed venous HCO_3 concentrations calculated from data from nine Identity relationship is shown for 60 minute mixed venous and values. The identity line indicates a slope and correlation coefficient of 1.0. Figure 16.



represents one pair of values. The identity line indicates a Hyperventilation was used to produce hypocapnia. Each symbol and unwarmed venous HCO_3^- concentrations calculated from data from nine animals during respiratory acidosis and alkalosis. Identity relationship is shown for 60 minute mixed venous slope and correlation coefficient of 1.0. Figure 17.



CHAPTER V

DISCUSSION

The following discussion is organized according to the two major comparisons posed in the results:

- Comparisons between arterial blood (A) and peripheral venous blood [warmed (WV) and unwarmed (UV)].
- Comparisons between mixed venous blood (MV)
 and peripheral venous blood [warmed (WV) and un warmed (UV)].
- 1. Arterial Blood vs Peripheral Venous Blood (WV and UV) pH, pCO₂ and [HCO₃]

The results of this study show that peripheral venous blood (both WV and UV) correlates closely with arterial blood in terms of the blood-gas parameters of pH, pCO $_2$ and [HCO $_3$] during controlled respiratory acid-base disturbances. This finding is of particular importance as pH, pCO $_2$, and hence, [HCO $_3$] are the essential parameters used in the determination of acid-base status. The use of free-flowing peripheral venous blood in lieu of arterial blood to assess these parameters during respiratory acid-base disturbances appears to be valid on the basis of these results.

It is apparent from visual inspection of Tables 6 and 7 that there are no significant differences between a

given set of parameters in warmed and unwarmed venous blood. This finding differs from that of Carveth (1979), who compared the blood-gas parameters of arterial blood to those of warmed and unwarmed venous blood during controlled metabolic acid-base disturbances. In her study, it was found that warming (arterializing) the peripheral venous sampling site produced blood-gas values which more closely approximated arterial values than did those values obtained from an unwarmed venous site. Similar results were also reported by other previous investigators (Brooks, 1959; Collis & Neaverson, 1967; Goldschmidt & Light, 1925; Harrison & Galloon, 1965).

The absence of a significant difference between warmed and unwarmed venous blood in the present study may be due to a difference in the placement of the venous catheter. In the Carveth study, superficial veins in the forepaws were used for the collection of peripheral venous samples. These superficial veins drained skin, as opposed to deeper tissues. Warming (or arterializing) the skin caused blood flow to increase disproportionately in relation to the metabolic activity of the surrounding structures. Such an increase in flow caused the (warmed) venous blood composition to be more similar to that of arterial blood.

However, in the present study, a somewhat deeper vein in each forepaw was used. Hence, flow was greater than

would be expected from one of the more superficial veins of the skin. Warming (arterializing) the venous sampling site did not appear to increase flow significantly in relation to the low metabolic activity of the surrounding tissues. Thus, as flow was not altered as appreciably by warming, the blood-gas values obtained from the warmed and unwarmed venous sites were quite similar.

As discussed earlier, substituting peripheral venous blood for arterial blood may be particularly advantageous in pediatric patients and those critically ill adult patients requiring placement of an arterial catheter or multiple arterial punctures for use in repeated acid-base assessment. Indeed, a host of risks, as well as complications, from arterial puncture might be avoided by using peripheral venous blood obtained from a single venipuncture or indwelling venous catheter.

While the results of this study indicate that warming the venous sampling site may not be necessary if the venous catheter is placed in a relatively deep vein, one should consider the clinical implications of such a procedure. In most patients, indwelling venous catheters are placed in one of the superficial veins on the dorsum of the hand for reasons of accessibility, convenience and ease of movement. As demonstrated in the Carveth study, warming such superficial regions of the skin produces venous samples with a

blood-gas composition more like that of arterial blood.

 $\underline{p0}_{2-}$

In these experiments, the pO_2 values obtained from warmed and unwarmed peripheral venous blood proved to be an unreliable indicator of arterial pO_2 . The comparison certainly appears more favorable when arterial pO_2 is less than 150 mm Hg. This finding is similar to that of Forster, et al (1972), whose results show a close correlation between arterial and peripheral venous pO_2 at arterial pO_2 values below 70 mm Hg. Due to the shape of the oxyhemoglobin dissociation curve, at arterial pO_2 values greater than approximately 80 mm Hg, larger arteriovenous pO_2 differences occur. These differences were not lessened significantly by warming (arterializing) the peripheral venous sampling site in the present study. Therefore, the use of peripheral venous blood for determining pO_2 (and hence, pulmonary function) appears to be of limited usefulness.

2. Mixed Venous Blood vs Peripheral Venous Blood (WV and UV) pH, pCO₂ and [HCO₃]

In the present study, the blood-gas parameters of pH, pCO₂ and [HCO₃] of mixed venous blood also correlated closely with those of peripheral venous blood (both WV and UV). Refer to Figures 10-13, 16 and 17. Careful inspection of the correlation coefficients (Table 6) reveals that the

UV samples correlated slightly better with the MV samples than did those obtained from WV blood for these parameters. This finding may be due to a slight alteration in the WV samples as a result of the warming (arterialization) procedure.

As discussed above, the blood-gas parameters of pH, pCO_2 and $[HCO_3^-]$ are essential to the definitive assessment of acid-base status. The results of this study indicate that peripheral venous blood may be a reliable indicator of these parameters in mixed venous blood. The value of determining the pH, pCO_2 and $[HCO_3^-]$ of mixed venous blood lies in an understanding of the potential clinical usefulness of mixed venous blood.

Mixed Venous Blood

The acid-base status of a given individual is ultimately determined by the pH, pCO₂ and [HCO₃] of interstitial fluid (ISF). However, blood rather than ISF is generally used to assess these parameters. Arterial blood provides an input to the systemic capillaries from the pulmonary circulation. In the systemic capillaries, gas exchange and equilibration with the surrounding ISF occurs. It follows that the output of these systemic capillaries should more accurately represent the pH, pCO₂ and [HCO₃] of the ISF than does the arterial input. Mixed venous blood obtained from the pulmonary artery provides a flow-weighted average of this

output from perfused systemic capillaries.

On the other hand, arterial blood, representing the output from the lungs, undergoes gas exchange with alveoli. Thus, the blood-gas composition of arterial blood provides information regarding pulmonary function.

Therefore, it appears reasonable to conclude that mixed venous blood should provide the more accurate index concerning systemic (or total body) acid-base status.

Furthermore, if the blood-gas composition of peripheral venous blood is a reliable indicator of that found in mixed venous blood, then peripheral venous blood may prove to be a useful tool for evaluating systemic acid-base status.

pO₂₋

In general, peripheral venous pO_2 values (both WV and UV) did not correlate as well with those of mixed venous blood as did other blood-gas parameters (Figures 14 and 15). Correlation coefficients varied widely during the hypercapnia experiments. This was probably due to the fact that the animals were breathing an oxygen (as well as carbon dioxide) enriched gas mixture, producing very high arterial values of pO_2 . Mixed venous pO_2 values decreased considerably more than those of peripheral venous blood. It should be remembered that mixed venous blood drains tissues of both high and low O_2 uptake. In contrast, the peripheral venous

samples were obtained from veins draining tissues of low metabolic activity (and hence, low $\mathbf{0}_2$ consumption). Consequently, the reduction in peripheral venous $\mathbf{p0}_2$ was not as great as that observed in mixed venous blood.

It should be noted that during the hyperventilation (hypocapnia) experiments, r values were greater than those obtained during hypercapnia. In addition, pO₂ values obtained from unwarmed venous blood correlated more closely with those of mixed venous blood than did those obtained from warmed venous blood (Table 8).

