THE USE OF PERIPHERAL VENOUS BLOOD TO ASSESS ACID-BASE STATUS WHEN CARDIAC OUTPUT IS REDUCED

by

Paula Maurer Felden, R.N., B.S.

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APPROVED:

[redacted]

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

[redacted]

Wilma Peterson, Ph.D., Associate Professor, First Reader

[redacted]

Mary McFarland, M.S.N., Associate Professor, Second Reader

[redacted]

Carol A. Lindeman, Ph.D., Dean, School of Nursing

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CHAPTER I

INTRODUCTION

Arterial blood-gas composition is widely used clinically to assess cardiopulmonary function and acid-base status of critically ill patients. The use of arterial blood alone to assess acid-base status is, however, now being challenged (Roos & Thomas, 1967; Tung, Bettice, Wang & Brown, 1976). In fact, recent research indicates that mixed venous blood provides as accurate an assessment of acid-base status as does arterial blood (Griffith, McKenzie, Peterson & Keyes, In preparation).

Since arterial blood-gas composition is determined primarily by the ratio of alveolar ventilation to pulmonary blood flow, anything that interferes with the process of perfusion, diffusion of gases themselves and/or alveolar ventilation will produce changes in arterial blood-gas composition. Oxygenation blood returns from the pulmonary circulation and is mixed in the left side of the heart, thus circulating systemic arterial blood is of uniform composition. Hence, arterial blood-gas composition is a convenient, sensitive indicator of cardiopulmonary function.

In contrast to arterial blood, venous blood-gas composition is determined primarily by the ratio of tissue metabolic activity to tissue perfusion. Tissues have fluctuating, non-uniform ratios of metabolic activity to blood flow (Smith & Kampine, 1980). Therefore, systemic venous blood-gas composition is also non-uniform. However, upon reaching the pulmonary artery, venous blood has been thoroughly mixed in the right ventricle and is of uniform composition. The blood-gas

composition of mixed venous blood is a flow-weighted average of peripheral venous blood-gas compositions. Mixed venous blood-gas composition, then, seems to be the more logical indicator of systemic acid-base status than that obtained from arterial blood.

The physiological instability of critically ill patients necessitates frequent reassessment of their cardiopulmonary function and acid-base status. Currently, arterial blood-gas composition is widely used for both these assessments. It is routine for critically ill patients to have their arteries repeatedly punctured for sampling purposes. Decreased arterial sampling and increased mixed venous sampling can be anticipated if mixed venous blood proves efficacious in determining acid-base status. Unfortunately, acquisition of both kinds of blood samples can result in serious, sometimes life-threatening iatrogenic injuries to patients (Boontje, 1978; Puri, Carlson, Bander & Weil, 1980).

Peripheral venous blood is generally easier to obtain than arterial or mixed venous blood. Additionally, fewer hazards and complications are associated with venous sampling as opposed to arterial or mixed venous sampling. Because of these qualities, several groups of investigators (Collis & Neaverson, 1967; Schriver, 1981) have examined the use of peripheral venous blood in lieu of arterial or mixed venous blood (Schriver, 1981). The overall aim of this study is to determine whether peripheral venous blood can be used in lieu of arterial or mixed venous blood for blood-gas analysis. The following theoretical framework provides the basis for much of this reasearch.

Theoretical Framework

The model in Figure 1 depicts arterial and venous blood as flowing in parallel, but opposite directions within the body. Arterial blood represents output from the lungs or pulmonary circulation to systemic tissues and organs. Schematically, systemic tissues and organs (head, viscera, limbs, etc.) are depicted as parallel circuits within the systemic circulation. Venous blood represents the output from the systemic tissues and organs to the lungs. What the lungs add and remove (O_2 and CO_2 respectively) establish the physiological difference between systemic arterial and venous blood.

The differences between arterial and peripheral venous blood-gas compositions can be minimized through active hyperemia. Active hyperemia describes the process whereby microcirculatory vessels dilate in response to heat resulting in increased arterial and subcutaneous venous flow (Smith & Kampine, 1980). The following equations taken from Schriver, (1981) quantitate the relationships between peripheral venous and arterial blood-gas compositions:

$$\dot{Q}\ \{O_2\}a = \dot{Q}\ \{O_2\}v + \dot{V}O_2 \qquad (1)$$

where $\dot{Q}$ = flow

$\dot{V}O_2$ = oxygen consumption,

$\{O_2\}a$ = the concentration of oxygen in arterial blood,

$\{O_2\}v$ = the concentration of oxygen in venous blood.