The pO_2 values obtained during hyperventilation in this study deserve further scrutiny. It can be seen in Table 5 that while the arterial pO_2 increased during maximum hyperventilation with room air, the change was not significant. Conversely, the pO_2 of mixed venous blood decreased substantially during maximum hyperventilation. There are at least two possible explanations for this decrement in mixed venous pO_2 :

- 1. An increase in the uptake of oxygen due to an increase in metabolic activity may have caused mixed venous $p0_2$ to fall.
- 2. A decrease in cardiac output (C.0.) would have decreased delivery of oxygen to the tissues. Hence, the tissues would have extracted a greater per cent of O_2 , producing a decrement in mixed venous O_2 .

The first of these possibilities, an increase in metabolic activity, does not appear likely for a number of reasons. During hyperventilation experiments, the composition of the inspired gas mixture was held constant (room air). Further, the animals were anesthetized, curarized and mechanically ventilated. Consequently, minimal energy expenditure was required of each animal in order to maintain a constant alveolar ventilation rate. addition, body temperature (rectal) did not change significantly during the experiments. Thus, it is reasonable to assume that metabolic activity, and hence, oxygen consumption, remained relatively constant. Therefore, changes in mixed venous pO, in the hyperventilation experiments were probably due to changes in cardiac output. Positive pressure ventilation, as was employed in these experiments. may have caused a decrease in venous return, and thereby decreased cardiac output. The observed reduction in mixed venous pO, may be the result of decreased oxygen delivery to the systemic tissues, despite an increase in arterial p02.

Regardless of which of these two mechanisms played a role in this study, it can be seen from the results of the hyperventilation experiments that peripheral venous blood may be useful in predicting the direction of change of mixed venous pO_0 .

CHAPTER VI

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS Summary and Conclusions

Historically, arterial blood has been used in blood-gas analysis to assess pulmonary function and acid-base status. The many disadvantages and complications associated with arterial puncture led to investigations concerning the reliability of substituting peripheral venous blood for arterial blood in blood-gas analysis. In addition, recent investigations have explored the usefulness of mixed venous blood as a predictor of systemic acid-base status. All of these studies were conducted within a relatively narrow pH range.

In the present study, the blood-gas composition of peripheral venous blood (both warmed and unwarmed) was compared to that of arterial blood and mixed venous blood over a wide pH range during controlled respiratory acid-base disturbances.

Respiratory acidosis was induced in five experimental animals by sequentially increasing the amount of ${\rm CO}_2$ in the inspired gas mixture. Hyperventilation by means of increasing tidal volume was used to produce respiratory alkalosis in five dogs. Samples of arterial blood, warmed and unwarmed peripheral venous blood, and mixed venous blood were drawn simultaneously, and the blood-gas composition

determined during the states of respiratory acidosis and alkalosis.

The results of these experiments show that peripheral venous blood (warmed and unwarmed) correlate closely with both arterial and mixed venous blood for the blood-gas parameters of pH, pCO $_2$ and HCO $_3$ during induced respiratory acid-base disturbances. Peripheral venous blood did not prove to be useful in predicting arterial pO $_2$ when ${}^{\rm F}{}^{\rm I}{}_{\rm O2}$ was greater than 0.21. The pO $_2$ of peripheral venous blood was found to correlate more closely with that of mixed venous blood than with arterial pO $_2$.

It can be concluded from the results of these experiments that the pH, pCO_2 and HCO_3 of free-flowing peripheral venous blood is a reliable indicator of respiratory acidbase status. Thus, based upon the results of both this study and that on metabolic disturbances by Carveth (1979), peripheral venous blood can be used in lieu of arterial blood in the assessment of acid-base disorders.

Recommendations for Further Study

The following questions remain for further investigation:

1) What is the relationship between peripheral venous blood and mixed venous blood when cardiac output is artificially reduced either via hemorrhage or mechanical ventilation?

- 2) In the clinical setting, how does the blood-gas composition of peripheral venous blood obtained from a superficial hand vein compare with that of arterial blood?
- 3) Similarly, in a clinical setting, how does the blood-gas composition of peripheral venous blood obtained from a deeper vein draining muscle as well as connective tissue (such as a femoral or subclavian vein) compare with that of mixed venous blood obtained from critically ill patients?

REFERENCES

- Brooks, D., & Wynn, V. Use of venous blood for pH and carbon-dioxide studies. Lancet, 1959 1, 227-230.
- Carveth, L.I. The use of free flowing peripheral venous blood in assessing acid-base status. Unpublished Master's Thesis, University of Oregon Health Sciences Center, 1979.
- Cole, P., & Lumley, J. Arterial puncture. British Medical Journal, 1966, 1, 1277-1278.
- Collis, J.M., & Neaverson, M.A. Arterialized venous blood. British Journal of Anaesthesia, 1967, 39, 883-886.
- Davenport, H.W. <u>The ABC of Acid-Base Chemistry</u>. 6th Edition. The University of Chicago Press, Chicago, 1974.
- Forster, H.V., Dempsey, J.A., Thomson, J., Vidruk, E., & DoPico, G.A. Estimation of arterial pO₂, pCO₂, pH and lactate from arterialized venous blood. <u>Journal of Applied Physiology</u>, 1972, <u>32</u>, 134-137.
- Gambino, S.R. Collection of capillary blood for simultaneous determination of arterial pH, CO₂ content, pCO₂ and oxygen saturation. American Journal of Clinical Pathology, 1961, 35, 175-183.
- Goldschmidt, S., & Light, A.B. A method of obtaining from veins blood similar to arterial blood in gaseous content. Journal of Biological Chemistry, 1925, 64, 53-58.
- Harrison, E.M., & Galloon, S. Venous blood as an alternative to arterial blood for the measurement of carbon dioxide tensions. British Journal of Anaesthesia, 1965, 37, 13-18.
- Hofford, J.M., Dowling, A.S., & Pell, S. Arterial blood-gas measurements: usefulness and limitations of arterialized capillary bloods. <u>Delaware Medical</u> Journal, 1973, 45, 8-11.
- Keyes, J.L. Blood-gases and blood-gas transport. Heart and Lung, 1974, 3, 945-954.

- Lilienthal, J.L., & Riley, R.L. On the determination of arterial oxygen saturation from samples of capillary blood. <u>Journal of Clinical Investigation</u>, 1944, <u>23</u>, 904-906.
- MacIntyre, J., Norman, J.N., & Smith, G. Use of capillary blood in measurement of arterial pO₂. British Medical Journal, 1968, 3, 640-643.
- Macon, W.L., & Futrell, J.W. Median-nerve neuropathy after percutaneous puncture of the brachial artery in patients receiving anticoagulants. New England Journal of Medicine, 1973, 228, 1396.
- Meakin, L., & Davies, H.W. Observations on the gases in human arterial and venous blood. Journal of Pathology and Bacteriology, 1920, 23, 451-461.
- Neviaser, R.J., Adams, J.P., & May, G.I. Complications of arterial puncture in anticoagulated patients. The Journal of Bone and Joint Surgery, 1976, 58a (2), 218-220.
- Nicholls, J.C. Complications of arterial puncture. British Medical Journal, 1964, 2, 877.
- Paine, E.G., Boutwell, J.H., & Soloff, L.A. The reliability of "arterialized" venous blood for measuring arterial pH and pCO₂. The American Journal of Medical Sciences, 1961, 242, 431-435.
- Pape, K.E., Armstrong, D.L., & Firshardinge, P.M. Peripheral median nerve damage secondary to brachial arterial blood-gas sampling. The Journal of Pediatrics, 1978, 93 (5), 852-856.
- Selkurt, E. Physiology, 4th Edition. Little, Brown and Company, Boston, 1976.
- Slonim, N., & Hamilton, L. Respiratory Physiology, 3rd Edition. C.V. Mosby Company, St. Louis, 1976.
- Stern, J., Kaplan, S., & Furman, E.B. Arterialized venous oxygen tensions during paediatric anaesthesia. South African Medical Journal, 1973, 47, 1878-1879.
- Wintrobe, M.W., Thorn, G.W., Adams, R.D., Braunwald, E., Isselbacher, K.J., & Petersdorf, R.G. <u>Harrison's</u>

 <u>Principles of Internal Medicine</u> 7th Edition. McGraw-Hill Brook Company, New York, 1974.

APPENDIX A

Definition of Terms

Definition of Terms and Variables

- 1. pH: The pH of a solution is defined as the negative logarithm of the hydrogen ion [H+] activity in that solution: pH = -log [H+] or log 1/[H+] (Keyes, 1976; Slonim & Hamilton, 1976).
- 2. pCO_2 : The pCO_2 is the partial pressure of CO_2 in a solution. It is proportional to the amount of CO_2 that is physically dissolved in that solution (Keys, 1976; Slonim & Hamilton, 1976).
- 3. pO_2 : The pO_2 is the partial pressure of O_2 in a solution. It is proportional to the amount of O_2 that is physically dissolved in that solution (Keys, 1976; Slonim & Hamilton, 1976).
- 4. [HCO₃]: The bicarbonate concentration was calculated according to the Henderson-Hasselbalch equation:

a) pH = pK'a + log
$$[HCO_3^-]$$

 $\overline{S \cdot pCO_2}$

consequently,

b) $[HCO_3^-] = (10^{(pH - pK'a)}) (S \cdot pCO_2)$ where pK'a = 6.1 at 37° C., and S = 0.0301 m Moles of CO_2/mm Hg pCO_2 (Selkurt, 1976)

- 5. Arterialized venous blood is produced when a venous sampling site is warmed to $35-45^{\circ}$ C., leading to vasodilation and increased blood flow to the area. This increase in blood flow causes increased 0_2 delivery, providing venous blood with a composition resembling that of arterial blood (Collis & Neaverson, 1967).
- 6. Respiratory acidosis is an acid-base disturbance resulting when the production of volatile acids (${\rm CO}_2$) exceeds the excretion of volatile acids (Keyes, 1976; Slonim & Hamilton, 1976).
- 7. Respiratory alkalosis is an acid-base disturbance resulting when the loss of volatile acids (CO_2) exceeds the production of volatile acids (Keyes, 1976; Slonim & Hamilton, 1976).

APPENDIX B

Protocol for Calibration of Electrodes

APPENDIX B

Calibration Technique

The BMS 3 Mark 2 blood-gas analyzer consists of three units. The BGA 3 component contains the electrodes. The GMA 2 precision gas supply is used for calibration of the pCO $_2$ electrode. The PHM 73 pH/Blood-Gas monitor provides a digital readout for pH, pCO $_2$ and pO $_2$.

The pH, pCO_2 and pO_2 electrodes were calibrated in order to assure that each individual electrode was responding within specific limits.

Preparation for calibration

The blood-gas analyzer was turned on at least one hour prior to calibration in order to allow the water bath to reach and maintain a temperature of 37°C $^{+}$ 0.05 $^{\circ}$ C.

Barometric pressure was measured using a mercury barometer and then corrected for temperature. The corrected barometric pressure was used as a reference to calibrate the blood-gas analyzer. Specific calibration values for ${\rm pCO}_2$ and ${\rm pO}_2$ at that pressure were obtained from tables supplied with the BMS 3 Mark 2 system.

The membranes for the ${\rm pCO}_2$ and ${\rm pO}_2$ electrodes were changed each week just prior to calibration.

pH Electrode

Two buffers having pH values of $7.383 \, -0.005$ and

 $6.841 \, ^{+}0.005$ were used to calibrate the pH electiode initially. Repeat calibration was performed using the pH 7.383 buffer whenever necessary.

pCO₂ Electrode

The pO_2 electrode was calibrated using two different solutions: one which was oxygen free, and the other being thermostat water which was equilibrated with air.

Note: Refer to the BMS 3 Mark 2 Manual for specific calibration procedures.

APPENDIX C

Raw Data from Nine Experimental Animals:

Values for Blood-Gas Parameters

Obtained during

Hypercapnia and Hyperventilation

Dog #2 January 17, 1980

Хg
6
15
H
Weight

Hematocrit: beginning 46.2 0

Dog #3 January 24, 1980 Weight = 10.9 kg

		₩	27.17	25.72	27.80	28.40	29.59	31.21	35.10	29.29	30.39	28.03	26.88	,
Į,		3	25.62	26.22	26.81	28.21	28.85	30.46	34.17	26.20	29.65	26.35	25.25	
HCO, mEq/L	,	MV	27.10 26.28	24.78 25.71	26.49 27.29	26.41 27.97	29.0 28.43	28.63 30.93	33.67 32.03	26.01 27.14	28.52 28.36	25.19 25.75	24.90 25.22	
	+	4	47.0 2	77.0 2				64.7 2		85.0	0.0	70.3	67.7	
		UV MV	51.0 47	0.00	238.7 102.3	258.0 101.0 0.0	58.7 106.0	52.0 6	123.0 114.0	302.7 8	286.7 7	7 7.712	276.0 6	
po mm Hg	7	WV	56.0	140.0	149.0	207.0	143.0	99.0	127.0	335.7	195.0	168.7	380.0	
		A	75.7	469.7	469.7	453.3	427.0	245.7	177.3	432.7	458.0	518.3	483.7	
		MV	54.4	60.5	78.6	92.2	118.2	103.7	243.6	122.5	100.8 103.3	83.0	79.6	
5		3	53.7	63.1	75.8	91.6	117.9	127.4 103.7	226.5	104.9 122.5	100.8	74.5	71.4	
рСО лим На	7	WV	52.6	59.1	73.7	90.8	116.2	115.3	212.3	108.4	96.4	74.5	69.7	
		æ	51.8	54.4	73.2	83.8	113.2	_	228.4	103.9	90.5	69.6	68.8	4
		W	7.298 7.319	7.241 7.254		7.114 7.108			6.798 6.776	7.020 6.997	7.094 7.094	12.0	7.169 7.150	2.4
1	pu	WV UV	7.319 7.0.001 0					5-	1		3			1
		*	rol kr 7.335 SD 0.002	i	180	124	7.033	02 7.036	6.788	7.024	7.118	7.184	7.185	10.00
		Period	Cont.	20" 3% CO ₂ 7.284 SD 0.0	60" 3% CO ₂ SD	20" 5% CO ₂ 7. SD 0	60" 5% CO ₂ , 7.	20" 10% CO2	60" 10% CO ₂ 6.788 50 0.001	20" 51 CO ₂ 7.	60" 51 CO2	20" 38 CO ₂ 7.184 SD 0.002	31 CO2	de la
		Time Samples Analyzed	1052	1126	1158	1252	1335	1442	1526	1621	1707	1758	1820	
		Time		1049	1131	1213	1255	1335	1415	1451	1534	1608	1650	

KEY: A = Arterial blood
 Wy = Warmed venous blood
 Uy = Unwarmed venous blood
 My = Mixed venous blood