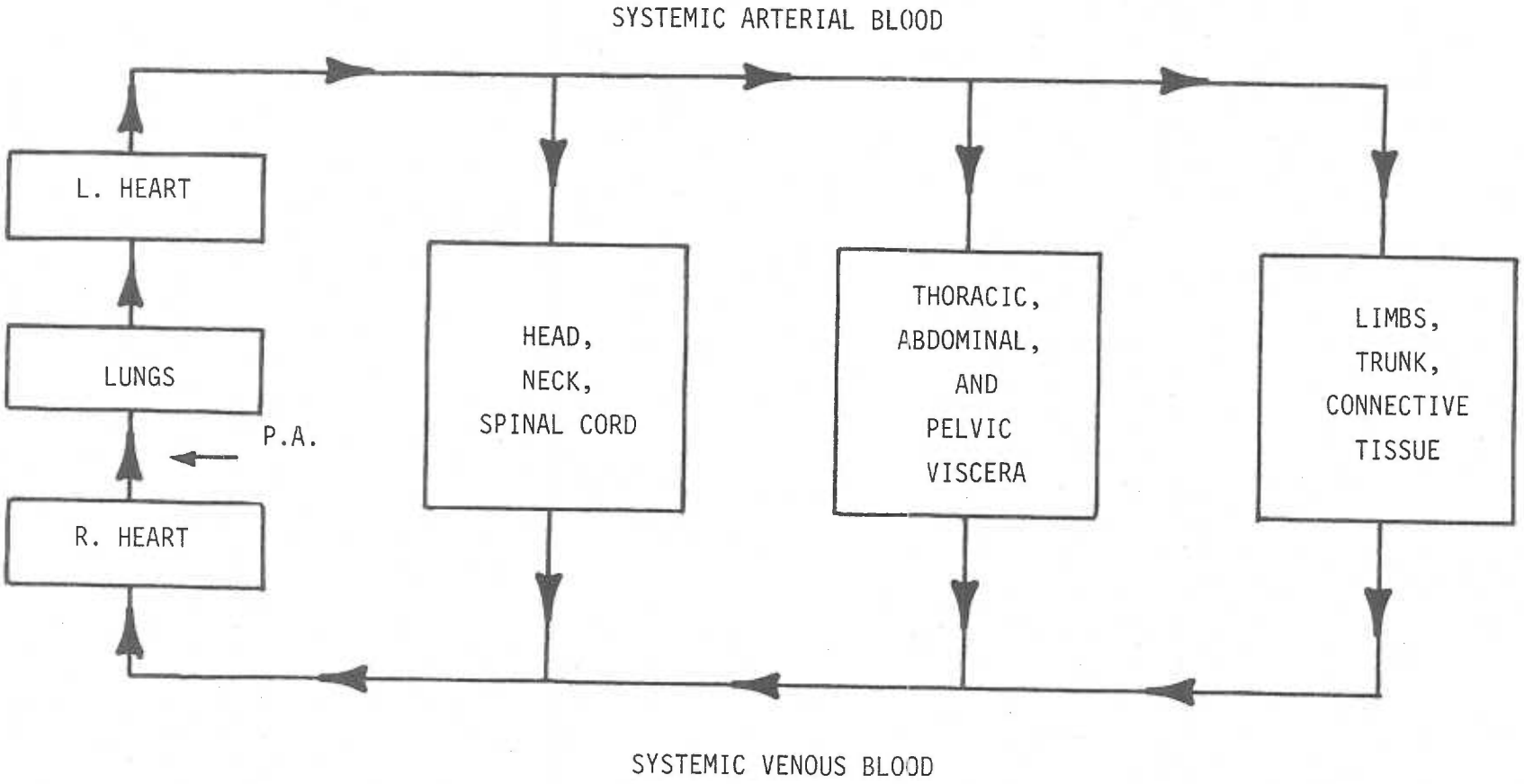


Figure 1. A model depicting the relationship between systemic arterial and venous blood. Arterial blood represents output from lungs to systemic tissues and organs. Venous blood represents output from the systemic tissues and organs to the lungs. P. A. is the pulmonary artery. (Revised from Griffith, 1981.)

$$\dot{Q}\ \{O_2\}a - \dot{Q}\ \{O_2\}v = \dot{V}O_2 \qquad (2)$$

$$\dot{Q}\ [\{O_2\}a - \{O_2\}v] = \dot{V}O_2 \qquad (3)$$

$$\{O_2\}a - \{O_2\}v = \frac{\dot{V}O_2}{\dot{Q}} \qquad (4)$$

From equation (4) it can be seen that if flow is increased relative to tissue oxygen consumption then the difference between arterial and venous O_2 concentration will decrease. Thus, under these circumstances, venous O_2 concentration may more closely resemble arterial O_2 concentration. Hence, venous blood under conditions of active hyperemia might be used to estimate the pO_2 arterial blood.

The same principle holds for CO_2 concentration as shown in equation (5):

$$\{CO_2\}v - \{CO_2\}a = \frac{\dot{V}CO_2}{\dot{Q}} \qquad (5)$$

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

$\{CO_2\}a$ = CO_2 concentration in arterial blood.

$\{CO_2\}v$ = CO_2 concentration in venous blood.

Warming the site from which the venous sample is obtained produces the vasodilation necessary to increase blood flow to tissues in disproportion to their metabolic needs. As a result, the blood-gas composition of the venous blood sample will more closely resemble that of arterial blood. The term "arterialized peripheral venous blood" is used to identify venous blood drawn from such a warmed site.

The use of peripheral venous blood in lieu of mixed venous blood to assess systemic acid-base status is just beginning to be explored.

Results from recent research (Schriver, 1981) suggest that non-arterialized peripheral venous blood may be a satisfactory substitute for mixed venous blood.

Review of the Literature

The following review of the literature includes: (1) the reported complications associated with arterial and mixed venous sampling, and (2) studies pertaining to the use of arterialized and non-arterialized peripheral venous blood as opposed to arterial or mixed venous blood respectively.

Complications of arterial sampling. A variety of iatrogenic injuries to patients may result from arterial punctures. The potential for such injuries increases as the frequency of arterial sampling increases. Arterial punctures are painful and may result in arterial spasm or damage to the vessel wall (Goldschmidt & Light, 1925; Hall, 1971; Stern, Kaplan & Furman, 1973). Specifically, injuries such as false aneurysms and arteriovenous fistulas may occur (Boontje, 1978). In addition, ecchymosis and hematoma formation may occur, especially with repeated sampling (Cole & Lumley, 1966).

Patients receiving anticoagulant therapy such as heparin and coumadin seem more prone to complications secondary to arterial sampling than patients not receiving anticoagulant therapy. Median, obturator, sciatic and femoral neuropathies may result from spontaneous hemorrhage following arterial punctures in patients receiving anticoagulants (Macon & Futrell, 1973; Neviaser, Adams & May, 1976). According to Neviaser, et al. (1976), femoral or brachial punctures on patients receiving heparin therapy may produce hematomas resulting in skin

sloughing and/or infection. Subsequent incision and drainage and/or skin grafting may be required in such cases.

Indwelling arterial catheters have alleviated the necessity of repeated arterial punctures. Unfortunately, these too are associated with complications. Factors recognized to predispose patients to complications include prolonged cannulation of the vessel, low cardiac output, large catheter size, pre-existing arterial disease and sustained local pressure required to stop arterial bleeding after the catheter has been removed (Baker, Chunprapaph & Nyhus, 1976; Hall, 1971).

Studies on the use of arterial catheters reveal that the following complications may occur: hematoma formation, hemorrhage which may require blood transfusions, arterial thrombosis with arterial occlusion which may result in severe ischemic injuries, and sepsis (Baker, et al., 1976; Hall, 1971; Puri, et al., 1980).

Complications of mixed venous sampling. Acquisition of mixed venous blood involves the more invasive procedure of introducing a Swan-Ganz flow-directed catheter into the pulmonary artery. Both the procedure and the catheter can produce serious, sometimes life-threatening complications.

Two recent prospective studies (Puri, et al., 1980; Sise, Hollingsworth, Brimm, Peters, Virgilio & Schackford, 1981) of pulmonary artery catheterizations document that complications may occur. These include pneumothorax, ventricular arrhythmias which may require treatment including direct current cardioversion, and subclavian vein thrombosis. Somewhat less serious complications which may result from this procedure include: arrhythmias not requiring treatment, puncture

of the subclavian artery or venous bleeding at the catheter insertion site, cellulitis at the insertion site, and sepsis (Puri, et al., 1980; Sise, et al., 1981).