42

35, end

Hematocrit: beginning

Dog #4 January 31, 1980 Weight 14.1 kg

				Hd				рсо ₂ птн9	пНд			pO2mmHg	Hg		HCO3	mEq/L	
Time Samples Drawn	Time Samples Analyzed	Period	K	W	3	W	4	M V	3	W	4	¥.	3	¥	¥ W	3	\$
1012	1056	Control Rm Air	7.407	7.383	7.387	7.396	41.4		44.6		1.2	67.7			. 29	2	26.30
1058	1128	20" 3% CO ₂		7.268	7.266	7.287	52.0	57.4	56.7		481.7	40	153.0	4 .	25.26 25.44	1 25.0	23.76
1134	1202	60" 3% CO ₂		7.255	7.254	7.266	58.7	62.4	63.0	59.2		106.0 1	181.0	81.0	27.34 26.86	6 27.02	26.09
1200	1234	20" 5% CO2		7.188	7.205	7.199	65.1	71.4	68.7	69.2	467.3	1	145.0	85.3	25.84 26.2	9 26.35	26.16
1240	1308	60 " 58 CO ₂ SD	7.200	7.184	7.179	7.183	69.2	70.4	74.8	74.8	- 1	- 4		78.0	26.25 25.71	1 27.01	27.24
1305	1345	20" 10% CO ₂ SD		7,105	7.114	7.122	80.4	88.5	85.2	85.3	4	0 0	127.0		26.30 26.95	5 26.48	27.0
1355	1427	60" 10% CO ₂ SD	7.033	7.03	7.022	7.035	108.3	0 4	112.2	111.5	432.0	0 0		4	27.94 27.93	3 28.22	28.9
1420	1459	20" 5% CO ₂		7.111	7.09	7.089	83.0	85,8	88.7	91.7		136.3 1	127.0	85.0	26.27 26.41	8 26.10	26.90
1500	1536	60" 5% CO ₂	7.117	7.10	7.082	7.093	87.5	86.7		93.6	100		115.0	87.0	27.40 26.10	27.11	27.73
1525	1610	20" 3% CO ₂		7.114	7.116	7.115	77.77		83.6	86.2	463.7	0.0	130.7	82.7	25.64 26.03	3 26.10	26.87
1605	1642	98 CO 2 SD		7.149	7.161	7.142	70.0		75.4		451.0		159.7		25.15 25.10	6 26.12	26.45
1631	1710	20" Rm Air SD		7.238	7.234	7.217	63.6	8.09	61.5		48.2	66.8	60.6		26.35 25.1	5 25.22	26.76
1710	1731	60" Rm Air SD	7.327	7.318	7.309	7.298	47.2	48.5	51.5	54.5	79.7	67.3	68.6	48.6	23.96 24.10	3 25.10	25.89

KEY: A = Arterial blood
WV = Warmed venous blood
UV = Unwarmed venous blood
MV = Mixed venous blood

Dog #5 February 7, 1980 Weight = 11.4 kg

				Hd	_			pco2 m	mmHg			ро2 ш	pmmHg		нсо	1 -	mEq/L	
Time Samples Drawn	Time Samples Analyzed	Period	4	PW.	3	W	<	ΛM	5	М	4	W	3	£		5		¥
1022	1051	Control Rm Air	7.360 7	7.364		7.353	ω,	e, .	7	9 .	83.6	73.7	73.2	45.4	21.25 21.	.17	21.29 2	22.96
1055	1118	20"	7.295	7.286	7.294	7.261	47.4	49.1	48.4	54.6	474	421.3	345.3	65.6	22.35 22	68	22.77 2	3.81
1135	1157	50 CO SD 60 "		7.267	7.266	7.249	9 .	-0 0	4.	56.3	459.7		318	65.0	22.79 23.	43	23.12 2	23.88
1200	1221	20" 5% CO ₂	7.206 7.194	1_	7.198	7.182	60.8	62.0	62.4	68.4	458	274.3	334.3		23.36 23	23.17	23.54 2	4.87
1238	1300	60" 5% CO ₂	7.230 7.215		7.226	7.199	8.09	0 -		66.1	74			61.3		32	- 1	24.99
307	1333	20" 10% CO ₂ SD	7.092	7.072	7.085	7.078	4 0 4			94.0	9	8 -	295.7	77.7	8	20	1	26.90
344	1405	60" 10% CO ₂ SD	1	7.048	7.048	0.001	97.1	95.4		. 4		હું ન		- 4	. 59		- 1	26.96
1410	1434	20" 5% CO ₂	0.002 0.003	7,160	7.157	7,126				82.2		355.3	371.3			12		26.27
450	1512	60" 5% CO ₂ SD		7.158	7.161	7.141			71.3		530.7	8 4		64.3	24.61 24.	6	24.70 2	26.30
1516	1537	20" 3% CO ₂ SD		7.223	0.001	0,001	54.5	9.7.9	· d	: 3	. 10	0.6	386.3	9,00	2	;		
1600	1618	20" 3% CO2 SD		7.221	0,001	7.194	55.B 0,1	- 4	58.1		2 5	4 a	2.0	0.6	و ا	_ 1.	7	76.37
1625	1649	20" Rm Air SD	100	7,325	0.001	0.001	42.3		42.7	50.1	89.5				7 1	_		23.85
1700	1722	60" Rm Air SD	0.002 0.00		7,366	7.339	38.1	38.5	38.3	0.4	102.8	84.7	85.9 0.5	24.9	21.40 21	81.12	21.21	7:17
XEY: A		blood	puo								Неп	Hematocrit:		beginning	41; end	3 41		91