Once a flow-directed catheter has been placed in the pulmonary artery, mixed venous blood can be obtained. However, Puri, et al. concluded that the intracardiac position of the catheter coupled with the repeated break of this closed system for samples of mixed venous blood may result in an increased incidence of catheter-related sepsis and endocarditis.

Arterialized peripheral venous blood. Meakins and Davies (1920) compared oxygen (O_2) saturation of hemoglobin in arterial and peripheral venous blood. Varying the local temperature of tissues at the sampling site was an important aspect of their study. The hand and forearm of one subject was exposed to five different temperature conditions: (1) room air, (2) cool atmosphere, (3) cold atmosphere, (4) arm immersed in a water bath at 45°C for 10 minutes prior to sampling, and (5) arm immersed in a water bath at 45°C for 20 minutes prior to sampling. Paired arterial and peripheral venous blood samples were obtained from the treated forearm after each temperature exposure.

Results of this study indicated that arterial O_2 saturation remained stable at 96.1% despite exposure of the sampling site to different temperatures. However, peripheral venous O_2 saturation varied widely in response to the different temperatures. Exposure of the sampling site to progressively lower temperatures resulted in the steady decline of peripheral venous O_2 saturation from 56.4% (room air) to 0.0% (cold atmosphere). Warming of the sampling site for 10 and

20 minutes raised the peripheral venous O_2 saturation to 94.2%. Meakins and Davies concluded that the temperature-related instability of peripheral venous O_2 saturation made it an unreliable indicator of arterial O_2 saturation. They did not comment, however, on the fact that warming the subject's sampling site produced values of peripheral venous O_2 saturation similar to those of arterial blood.

Based in part on the work of Meakins and Davies, Goldschmidt and Light (1925) investigated the use of arterialized peripheral venous blood in lieu of arterial blood to determine oxygen content (vol%), oxygen capacity of hemoglobin (vol%), O_2 saturation and carbon dioxide content (vol%).

Arterialization of the peripheral venous sampling site was achieved by immersing a hand and wrist of each subject in a water bath at 45-47^{o}C for 10-15 minutes. Peripheral venous blood samples were then obtained from the dorsal surface of the hand. Arterial blood samples were drawn from the radial or brachial arteries.

Paired samples of arterial and arterialized peripheral venous blood were obtained from six subjects for purposes of comparison. The O_2 content of arterialized peripheral venous blood ranged from 16.03 vol% to 21.20 vol% while that of arterial blood ranged from 14.56 vol% to 21.13 vol%. It should be noted that in one subject the O_2 content was lower in arterial blood than arterialized peripheral venous blood. Goldschmidt and Light attributed this paradox to the subject's heart condition, pulmonary stenosis with a patent ductus arteriosus, coupled with breath-holding at the time of arterial sampling. The subject had remained calm during the venous sampling which preceded the arterial

sampling.

The CO_2 content of arterial and arterialized peripheral venous blood were also similar. The values of CO_2 content for arterialized peripheral venous blood ranged from 29.7 vol% to 49.6 vol% compared to the range of 31.4 vol% to 49.9 vol% for arterial blood. Additionally, Goldschmidt and Light found that arterial and arterialized peripheral venous blood had comparable O_2 capacity and saturation values. They therefore concluded that peripheral venous blood, arterialized by their method, was reliable for determining arterial O_2 content, O_2 capacity and CO_2 content.

Brooks and Wynn (1959) further explored the possibility of using arterialized peripheral venous blood to determine the pH and pCO_2 of arterial blood. In this study arterialization of peripheral venous blood was accomplished by wrapping the subject's hand and forearm in 2 electric heating pads for 15 minutes. Peripheral venous blood samples were drawn from the dorsal surface of the hand when the skin temperature reached approximately $38^{o}C$. Arterial blood samples were drawn from brachial or femoral arteries.

Three sets of subjects were investigated: ambulatory, non-ambulatory, and anesthetized patients. Results from the first group of subjects, 5 ambulatory patients, indicated much smaller arteriovenous differences when peripheral venous blood was arterialized as opposed to being non-arterialized. The mean arteriovenous differences in pH and pCO_2 were 0.018 and 3.5 mmHg, respectively, using arterialized peripheral venous blood.

The second group of subjects consisted of 9 patients confined to bed prior to the test. The mean arteriovenous differences, again using arterialized peripheral venous blood, were less than in the previous group. Comparison of arterial and arterialized peripheral venous blood revealed a mean pH and pCO_2 difference of 0.002 and 0.8 mmHg respectively.

Brooks and Wynn then studied 10 anesthetized patients undergoing unspecified surgical procedures. As general anesthesia induces peripheral vasodilation, heating of the sampling site was unnecessary to achieve arterialization. The mean arteriovenous differences for this group of patients were comparable to the other 2 groups. The differences for pH and pCO_2 were 0.002 and 1.1 mmHg respectively. Brooks and Wynn noted, however, that significant arteriovenous differences would result if other factors resulted in decreased skin flow during surgery.

Brooks and Wynn concluded that the proper degree of arterialization of peripheral venous blood exists when the subject is at rest and the skin temperature of the sampling site is at least $35^{o}C$. If these conditions of arterialization were met, the authors concluded that peripheral venous blood could be used to assess the pH and pCO_2 of arterial blood.

Paine, Boutwell and Soloff (1961) also investigated the use of arterialized peripheral venous blood to assess the pH and pCO_2 of arterial blood. Venous blood was arterialized by wrapping a subject's hand in a hot towel or placing it in a container of hot water for 15 to 20 minutes. Temperatures of the water bath and the skin sampling site were not reported. All subjects were recumbent for 1½ hours prior to

sampling. Arterialized peripheral venous blood samples were drawn from the dorsal surface of the hand. Simultaneous samples of venous blood from the antecubital vein and arterial blood (vessel not specified) were drawn for comparison with arterialized peripheral venous blood.

Six subjects had tourniquets applied above the elbow and 10 ml vacuum tubes were used for blood collection. In 9 subjects, samples were drawn with heparinized syringes and with the tourniquet placed as described above. Fourteen subjects had tourniquets applied at the wrist. Finger flexion was necessary to induce vein filling in 50% of the subjects.

The following results were obtained for each of the methods. 1) The use of vacuum tubes for the collection of arterialized peripheral venous blood produced the greatest arteriovenous differences in pH and pCO_2 values. The mean pH difference between arterial and arterialized peripheral venous blood was 0.050 and that of pCO_2 was 4.9 mmHg. (Standard deviations were not reported). 2) An arteriovenous pH difference of 0.038 and pCO_2 difference of 8.5 mmHg resulted when tourniquets were placed above the elbow and heparinized syringes were used to collect the arterialized peripheral venous blood. 3) The final technique used on subjects, i.e., tourniquet above the wrist and use of heparinized syringes, proved superior to the other two methods. The authors reported the pH of arterialized peripheral venous blood to be 0.022 and the pCO_2 to be 2.86 mmHg lower than arterial blood.

Since it is highly unusual for the pCO_2 of arterialized peripheral venous blood to be lower than that of arterial blood, I recalculated the mean difference from the data reported by the authors.

The mean pCO_2 of arterialized peripheral venous blood was found to be 1.52 mmHg higher than that of arterial blood.

The values obtained for arterialized peripheral venous blood were closer to those of arterial blood than were those obtained from non-arterialized peripheral venous blood. The mean pH difference between arterial and non-arterialized peripheral venous blood was 0.11, while the pCO_2 difference was 3.4 mmHg.

According to Paine, et al., arterialized peripheral venous blood is best obtained by: warming the dorsal surface of the hand for approximately 20 minutes, then applying a tourniquet at the wrist, and finally drawing venous blood from the dorsal surface of the hand. Using this method of arterialization, they concluded that venous blood was acceptable for estimation of arterial pH values. Unfortunately, Paine, et al. stated no conclusions concerning the use of this method for estimating arterial pCO_2 values. Contrary to their report, calculations using their data indicate that arterialized peripheral venous blood provides a reasonable estimate of arterial pCO_2 values.

Harrison and Galloon (1965) further studied the use of arterialized peripheral venous blood for estimating arterial pCO_2 values. Values of pH and pO_2 were not reported. The subjects consisted of 13 patients, 12 of whom were undergoing surgery and one of whom was being ventilated by a respirator.

Peripheral venous blood was arterialized by wrapping the subject's hand in an electric heating pad which had a maximum temperature of $60^{o}C$. The authors stated that the hand was warmed to "body" temperature, but the exact skin temperature was unspecified. Peripheral venous blood

samples, arterialized and non-arterialized, were obtained from an indwelling cannula on the dorsal surface of the hand. Within 30 seconds of obtaining the venous sample, arterial samples from an indwelling catheter in the radial artery were obtained.

Peripheral venous and arterial samples were drawn at the beginning of and during anesthesia under a variety of conditions. Forty-eight paired arterial and peripheral venous samples were obtained under non-ideal conditions: cold hand with venous stasis, cold hand without venous stasis, and warm hand with venous stasis. The smallest arteriovenous difference (value not specified) in pCO_2 was observed in samples drawn at the beginning of anesthesia, hand below body temperature and without venous stasis. The authors attributed these results to spontaneous arterialization secondary to anesthesia-induced vasodilation.

Fifty-one paired samples were drawn under ideal conditions, i.e., warm hand and without stasis. The mean arteriovenous difference for pCO_2 values was 0.5 mmHg $\pm$ 0.7 mmHg SD.

Harrison and Galloon concluded that arterialized peripheral venous blood could be used clinically and experimentally to estimate arterial pCO_2 values under the following conditions: (1) venous blood must be taken from the dorsal surface of the hand, (2) the hand must be warmed to at least body temperature, and (3) there must be no stasis of venous blood flow before and during sampling.

Collis and Neaverson (1967) compared the pH, pCO_2 and pO_2 values of arterialized peripheral venous blood to those of arterial blood. Arterialization of peripheral venous blood was achieved by immersing

the subject's hand in a water bath at 45°C for 5 minutes.

Peripheral venous blood samples were drawn without venous stasis from the dorsal surface of the hand. Arterial samples were drawn from a radial artery. Blood samples were collected in heparinized syringes and immediately analyzed. The subjects were 23 ambulatory patients. In 10 cases, the arterialized peripheral venous blood was obtained first followed by the arterial blood, and in 13 cases this order was reversed.

Reversing the order in which blood samples were drawn produced no statistically significant differences in the parameters of pH and pCO_2. The pH and pCO_2 differences between arterial and arterialized peripheral venous blood were 0.0052 (SD 0.0075) and 0.76 mmHg (SD 0.81) respectively. However, pO_2 values differed by as much as 40 mmHg. Peripheral venous blood was useless as a guide to the pO_2 of arterial blood when the pO_2 of the arterial blood was greater than 60 mmHg.

These investigators concluded that arterialized peripheral venous blood provided an adequate estimate of arterial pCO_2 and pH values, but not arterial pO_2 values.

Non-arterialized peripheral venous blood. This investigator could find no published literature on human research comparing peripheral venous blood-gas composition to that of mixed venous blood. Schriver (1981) conducted such a comparison using animal subjects. Specifically, Schriver compared the blood-gas composition of peripheral venous blood, arterialized and non-arterialized, to that of mixed venous blood and arterial blood during induced respiratory acid-base disturbances.

Ten healthy dogs were anesthetized, intubated and placed on ventilators. Respiratory acidosis was induced in half of these dogs by varying the concentration of CO_2 in the inspired air. Respiratory alkalosis was induced in the remaining 5 dogs by increasing their tidal volume.

Peripheral venous blood was arterialized using a goose neck lamp with a 100 watt bulb placed 2 to 3 cm above the sampling site. Warming continued until the temperature of a thermometer placed in a skin pocket reached 38-42^{o}C. Peripheral venous samples were obtained from indwelling catheters placed into a deep vein of each forepaw. Arterial samples were obtained from a femoral artery catheter. Samples of mixed venous blood were obtained from an indwelling flow-directed (Swan-Ganz) catheter placed in the pulmonary artery. Samples were collected within 5 minutes of each other using heparinized glass syringes.

Results indicated that peripheral venous blood arterialized and non-arterialized, correlated closely with arterial blood in terms of pH, pCO_2 and $[HCO_3^-]$ values. For the pH and pCO_2 values, the correlation coefficient (Pearson's r) was greater than 0.92 while for $[HCO_3^-]$ it was greater than 0.84. However, the pO_2 values for arterialized and non-arterialized peripheral venous blood did not correlate as strongly with that of arterial blood. With respiratory acidosis, the pO_2 correlation coefficient was 0.67 or greater. The pO_2 correlation coefficient varied from -0.53 to 0.61 during states of respiratory alkalosis.