WV = Warmed venous blood UV = Unwarmed venous blood AV = Mixed venous blood

Dog #6 February 14, 1980 Weight = 13.6 kg

		47	Т	89	- _T	4		. 36		26	T	8.55	1	4.4		61	7	96		90	T	0 2		75		81	٦	04
	Ŋ.	25.47		22.68		20.44		20	- 1	17.56		6 18.		17.44		17.61		17.96		18.		20.02		5 21.75		1 22.		
1/6	3	24.54		22.04		19.92		18.93		18.72		17.2		17.26		17.08		17.56		17.94		19.02		20.16		20.91		42
HCO3 mEq/L		43	,	22.29		9.53		18.68		18.62		17.23		17.21		85		17.18		8.48		9.83		21.09		23		
FC 5	WV	24				-	1		- 1				1			3 16.	- 1			-		-	- 1			2 21.		end t
	K	24.22		21.43		20.98		18.60		17.42		17.10		16.20		16.0		16.92		16.94		18.32		19.94		20.32		44.57
	MV	50.2	0.1	40.5	0.2	40.7	0.1	38.2	0.5	37.1	0.2	34.6	0.1	32.9	0.3	33.5	0.1	35.6	0.3	38.3	0.5	36.8	0.2	39.7	0.1	40.7	0.3	beginning 5
mmHg	3	45.9	0.2	67.4	0.4	56.4	0.0	50.4	0.1	41.8	0.1	47.4	0.1	36.9	0,1	64.9	0.2	58.6	0.1	55.6	0.1	60.5	0.0	57.3	0.1	60.1	0.2	P
po ₂ mm	WV	62.9	0.1	58.7	0,3	61.4	0.0	54.7	0.4	48.7	0,2	53.9	0.2	44.4	0,2	48.1	0,1	50.3	0,1	52.5	0.1	50.9	0.1	62.4	0,3	63.8	0.1	
	A	88.4	0.3	94.5	1	107.4	0.1		2	00	2	107.4	0.3	102.9	0.3	98.4	0.2	97.2	0.4	107.7	0.4	97.0	0.3	86.1	0.0	90.4	0.2	Hematocr Protein:
		9.	7	ε.	-	23.6 1	_	20.1	=			17.9	0.0	5.0		6.8	0.0	8.4		22.8	170	25.0	0.3	34.5	0.3	38.6	6-3	
	MV	40	d	27	d		٩			-	a		٦	-			Ĭ	1										
Нд	3	44.6	0 2		9	23.0	0	18.6	0.0	18.4	0	17.0	0	15.4	0.2	16.1	0.1	17.7	10	21.0	0.1	23.8	0.1	32.2	0,3	35.3	0.2	
pco ₂ mmHg	WV	39.5	0.1	26.7	0.1	22.5	0.2		p.1	18.3	0.1	16.7	0.0	15.0	0	17.1	0.1	17.8	0.1	22.7	0.1	25.3	0.0	33.3	0.3	36.6	0.2	
	A	37.9	1.0	23.3		23.4	0.2	17.5	0.0	16.2	0.0	15.5	0.1	12.3	0	14.6	0	16.1	0	19.5	0.1	21.4	0.0	31.7	,	33.3	0.2	
		.419	0	141	0.002	7.559	003	_	0.0	7.651	0.0	7.637	0.001	7.687	0 00 0	7.642	0	4 40	0.002	7.540	0.0	7.525	0.003	7.421	200 0	7.393	0.001	
	WV	-	1				O												i									
	3	7.362	0.001	7.548	0.00	7.559	0.003	7.629	0.001	7.629	0.001	7.628	0.00	7.671	00	7.647	00	7.618	0.002	7.553	0.003	7.524	0.001	7.418	0	7.394	0.002	
Hď	VW	7.416	0.003	7.543	0.001	7.560	0.001	7.621	0.002	7.629	0.002	7,635	0.003	7.681	100	7.615	0.001	7.606	0.003	7.532	0.001	7.517	0.001	7.423	F 00 0	7.391	0.001	
	V	7.427	0.001	_	0.001		0.001		0.001		0.002	7.664	0.001	7.741	100	7.662	0 001	7.643	0.003	7.567	0.003	7.554	0	7.420	0		0.001	ood blood od
			-	350 7	Sp	350 7	0.0	500	SD	200	Sp	650	CS		C	200	C		CS.	350	C.	-	S	+	2	7200	SD	ood us bl
	Period	Control	SD SD	20"TV350	RR 12	60"TV350 7.574	RR 12	20"TV500 7.648	71 VV	60"TV500 7.653		20 "TV650	NH TO	60"TV650	RR 12	20"TV500	RR 12	60"TV500	KK 12	20"TV350	RR 1	60"TV350	KK 12	20"TV200	RR 12	60"TV200	RR 1	Arterial blood Warmed venous blood Unwarmed venous blood Mixed venous blood
	Time Samples Analyzed	1028		1103		1140		1208		1244		1 308		1353		1425		1451		1518		1602		1627		1656		
	Time Samples Drawn	1 00 5		1030		1108		1135		1214		1241		1321		1347		1427		1452		1532		1557		1637		XEY: W A

Dog 47 February 21, 1980 We1ght = 15.9 kg

	1			Hd				pco ₂ mmHg	19			PO ₂ mmHg	61		HCO3 mEq/L	7/52	
Time				A L	21	NA NA	4	W	λn	WV	A	WV	3	W.	A WV	UV MV	
Analyzed 0957	U	Control Rm Air	7.314	284	7.282	7.269	47.4	52.7	53.4	57.3	69.1	53.5	54.3	39.7	23.35 24.23	24.44 25.4	υ ,
1027		20"TV350 RR 12 SD		7.379	7.378	0.0	37.4	39.8 0.1	40.1	43.2	78.1	47.9	49.5	40.2	22.7	89 2	
1105	1	60"TV350 RR 12 SD		7.414	7.411	7.414	33.5	37.3	36.3	39.2	73.5	48.5	43.6	36.3	10 23.1	36 2	- ·
1137		20 "TV500 RR 12 SD		1 1	7.452	7.457	25.8	30.3	32.3	33.8	76.3	44.6	40.1	29.6	. 81 21.	21.87	w 3
1214		60"TV500 RR 12	7.527	7.509	7.454	7.470	25.2	27.5	31.8	32.2	74.4	53.6	34.1	28.6	28 21.	21.63	9
1241		20"TV650 RR 12 SD		1 1	7,525	7.456	19.8	23.6	25.7	26.5	75.8	37.6	36.2	24.6	9.37 19.7	20.58 22	2 1 2
1321		60"TV650 RR 12 SD		7.541		7.557	19.6	24.3	25.1	24.7	72.5	36.4	37.9	27.0	9.40 20.	20.86	5 5
1352	1	20"TV500 RR 12 SD	7.542	7.492	7.487	7.496	22.4	25.8	26.0	27.5	98.5				8.66 19.	19.08 20	2 6
1428		60"TV500 RR 12 SD	7,518	7.502	7.478	8 7.480	22.9	24.0	25.7	28.0	77.5	53.3	0.1	00 .		18.47 20	77
1458		20"TV350 RR 12 SD	7.414	7.404	7.378	8 7.392 1 0.001	30.6	31.2	33.2	34.2		6.0	m o .	34.6	8 9 6 6	10.33	
1530		60"TV350 RR 12 SD	7.399	7.377	7.360	1 0.001	31.1	32.4	34.4	37.3	62.3	53.4	90		18.01.01	60.01	2 8
1600		20"TV200 RR 12 SD	7,233	3 7.223	7.211	1 7.218	48.3	47.7	52.8	52.9	49.2	0.5	60	1	19.75.19.	20.52 20	6 9
1633	_	60"TV200 RR 12 SD	7.217	7 7.186	1 0.001	73 7.170	49.3	54.7	58.1	58.5	77.0	61.3	0.4	4.4	19.43 20.0	S. Br end	2 %
43	are l	A = Arterial blood WV = Warmed venous blood	blood										Hematocriti Protein:		beginning	5.5; end	93

KEY: A = Arterial blood
Wy = Warmed venous blood
Uy = Unwarmed venous blood
My = Mixed venous blood