Schriver attributed the absence of a significant difference between arterialized and non-arterialized blood to the placement of the catheters in a relatively large, deep vein. It was reasoned that

(1) flow is greater in the deeper veins relative to the superficial veins and that (2) flow in these deep veins is not significantly increased by arterialization (warming). Therefore, the blood-gas compositions of arterialized and non-arterialized peripheral venous blood were not significantly different.

The values obtained for the pH, pCO_2 and $[HCO_3^-]$ of peripheral venous blood, arterialized and non-arterialized, were also found to correlate closely with those of mixed venous blood. Correlation coefficients for pH, pCO_2 and $[HCO_3^-]$ were 0.98, 0.97 and 0.73 or greater, respectively. The pO_2 values correlated somewhat more closely with mixed venous than with arterial blood. The pO_2 correlation coefficient varied from -0.12 to 0.89 with respiratory acidosis and from 0.07 to 0.94 with respiratory alkalosis.

Schriver concluded that arterializing peripheral venous blood may not be necessary for estimating arterial values of pH, pCO_2 and $[HCO_3^-]$ if a deeper vein is used. Furthermore, it was concluded that peripheral venous blood-gas composition may provide reliable estimates of the blood-gas composition of mixed venous blood.

The full significance of Schriver's findings regarding mixed venous and non-arterialized peripheral venous blood is appreciated when an earlier study by Tung, Bettice, Wang & Brown (1976) is considered. These investigators compared the blood-gas composition of mixed venous and arterial blood in states of hemorrhagic shock.

Twenty-one mongrel dogs were anesthetized, intubated and nephrectomized. Catheters were placed in the left carotid and pulmonary artery for obtaining arterial and mixed venous samples respectively.

Hemorrhagic shock was induced by bleeding the dogs from a femoral artery catheter until the carotid arterial pressure was reduced to a level of 50 mmHg. Blood pressure was maintained at this level for 2 hours by reinfusing shed blood or withdrawing additional blood. Arterial and mixed venous blood samples were obtained 30, 60, 90 and 120 minutes after the steady state hypotension was established.

Comparison of arterial and mixed venous blood-gas compositions revealed that a-v differences in pH, pCO_2, pO_2 and $[HCO_3^-]$ were markedly increased when tissue perfusion was decreased. The acid-base picture presented by arterial blood-gas composition was one of partially compensated metabolic acidosis, i.e., values for pH, pCO_2 and $[HCO_3^-]$ less than normal. However, mixed venous blood-gas composition presented the picture of mixed respiratory and metabolic acidosis, i.e., pH and $[HCO_3^-]$ values less than normal, pCO_2 values greater than normal. Additionally, arterial pO_2 increased during hemorrhagic shock whereas mixed venous pO_2 decreased.

These investigators contrasted the acid-base status presented by arterial and mixed venous blood. They argued that arterial blood-gas composition reflects the adequacy of pulmonary ventilation and the respiratory response to conditions in peripheral tissues whereas mixed venous blood reflects the condition in peripheral tissues. Therefore, one can infer from these arguments that mixed venous blood-gas composition reflects the acid-base status of interstitial fluid (ISF).

Summary of research using peripheral venous blood. Research involving arterialized peripheral venous blood has been conducted sporadically since 1920. Most of the investigators have compared the

pH and/or pCO_2 values of arterialized peripheral venous blood to those of arterial blood when physiological conditions were varied (Brooks & Wynn, 1959; Collis & Neaverson, 1967; Harrison & Galloon, 1965; Paine, et al., 1961). Arteriovenous differences for these values were small and clinically insignificant. The general consensus was, therefore, that arterialized peripheral venous blood could be used in lieu of arterial blood to assess pCO_2 and pH.

Conflicting results have been obtained from studies investigating the use of arterialized peripheral venous blood in determining pO_2 and O_2 saturation. While some studies (Goldschmidt & Light, 1925; Meakins & Davies, 1920) showed the pO_2 saturations of arterialized peripheral venous blood and arterial blood to be comparable, one study (Collis & Neaverson, 1967) showed large discrepancies. Additionally, Collis and Neaverson concluded that arterialized peripheral venous blood could not be used for estimation of arterial pO_2.

Review of the literature reveals that "arterialization" of peripheral venous blood has been achieved in a variety of ways:

1) Warming the hand or extremity in a water bath at 45-47^{o}C for 5-20 minutes (Collis & Neaverson, 1967; Goldschmidt & Light, 1925; Meakins & Davies, 1920; Paine, et al., 1961).

2) Wrapping the hand or forearm in heating pads for 5-15 minutes (Brooks & Wynn, 1959; Harrison & Galloon, 1965).

3) Wrapping the hand in a hot towel for 15 to 20 minutes (Paine, et al., 1961).

Samples of arterialized peripheral venous blood were drawn primarily from the dorsal surface of the hand.

There is no reported human research comparing peripheral venous blood-gas composition to that of mixed venous blood. However, recent research on dogs by Schriver (1981) indicates that non-arterialized peripheral venous blood closely correlates with mixed venous blood in terms of pH, pCO_2 and $|HCO_3^-|$. Thus, peripheral venous blood may serve an additional role for accurately identifying the acid-base status of ISF.

Problem Statement

Studies have shown that arterialized peripheral venous blood is a reliable substitute for arterial blood in assessing acid-base status. However, little or no information was available in these studies regarding the cardiac output of the subjects. Changes in cardiac output will produce changes in peripheral venous blood-gas composition. Therefore, it may be asked, is arterialized peripheral venous blood still a reliable substitute for arterial blood when cardiac output is reduced?

Mixed venous blood may provide a more accurate assessment of acid-base status than arterial blood. A more convenient and less hazardous substitute for mixed venous sampling may prove to be non-arterialized peripheral venous blood. Schriver's study (1981) showed a close correlation between non-arterialized peripheral venous blood and mixed venous blood in respiratory acid-base disturbances. However, it is not known what happens to peripheral venous blood-gas composition when cardiac output is reduced.

In the present investigation I propose to answer the following questions:

1) How does the blood-gas composition of arterial and arterialized peripheral venous blood compare when cardiac output is acutely reduced?

2) How does the blood-gas composition of mixed venous and non-arterialized peripheral venous blood compare when cardiac output is acutely reduced?

Implications for Nursing Practice

Care of patients in the critical care setting, as in any other setting, is guided by the nursing process. Assessment, an essential component of this process, leads to nursing diagnosis, intervention and evaluation. Critical care nurses routinely make nursing diagnoses pertinent to their patient's cardiopulmonary function and acid-base status. These diagnoses require in part an accurate assessment and evaluation of each patient's arterial blood-gas composition. Critical care nurses then use these diagnoses to plan and implement nursing interventions.

Recently, mixed venous blood-gas composition has been investigated for use in assessment of both acid-base status and adequacy of tissue perfusion (Griffith, 1980, Tung et al., 1976). If further studies support these findings then mixed venous blood samples may be obtained more frequently in clinical settings. Critical care nurses will then be expected to be able to assess and evaluate both arterial and mixed venous blood-gas compositions.

Currently, critical care nurses routinely obtain arterial and, less frequently, mixed venous blood samples. Sampling arterial and mixed venous blood are high risk procedures. As noted previously,

arterial sampling is associated with multiple complications such as hemorrhage and neuropathies. Furthermore, the procedure is painful and, especially with repetition, can increase patient anxiety. Mixed venous sampling requires that a Swan-Ganz catheter be introduced into the pulmonary artery by a physician. Life-threatening complications associated with this painful procedure, such as ventricular arrhythmias and subclavian vein thrombosis, have been discussed. Furthermore, an increased incidence of catheter-related sepsis and endocarditis can be anticipated (Puri et al., 1980).

Clearly, equally informative, but less hazardous and compromising assessment alternatives are desirable. In this study I evaluated such an assessment alternative. If the blood-gas composition of peripheral venous blood could be shown to be useful for assessment of acid-base disturbances, then a simple venipuncture could provide this assessment alternative with less physical and emotional trauma to the patient.

It is important for critical care nurses to be conducting such research since they provide <u>total</u> patient care. Provision of total patient care includes the identification of factors which have the potential for compromising patient status. The sampling of arterial and mixed venous blood are two such factors. A study of the blood-gas composition of peripheral venous blood during reduced cardiac output and the subsequent acid-base disturbance may provide data that (a) will help alleviate an identified patient problem and (b) still provide a means for determining cardiopulmonary function and acid-base status.

CHAPTER II

METHODS

Procedure and Controls

Ten healthy mongrel dogs were used as subjects. The dogs were anesthetized with intravenous injections of sodium pentobarbital (30 mg/kg body weight). To maintain anesthesia sodium pentobarbital was given in 30 mg/kg doses every ½ to 1 hour as needed. The dogs were intubated and allowed to breathe room air.

An indwelling femoral arterial catheter was inserted and attached to a pressure transducer, which in turn, was attached to a polygraph (Grass, Model 7C). The pressure waves recorded by the polygraph allowed monitoring of the animals' blood pressure and heart rate. Arterial blood samples were obtained from this femoral catheter.

A Swan-Ganz flow-directed thermodilution catheter, size 7F, was passed via the right jugular vein into the pulmonary artery. This catheter was attached to a second pressure transducer which was also connected to the polygraph. Positioning of the catheter tip in the pulmonary artery was confirmed by the wave form recorded by the polygraph. Catheter position was routinely checked prior to mixed venous sampling and cardiac output measurements. A post mortem examination was conducted at the completion of each experiment to visually reconfirm catheter position. Patency of the Swan-Ganz catheter was maintained by the continuous slow infusion (25 ml/hr) of a 5% glucose solution containing 100 units of sodium panheparin per 100 ml of solution.

An indwelling venous catheter was placed in each forepaw of the animal. Catheters were inserted with the tip directed proximally. One forepaw was warmed thereby serving as the sampling site for obtaining arterialized peripheral venous blood. Accordingly, the other forepaw provided the non-arterialized peripheral venous blood. The skin temperature of the warmed forepaw was measured by a themometer placed in a skin pocket distal to the catheter insertion site.

The following procedure was used to arterialize peripheral venous blood: approximately 5 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 skin that was distal to the catheter hub. Warming was continued until the skin pocket temperature reached 38-42^{o}C. When this range was attained, a venous sample was drawn.

Samples of arterial, mixed venous and peripheral venous blood (arterialized and non-arterialized) were obtained within 5 to 8 minutes of each other. Each 1 ml sample was drawn anaerobically into heparinized glass syringes. The syringes were prepared in the following specialized manner: 1) The syringe barrel and plunger were lubricated with stopcock grease to minimize potential contamination from air or blood leakage. 2) The syringes were heparinized with sodium panheparin (1000 units/ml) to prevent the sample from clotting. 3) Approximately 0.08 cc of mercury was drawn up into the syringe to facilitate mixing of the sample. Prior to sampling, the mercury was advanced into the syringe hub thereby removing any excess heparin. Additionally, an initial volume of blood equal to the dead space of the catheter was withdrawn and discarded. After samples had been obtained

the needles were immediately flushed with blood, capped with a rubber stopper and placed in ice to reduce the oxygen consumption and metabolic rate of the blood cells. All catheters were then flushed with a solution of heparinized 0.9% saline.

At the beginning of each experiment the cardiac output of the animal was determined followed immediately by sampling. Thus, baseline values were established for later comparison with those obtained after cardiac output was reduced.

To measure cardiac output, the Swan-Ganz catheter was attached to a cardiac output computer (Edwards Laboratories, Model 9520A). This computer was in turn connected to a strip chart recorder (Edwards Laboratories, Model 9810) for recording thermodilution curves. The actual thermodilution procedure for measuring cardiac output was conducted as outlined in the instruction manual. In this study, 3 ml volumes of 5% glucose solution cooled to 0-2^{o}C in an ice bath, were used as the injectate when measuring cardiac output. Four to 6 successive measurements of the cardiac output were made each time. The first measurement was routinely discarded.

Successive decreases in cardiac output were achieved by reductions in the blood volume of each animal. The femoral artery catheter was unclamped and a volume of blood ranging from 75 to 250 ml per bleeding was removed. The exact volume removed per bleeding was determined by the animal's predicted blood volume based on weight. After a 45 minute stabilization period, the animal's cardiac output was again measured and samples were drawn. This entire sequence of events was repeated until 5 sets of samples were obtained.

Rectal temperature was monitored throughout each experiment to assure that body temperature was maintained within acceptable limits for blood-gas analysis. Hematocrit and protein concentrations were measured after each set of samples was obtained. Hematocrit was measured using heparinized capillary tubes. Protein concentration was measured using a protein refractometer (Hitachi).

Reliability of Measurements

An Edwards Laboratories Cardiac Output Computer (Model 9520A) was used to calculate cardiac output. This computer has an accuracy of $\pm$ 3% + 0.02 L/min and a repeatability better than $\pm$ 2%.