Dog #8 February 28, 1980 Weight = 16.8 kg

	The state of the last of the l	-													
			Hď			PCO ₂ mmlig	lg			pO ₂ mmHg	Нд		нсо3	HCO3 mEq/L	
Time Samples	Time	3	4. A.A.	VW VIII	A	ΔM	3		K	W	3	ΔW	A WV	Ŋ	MV
1112	1142	1	.364	349 7	6.7	1.	36.5	39.5	132.9	0.09	59.9	42.0	21.30 20.81	1 19.72	21.09
		RR 12 SD	0.0	- 1	0.1		0.1	0.2	0.2	0.2	0.2	0.0	10 41 10 44	18 44	70.00
1137	1209	20"TV400	7.470 7.430	7.429 7.425	26.5	29.1	73.4	23.5	0.16	7	53.1	7 . 2		4	
		KK 12 SD	0.001 0.0	0.00100.0	0.0	0.1	0.1	0.2	0.1	0.1	0,3	0.2		- 1	10 77
1220	1247	60"TV400	1				30.9	29.6	87.5	96.4	51.3	36.6	18.05 17.58	8 19.43	10.11
		RR12 SD	0.001		0.1		0.2	0.1	0.4	0.5	0.2	0.2	- 1	1	10 67
1247	1316	141	7.542 7.51	7.457 7.494	20.6	23.1	27.2	7.97	74.3	7.00	39.5	33.2	17.36 19.04	1.61	10.04
		RR 12	0.0	0.001 0.002	0.0		0.1	0.3	0.1	0.2	0.1	0.1	- 1		0
1325	1353	60"TV550	7.551 7.517	7.460 7.499	-	22.8	27.1	25.4	1.76	20.2	35.6	32.4	16.49 17.82	2 18.41	18.18
		RR 12		0 0 0 001	0.0	0.1	0.2	0.0	0.3	0.2	0.2	0.1			1
1354	1418	20"TV700	7.595 7.562	7.480	17.4		25.8	22.5	0.66	50.9	34.0	31.8	15,95 16,36	6 18.52	18.23
		RR 12	100	0.002 0.001	0.1	0.0	0.0	0.0	0.4	0.1	0.0	0.1			- 1
1435	1452	00LAL.09	7.587 7.558	7.518	16.6		23.7	21.4	6.86	51.7	40.1	31.5	15.23 15.10	0 19.44	16.97
		RR 12	0	0 001 0 001	0.0	0.1	0.2	0.1	0.1	0.0	0.3	0.0			
1459	1523	20"TV550	1	7.454	21.2		26.2	24.6	1.96	51.1	41.1	33.5	16.20 16.63	3 16.89	17.73
		RR 12	0000	0.001 0.0	0.1	0.0	0.1	0.1	0.2	0.1	0.1	0.1			1
1538	1600	60"TV550	7.499 7.472	7.457	21.7	22.8	23.4	23.8	94.9	54.8	43.3	33.8	16.25 15.89	15.8	6 TP 6
		RR 12 SD	0.0 0.002		0.1		0.2	0.1	9.0	0.0	0.0	0.1	7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7	00 21 4	3000
1604	1625	20 "TV400	7.427 7.40	7.398		27.9	9.87	31.2	00.00	0.4.0	64.4	28.9	10.46 10.01		
		RR 12 SD		0.001		0.0	0.2	0.1	0.1	0.2	0.1	0.3	1000		10 13
1642	1702	TV6	7.402 7.37	7.372 7.389	27.5	30.8	32.9	31.9	83.0	7.85	46.5	36.8	16.29 11.12		
		RR 12	0.001	0.001		0.1	0.1	0.3	0.2	0.3	0.3	0.1		1	
1707	1725	20"TV250	7.324 7.306	7.296	35.9	34.9	37.7	39.4	80.5	60.5	50.5	43.0	18.05 17.12	12 17.41	16.81
		RR 12 SD	0.001 0.001	0.002 0.001	0.1	0.2	0.2	0.1	0.2	0.2	0.2	0.0			
KEY:	A = Arterial blood WV = Warmed vehous blood	= Arterial blood = Warmed venous b	lood						Hema	Hematocrit: Protein: b	9	beginning jinning 7	40; end 7	42	

KEY: A = Arterial blood
WV = Warmed venous blood
UV = Unwarmed venous blood
MV = Mixed venous blood

38

Hematocrit: beginning 40; end Protein: beginning 6; end 6

Dog #9 March 6, 1980 Weight = 13.2 kg

od A WV UV MV HV HV </th <th></th> <th></th> <th></th> <th></th> <th>нф</th> <th></th> <th></th> <th>рсо₂mmн9</th> <th>0Hu</th> <th></th> <th></th> <th>pO₂mmHg</th> <th>ıHg</th> <th></th> <th></th> <th>HCO3_mEc</th> <th>mEq/L</th> <th></th>					нф			рсо ₂ mmн9	0Hu			pO ₂ mmHg	ıHg			HCO3_mEc	mEq/L	
1027	Time amples Drawn	Time Samples Analyzed	Period	A WV			æ	WV	λ'n	νM	A	WIV	Ŋ	МУ	Æ	1	5	WV
1107 20"TVJ50 7.525 7.503 7.493 7.514 21.0 26.4 25.2 2.2 1142 60"TVJ50 7.566 7.539 7.528 7.547 21.4 23.5 24.3 2 2 1142 60"TVJ50 7.566 7.539 7.528 7.547 21.4 23.5 24.3 2 2 1208 20"TVJ50 7.643 7.593 7.606 7.612 15.8 18.4 18.3 1 1252 60"TVJ50 7.643 7.593 7.606 7.612 15.8 18.4 18.3 1 1323 20"TVJ50 7.643 7.615 7.611 7.628 16.3 17.8 17.6 1 1324 20"TVJ50 7.676 7.648 7.615 7.611 7.628 16.3 17.8 17.6 1 1325 60"TVJ50 7.676 7.648 7.657 7.679 14.9 15.0 15.1 1 1326 60"TVJ50 7.676 7.648 7.651 7.670 0.10 0.1 0.1 0.1 0.1 1424 20"TVJ50 7.659 7.653 7.651 7.657 15.7 15.6 15.6 1 1508 60"TVJ50 7.659 7.652 7.631 7.627 15.7 15.6 15.6 1 1508 60"TVJ50 7.644 7.515 7.618 7.524 20.8 21.4 22.5 2 1516 60"TVJ50 7.544 7.515 7.518 7.524 20.8 21.4 22.5 2 1516 60"TVJ50 7.542 7.520 7.518 7.515 21.8 21.8 21.4 22.5 2 1517 1616 60"TVJ50 7.542 7.520 7.515 7.515 21.8 21.8 21.8 23.4 2 1518 18 12	1002	1027	TV200 RR 12	7.381		00.00	41.5				94.6	62.6	62.1	52.6	23.86		6	23.29
1142 60"TVJ50 7.566 7.539 7.528 7.547 21.4 23.5 24.3 2 2. 1208 20"TV504 7.641 7.593 7.606 7.612 15.8 18.4 18.3 11 1252 60"TV504 7.648 7.615 7.611 7.628 16.3 17.8 17.6 1 1. 1323 20"TV504 7.648 7.615 7.611 7.628 16.3 17.8 17.6 1 1. 1324 20"TV504 7.648 7.615 7.611 7.628 16.3 17.8 17.6 1 1. 1325 60"TV504 7.676 7.648 7.644 7.649 14.9 15.0 15.1 1 1. 1326 60"TV505 7.676 7.648 7.644 7.649 14.9 15.0 15.1 1 1. 1327 20"TV504 7.659 7.663 7.661 7.670 12.5 14.3 13.8 1 1. 1328 20"TV507 7.659 7.663 7.661 7.670 12.5 14.3 13.8 1 1. 1424 20"TV507 7.659 7.652 7.631 7.627 15.7 15.6 15.6 1 1. 1508 60"TV507 7.635 7.615 7.606 7.607 17.1 16.6 17.3 1 1. 1543 20"TV507 7.544 7.535 7.518 7.524 20.8 21.4 22.5 2 2. 1544 20"TV507 7.542 7.520 7.518 7.524 20.8 21.4 22.5 2 2. 1545 RR 12	1042	1107	20"TV350 RR 12	0.002 0.00			21.0	26.4	50	24.9	120.7	76.3	61.4	48.3	9	0.10	18.75	19.44
1208 20 TV500 7.643 7.593 7.606 7.612 15.8 18.4 18.3 17.8 12.5	1122	1142	60"TV350 RR 12 SD	0.003 0.0			21.	23.5	24.3	23.7	115.5	65.2	57.6	8 0		4	10	19.97
1252 60"TV500 7.648 7.611 7.628 16.3 17.8 17.6 17.8 13.2 1323 20"TV550 7.676 7.648 7.644 7.649 14.9 15.0 15.1 1 1323 20"TV650 7.676 7.648 7.644 7.649 14.9 15.0 15.1 1 1352 RR 12	1148	1208	20"TV500 RR 12	.643			15.	18.4	18.3	17.9	118.9	55.9	56.7	4 0	16.60	53	17.66	17.52
1323 20"TV650 7.676 7.644 7.649 14.9 15.0 15.1 1.0 1352 60"TV650 7.659 7.663 7.661 7.670 0.2 0.0 0.0 1352 60"TV650 7.699 7.663 7.661 7.670 12.5 14.3 13.8 1 1424 20"TV500 7.650 7.652 7.631 7.627 15.7 15.6 15.6 1 1508 60"TV500 7.650 7.652 7.631 7.627 15.7 15.6 15.6 1 1543 20"TV500 7.635 7.615 7.606 7.607 17.1 16.6 17.3 1 1543 20"TV350 7.644 7.535 7.518 7.524 20.8 21.4 22.5 2 1647 20"TV200 7.544 7.535 7.515 7.515 21.8 21.8 23.4 2 1647 20"TV200 7.355 7.346 7.337 7.335 36.4 37.5 36.8 4	1227	1252	60"TV500 RR 12	0.001 0.0			16	17.8	17.6		120.4	62.5	56.4	45.2	17.33	7.54	17.18	17.97
1352 60"TV650 7.699 7.663 7.661 7.670 12.5 14.3 13.8 1	1259		20"TV65U RR 12 SD	7.676 7.6			14.	0.0	15.1	16.1		0.2	56.7		· 6	5.95	5.0	7.1
1424 20"TV500 7.650 7.622 7.631 7.627 15.7 15.6 15.6 1 1508 60"TV500 7.635 7.615 7.606 7.607 17.1 16.6 17.3 1 1543 10"TV350 7.644 7.535 7.518 7.524 20.8 21.4 22.5 2 1543 10"TV350 7.544 7.535 7.518 7.524 20.8 21.4 22.5 2 1616 60"TV350 7.544 7.535 7.518 7.515 21.8 21.8 23.4 2 1647 20"TV200 7.542 7.520 7.515 7.515 21.8 21.8 23.4 2 1647 20"TV200 7.393 7.369 7.373 7.385 34.6 35.7 35.2 3 1735 60"TV200 7.355 7.346 7.335 7.337 36.4 37.5 36.8 4	1332		60*TV650 RR 12 SD	0.002 0.0				14.3	13.8	14.7	130.3	0.1	0.1	38.5	14.94	5. /4	15.12	16.44
1508 60"TV500	1400	1424	20"TV500 RR 12	7.650 7.6	120	1000		15.6	15.6	16.7	128,4	0 0	0.1	38.6	16.77	5.62	15.95	16.92
1543 10"TV350 7.544 7.535 7.518 7.524 20.8 21.4 22.5 2 1616 60"TV350 7.542 7.520 7.515 7.515 21.8 21.8 23.4 2 1647 20"TV200 7.393 7.369 7.313 7.385 34.6 35.7 35.2 3 1735 60"TV200 7.355 7.346 7.335 7.337 3.337 36.4 37.5 36.8 4	1440	1508	60"TV500 RR 12	0.001 0.0	101 0.001		17.	16.6	17.3	19.0	129.3	0.1	0.1		68.71		10.00	17.13
1616 60"TV350 7.542 7.520 7.515 7.515 21.8 21.8 23.4 2 RR 12 SD 0.001 0.001 0.0 0.002 0.2 0.1 0.3 1647 20"TV200 7.393 7.369 7.373 7.385 34.6 35.7 35.2 3 RR 12 SD 0.001 0.0 0.003 0.001 0.1 0.2 0.1 1735 60"TV200 7.355 7.346 7.335 7.337 36.4 37.5 36.8 4	1515	1543	20 TV350 88 12 50	0.002 0.0	0.002		20.	21.4		24.4	123.4	0.1	0.2				17.02	27.71
1647 20"TV200 7.393 7.369 7.373 7.385 34.6 35.7 35.2 3 RR 12 SD 0.001 0.0 0.003 0.001 0.1 0.2 0.1 1735 60"TV200 7.355 7.346 7.335 7.337 36.4 37.5 36.8 4	1551	1616	60"TV356 RR 12 SD	0.001 0.0	0.0 0.0		21.	21.8	÷ 0	23.8	. 4	0.1	0.5	41.9	; ;	6.63	: !	61.11
1735 60"TV200 7.355 7.346 7.335 7.337 36.4 37.5 36.8 4	1625	1647	20"TV200 RR 12 SC	0.001 0.0	169 7.37	100		35	35.2	36.4	108.5	0.1		0.0			18.35	19.79
ST 0.001 0.0 0.001 0.001 0.2 0.1 0.2	1705	1735	60"TV200 RR 12 St	0.001 0.0		8 2		37	36.8	43.4	107.4	0.3	0.1	0.2	18.4	13.03	18.15	20.36