A Radiometer BGA3 Mark II blood-gas analyzer was used to measure the pH, pCO_2 and pO_2 of the blood samples. This analyzer, as stated in the BGA3 Instrument Manual, has a reproducibility of $\pm$ 0.001 pH units, $\pm$ 0.1 mmHg pCO_2 and $\pm$ 1.0 mmHg pO_2. Calibration of the pH, pCO_2 and pO_2 electrodes was checked prior to the analysis of each sample. The calibration procedure followed that described in the BGA3 manual.

Variables and Definition of Terms

The independent variable was the animal's cardiac output at the time of sampling. Successive reductions in cardiac output were produced by reducing the animal's blood volume.

The dependent variables were pH, pCO_2, pO_2 and $[HCO_3^-]$ concentrations of arterial, mixed venous and peripheral venous blood. Changes in each dependent variable were measured with each change in cardiac output.

Definitions:

1. pH: The pH of a solution is defined as the negative logarithm of the hydrogen ion activity in that solution (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 solution (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 solution (Slonim & Hamilton, 1976).

4. $[HCO_3^-]$: The bicarbonate concentration was calculated using the Henderson-Hasselbalch equation:

a) $$pH = pK'a + \log \frac{[HCO_3^-]}{S \cdot pCO_2}$$

therefore,

b) $$[HCO_3^-] = (10^{(pH - pK'a)}) (S \cdot pCO_2)$$

where $pK'a = 6.1$ at 37^oC, and

$S = 0.0301$ mM of CO_2/mmHg pCO_2

(Selkurt, 1976).

5. Cardiac Output: The cardiac output is the volume of blood pumped by each ventricle per minute, usually expressed as liters per minute (Smith & Kampine, 1980). Cardiac output was measured using an Edwards Laboratories thermodilution cardiac output computer.

6. Arterialized peripheral venous blood is produced when a venous sampling site is warmed to 35-45^oC leading to vasodilation and increased blood flow to the area. This increased blood flow causes increased O_2 delivery, providing venous blood with a composition resembling that of arterial blood (Collis & Neaverson, 1967).

CHAPTER III

RESULTS

General Description

The blood-gas compositions of peripheral venous blood [arterialized (APV) and non-arterialized (NAPV)], mixed venous (MV) and arterial (A) blood were determined when cardiac output was reduced. Ten healthy mongrel dogs whose weights ranged from 12.7 to 22.7 kilograms were subjects for these experiments. Control cardiac output of these animals ranged from 1.53 to 3.73 L/min. Subsequent hemorrhage reduced cardiac output to low values which ranged from 0.63 to 1.36 L/min (25 to 51% of control cardiac output).

Table 1 shows the ranges of values for pH, pCO_2, pO_2 and $[HCO_3^-]$ measured at the onset and just prior to termination of the experiments. The bicarbonate concentrations were calculated using the Henderson-Hasselbalch equation (pK = 6.1).

Table 2 contains a complete summary of all blood-gas values and the corresponding cardiac output. Data from Dog 1 were not included in this study because mixed venous pO_2 values were inappropriately high indicating that oxygenated blood had been withdrawn from the pulmonary capillary beds. For example, simultaneously drawn arterial and mixed venous blood samples had pO_2 values of 92 and 102 mmHg respectively. Therefore, pulmonary artery blood samples did not reflect true mixed venous blood-gas composition. Additionally, APV blood samples were not obtained in Dog 2. Any other omissions indicate the blood sample could not be obtained at that particular cardiac output.

The hematocrit and serum protein concentration were measured with each set of samples. Initial protein concentration and hematocrit ranged from 5.1 to 6.8 gm% and 28 to 51% respectively. Protein concentration and hematocrit at the end of these experiments ranged from 4.1 to 5.2 gm% and 36 to 50% respectively. It should be noted that hematocrit values tended to increase then decrease. This was an anticipated finding since splenic contraction is a known canine compensatory mechanism to hemorrhagic shock.

Heart rate, respiratory rate and blood temperature in the pulmonary artery were monitored throughout the experiments. A summary of these measurements during control and low (25-51% of control) cardiac output is shown in Table 3. Some of the variability in animal heart and respiratory rate was attributed to the anesthetic. Blood temperature was measured by the Swan-Ganz thermister electrode in the pulmonary artery and displayed on the cardiac output computer. In addition, warmed paw temperature was monitored during the heating period. Arterialized peripheral venous blood samples were drawn when paw temperature reached $40^{o}C$.

Specific Parameters

Description of the results is organized under the following headings:

1. Comparison of peripheral venous blood-gas composition (APV and NAPV) to that of arterial blood.
2. Comparison of peripheral venous blood-gas composition (APV and NAPV) to that of mixed venous blood.

Each blood-gas parameter i.e., pH, pCO_2, pO_2 and $[HCO_3^-]$ will be separately addressed under each heading.

1. Comparison of peripheral venous blood-gas composition (APV and NAPV) to that of arterial blood.

pH The relationship of the pH of APV and NAPV blood as a function of arterial pH is shown in Figure 2a. The further a given point is from the identity line, the greater the arteriovenous difference (A-V difference) for that blood-gas parameter. A summary of mean A-V differences for specified ranges of cardiac output are displayed in Table 4. Figure 3a shows mean A-V differences plotted as bar graphs for specified cardiac output intervals. It is apparent from inspection of this figure in relation to Figure 2a that the widest scattering of points noted on the identity plot occurred at cardiac output values less than 50% of control. When cardiac output was greater than 50% of control, mean A-V differences remained relatively stable.

pCO_2 Comparison of the pCO_2 of peripheral venous blood (arterialized and non-arterialized) to the pCO_2 of arterial blood is shown in Figure 4a. Mean A-V differences for pCO_2 over different ranges of cardiac output are shown in Figure 5a. Comparison of this bar graph with Figure 3a shows that mean A-V differences for pCO_2 as with pH, increased markedly at cardiac output values less than 50% of control. Refer to Table 4 for actual values. It should be noted that pCO_2 values for APV and NAPV blood increased whereas the pCO_2 values for arterial blood decreased when cardiac output was reduced (Table 2).