XEY: A = Arterial
WV = Warmed venous blood
UV = Unwarmed venous blood
MV = Mixed venous blood

-				Hd				pCO ₂ mmHg	няд			PO ₂ mmHg	иHg		HCO3 mEq/L
Time Samples Drawn A	Time Samples Analyzed	Period	æ	WV	3	MV	«	W	3	W	×	ΛM	N.	MV	A WV UV MV
0957	1001	TV 350 RR 12	7.351	7.343	7.344	7.341	39.7	38.3	39.1	41.6	74.5	72.3	61.6	50.9	21.30 20.17 20.64 21.81
		CS	0.0	0.001	0.0	0.001	0.1	0.2	0.2	0.1	0.3	0.3	0.1	0.2	
1026	1039	20" TV 500 RR 12	7.447	7.439	7.348		29.1	30.7		34.6	19.6	75.5	0.09		19.47 20.17 19.53 21.91
		SD	0.001	0.001	0.001	0.0	0.2	0.1	0.0	0.1	0.0	0.3	0.2	0	
1107	1114	60" TV 500 RR 12	7.468	7.459	7.454	7.453	27.6		28.9	29.8	77.7	80.3		45.6	19.39 18.9919.65 20.22
1135	1146	20" TV 650	7.547	7 536	7 536	7 519	21.6	21.7	22 4	73.7	73.4	68.2	5.2.4	42 B	18.20 17.82 18.40 18.72
		RR 14 SD	0,001		0.001	0.001	0.1	0.2	0.1	0.0	0.2	0.1	0.2	0.2	
1216	1221	60" TV 650 RR 14	7,562	7.568	7,556	7.552	20.6	20.8	20.8	21.8	75.4	0.69	53,3	40.6	17.97 18.3917.89 18.
		SD	0.001	0.0	0.001	0.001	0.1	0.1	0.1	0.1	0.2	0.3	0.2	0.1	
1240	1251	20" TV 750 RR 16	7.585	7.594	7.575	7.577	17.0	16.5	18.8	19.2	72.7	61.7	49.3	38.2	15.63 15.4916.89 17.33
1320	1329	60" TV 750	7.621	7.614	7.609	7.607	16.1	16.9	17.7	18.1	68.3	a.	46.0	37.0	16.08 16.61 16.62 17.51
		RR 16	_		00.0	0.001	0 0	. 0	0 0	-	0 2	0.3	4.0	0.2	
1345	1354	20" TV 650	7.576	1	7.563	7.564	19.3	19.5		21.2	75.4	66.5	53.5		17.38 17.01 17.22 18.
		KK 14 SD	0,002	- 10	- 10	0.001	0.1	0.0	0.0	0.1	0.3	0.0	0.3	0.3	
1425	1435	60" TV 650 RR 14	7,574	7.5	~ 0	7.555	19.8	20.5	21.0	20.7	76.5	68.1	54.3	41.5	17.75 18.00 18.19 17.76
1450	1503	20" TV 500 RR 12	-	7.472	7.467	7.469	26.6	27.4	27.7	28.5	B2.4		54.4	43.8	19.21 19.42 19.41 20.06
			-	0.0	0.001	0.0	0.0	0.1		0.0	0.0	0.2	0.3	0.1	
1530	1540	60" TV 500 RR 12 SD		7.447	7.434	7.431	28.7	29.8	30.7	30.6	84.4	72.9	59.6	47.1	19.52 19.9419.94 19.74
1557	.1606	20" TV 350 RR 12 SD	7.360	7.3	7,341	7.346	36.8	37.9	38.6	40.4		71.5	61.4	50.5	20.16 19.87 20.24 21,43
1637	1644	60" TV 350 RR 12	_	7.3	7,322	7.317	37.3	37.3	39.2	40.3	84.2	72.9	59.0	50.7	18.94 18,8519.67 19.99
KEY; A	- Arteria	Arterial blood		414			1			Неша	Hematocriti	beginning		36; end	d 42

XEY: A = Arterial blood
WV = Warmed venous blood
UV = Unwarmed venous blood
MV = Mixed venous blood

APPENDIX D

Summary Table of "r", Slope and Intercept
For Each Animal

Summary Table of 'r', Slope and Intercept for Each Animal

		Нd			pco ₂			ро ₂ ттН9	HCO3	-	MIC
	AVSWV	AvsUV M	MVVSWV M	MVvs UV	AvsWV AvsUV	MVVSWV	MVvsUV	AVSWV AVSUV MVVSWV MVVSUV	AVSWV AVSUV	MANASWA MAN	ומי
HYPERCAPNIA Dog 2 r	0.951	0.928	0.997	0.999	0.996 0.982 1.047 0.911 -2.60212.671	2 0.990 1.173 1-17.641	0.995 1.040 -1.671	0.926 0.735 0.886 0.738 0.375 0.209 3.304 1.935 28.615 35.067 -75.501-29.62	0.837 0.901 0.838 0.274 3.867 20.397	0.960 0 2.000 0 -29.838 1	.919 .582 1.352
intercept Dog 3 r	0.996		96	0.997	8 0.		0.998 0.921 3.612	0.670 0.707 -0.118 -0.046 0.419 0.434 -0.498 -0.194 28.85821.066217.574188.96	0.978 0.966 0.757 1.066 55 6.519-1.782	0.977 0 0.747 1 5.908 -	0.996 1.084 -3.612
Dog 4 r	0.997	1	1	0.995 1.009	97 0.99 78 1.01 17 3.57	9 0.990 6 0.959 8 -0.309	0.994 0.998 -0.214	0.749 0.907 0.605 0.730 0.186 0.215 1.433 1.658 51.397 50.167 6.925 -1.089	0 0.931 0.949 8 0.873 0.658 9 2.884 9.399	0.731 0 0.915 0 1.129 5	
Dog 5 r slope	0.999			0.998	0.993 0.99 0.965 1.02 2.176-0.91	1 0.988 5 0.913 3 0.333	0.992 0.975 -3.226	0.943 0.990 0.801 0.747 0.661 0.664 7.267 6.44 18.198 14.391-158.827-113.	2 0.970 0.993 9 0.833 0.969 133 3.771 0.822	0.945 0 1.036 1 -2.282 -6	206 6. 232
HYPERVENTILATION Dog 6 r 0.	TION 0.995	0.982	0.998	0.983	0.986 0.984 0.986 1.108	4 0.996 8 0.909	75	530-0.244 0.879 647-0.343 1.325	3 0.930 0.965 6 0.841 0.840	0.973 (0.825 (0.962
intercept Dog 7 r	0.989 0.942	0.995		0.998	326 993 096	- 00	993	0.685 (0.522 (0.	0.980 0.92 1.043 0.89 -0.438 2.79	979 034 112	94.90
intercept Dog 8 r	0.398 0.998 0.944	0.979	0.989	0.986	90 0.	6 0.9 2 1.1 34-4.9	000	.342 0.542 0.924 0. .073 0.264 0.902 2. .953 20.796 23.145-24	0.916 0.426 0.827 0.274 3.220 13.830	0.974 1.267 -5.831	0.567 0.525 8.883
intercept Dog 9 r slope	0.999		, 000	0.998	95 0. 50 0. 25 2.	996 0.985 953 0.899 197 1.072	0.9	.057-0.502 0.149 0. .038-0.187 0.193 0. .816 82.550 61.479 40.	4 0.776 0.88 4 3.897 1.87	0.974	96.96
Dog 10 r slope intercept	0.999 1.004 -0.032	0.999 0.983 0.113	0.999 1.016 -0.114	1.000 0.997 0.027	0.998 0.998 0.936 0.98 1.950 1.30	95 0.997 89 0.893 09 1.437	0.996 0.945 0.715	0.608 0.584 0.6/6 0.87 0.618 0.461 0.735 0.74 23.199 21.563 38.081 23.94	43 0.712 0.824 44 5.368 3.505	5.066	0.858 2.307
KEY: A V.S. A V.S. MV V.S.	# # # # # # # # # # # # # # # # # # #	Arterial Arterial Mixed ver Mixed ver		vers lood	warmed venc unwarmed versus warmed	ous blood snous bloo venous b	od lood blood us blood				98

AN ABSTRACT OF THE THESIS OF

ALLAHNA O'CONNOR SCHRIVER

For the MASTER OF NURSING

Date of Receiving this Degree: June, 1981

Title: THE USE OF FREE-FLOWING PERIPHERAL VENOUS BLOOD

IN THE ASSESSMENT OF RESPIRATORY ACID-BASE

DISTURBANCES

Approved:

Jack L. Keyes, Ph.D., Thesis Advisor

Arterial blood has traditionally been used in blood-gas analysis to assess acid-base status and pulmonary function. Investigations have been conducted regarding the feasibility of substituting peripheral venous blood for arterial blood in blood-gas analysis. In this study, the blood-gas composition of peripheral venous blood (warmed and unwarmed) was compared to that of both arterial blood and mixed venous blood during controlled respiratory acid-base disturbances in dogs. Specifically, the usefulness of peripheral venous blood as an indicator of the pH, pCO₂, pO₂ and [HCO₃] of arterial and mixed venous blood over a wide pH range was explored.

ABSTRACT OF THESIS CONTINUED

Respiratory acidosis was induced in five dogs by increasing the inspired ${\rm CO_2}$ from that in room air to 10% ${\rm CO_2}$ in ${\rm O_2}$ during mechanical ventilation. States of respiratory alkalosis were achieved by increasing the tidal volume setting on the ventilator in 150 ml increments from an initial range of 200-350 ml to 750 ml. Samples of arterial blood, warmed and unwarmed peripheral venous blood, and mixed venous blood were drawn simultaneously and the blood-gas composition determined.

The results of these experiments show that peripheral venous blood (warmed and unwarmed) correlate closely with both arterial and mixed venous blood for the blood-gas parameters of pH, pCO $_2$ and [HCO $_3$] during induced respiratory acid-base disturbances. Peripheral venous blood did not prove to be useful in predicting arterial pO $_2$ when ${}^{\rm FI}{}_{\rm O2}$ was greater than 0.21. The pO $_2$ of peripheral venous blood was found to correlate more closely with that of mixed venous blood than with arterial pO $_2$.

It can be concluded from the results of the experiments that the pH, pCO_2 and $[HCO_3^-]$ of free-flowing peripheral venous blood is a reliable indicator of respiratory acid-base status. Thus, based upon the results of both this study and that on metabolic disturbances by Carveth (1979),

ABSTRACT OF THESIS CONTINUED

peripheral venous blood can be used in lieu of arterial blood in the assessment of acid-base disorders.