$[HCO_3^-]$ The $[HCO_3^-]$ in APV and NAPV blood did not appear to correlate closely with that of arterial blood. The identity relationship is shown in Figure 6a. With 3 exceptions, all points lie above the identity line. Mean A-V differences for $[HCO_3^-]$ are graphed as a function of specific intervals of cardiac output in Figure 7a. The magnitude of mean A-V differences for NAPV blood increased in a step-like fashion while those for APV blood remained more constant until cardiac output was less than 50% of control. See Table 4 for actual values.

pO_2 It is apparent from visual inspection of Figure 8a that the pO_2 of peripheral venous blood (arterialized and non-arterialized) correlated poorly with that of arterial blood. Inspection of Figure 9A shows that mean A-V differences increased as cardiac output was reduced. Mean A-V differences for pO_2 appear in Table 4. It is of special interest to note that while the pO_2 of APV and NAPV blood decreased when cardiac output was reduced, the pO_2 of arterial blood increased.

2. Comparison of peripheral venous blood-gas composition (APV and NAPV) to that of mixed venous blood.

pH A comparison of the pH of APV and NAPV blood to that of mixed venous blood is shown in Figure 2b. Points appear equally distributed on both sides of the identity line. However, points below the identity line were more widely scattered at pH values less than 7.28. Figure 3b shows mean MV-PV (mixed venous-peripheral venous) differences for pH grouped for different ranges of cardiac output. The mean MV-PV pH differences remained more constant than mean A-V pH differences when

cardiac output was greater than 50% of control values (cf., Figure 3a). Furthermore, it is apparent from visual inspection of Figures 3a and 3b that mean MV-PV differences for pH were less than mean A-V pH differences. Actual MV-PV differences are reported in Table 5. Both APV and NAPV blood had similar mean MV-PV differences for pH.

pCO_2 Figure 4b is an identity plot of the pCO_2 of APV and NAPV blood as a function of the pCO_2 of mixed venous blood. Mean MV-PV differences for specified ranges of cardiac output are shown in Figure 5b. This bar graph shows that mean MV-PV differences for APV blood remained more constant than those for NAPV blood until cardiac output decreased below 50% of control values. It is apparent from inspection of Figures 5a and 5b, and Tables 4 and 5 that differences in pCO_2 between MV and PV blood were of less magnitude than mean A-V differences.

$[HCO_3^-]$ It can be seen from inspection of Figures 6a and 6b that the $[HCO_3^-]$ of peripheral venous blood (arterialized and non-arterialized) correlated more closely with mixed venous rather than arterial blood. Figure 7b shows mean MV-PV differences plotted as bar graphs for specified cardiac output intervals. Inspection of this bar graph shows that the magnitude of mean MV-PV differences remained relatively constant for any given cardiac output interval. This is in contrast to the results shown in Figure 7a. Actual values are recorded in Table 5.

pO_2 Figure 8b shows the identity relationship between the pO_2 of peripheral venous blood samples as a function of the pO_2 of mixed venous blood. Approximately 1/3 of the points lie below the identity line (MV pO_2 > PV pO_2). The majority of points have a

scattered distribution above and away from the identity line (PV pO_2 > MV pO_2). The bar graph shown in Figure 9 represents mean MV-PV differences for pO_2 in APV and NAPV blood. The magnitude of mean MV-PV differences for pO_2 in NAPV blood varied less than that of APV blood. This result is in marked contrast to the change in mean A-V pO_2 differences shown in Figure 9a. Refer to Table 5 for actual values.

Table 1

Ranges of Values of Blood-Gas Parameters

Parameter	Source	Cardiac Output	
		100% Control	Lowest % Control
pH	A	7.242-7.370	7.263-7.421
	APV	7.254-7.405	7.086-7.383
	NAPV	7.251-7.391	7.021-7.340
	MV	7.244-7.381	7.184-7.308
pCO_2 mmHg	A	38.9-59.0	25.4-40.6
	APV	39.3-59.7	32.5-83.6
	NAPV	39.5-62.7	37.2-96.6
	MV	43.8-65.4	42.0-60.6
$[HCO_3^-]$ mEq/L	A	21.7-27.3	14.1-19.5
	APV	22.3-28.0	18.8-24.7
	NAPV	22.9-27.9	18.5-27.2
	MV	23.3-28.9	19.0-24.4
pO_2 mmHg	A	51.5-87.2	73.3-103.6
	APV	54.7-84.4	19.2-73.2
	NAPV	53.1-81.6	18.6-68.6
	MV	41.7-61.8	19.4-41.4

Key: A = Arterial Blood
APV = Arterialized Peripheral Venous Blood
NAPV = Non-arterialized Peripheral Venous Blood
MV = Mixed Venous Blood

Table 2

Summary of Data for Dogs 2-10

	Cardiac Output		pH				pCO_2 mmHg				$[HCO_3^-]$ mEq/L				pO_2 mmHg			
	$\bar{x}$	% Control	A	APV	NAPV	MV	A	APV	NAPV	MV	A	APV	NAPV	MV	A	APV	NAPV	MV
Dog 2	1.53	100	7.352		7.347	7.323	46.2		47.5	50.8	24.8		25.3	25.6	83.0		58.6	41.7
	1.20	78.4	7.312		7.300	7.282	47.6		48.2	54.2	23.3		23.0	24.8	85.3		59.8	40.4
	1.10	71.9	7.301		7.265	7.250	43.0		51.6	54.9	20.6		22.7	23.3	91.4		52.2	37.6
	0.63	41.2	7.263		7.145	7.184	40.6		62.6	59.5	17.8		20.9	21.7	98.3		38.1	29.1
Dog 3	1.89	100	7.332	7.307	7.333	7.296	42.3	48.7	44.4	49.2	21.7	23.6	22.9	23.3	87.2	61.0	70.4	53.7
	1.42	75.1	7.356	7.276	7.324	7.288	37.6	46.2	44.6	51.4	20.4	20.9	22.5	23.9	93.3	67.1	69.8	49.5
	1.00	52.9	7.312	7.303	7.325	7.271	40.2	43.4	41.2	49.9	19.7	20.9	20.8	22.3	91.3	68.4	66.5	45.7
	0.97	51.3	7.334	7.325	7.310	7.285	35.1	38.4	40.8	44.6	18.1	19.4	19.9	20.6	92.1	59.0	54.9	41.4
Dog 4	3.25	100	7.370	7.376	7.391	7.359	38.9	39.3	39.5	43.8	21.8	22.3	23.2	23.9	73.2	71.6	62.4	47.3
	1.84	56.6	7.264	7.259	7.282	7.252	52.8	54.2	51.8	55.5	23.2	23.5	23.7	23.7	56.4	49.9	50.9	34.1
	1.25	38.5	7.421	7.383	7.340	7.308	29.4	32.5	37.2	47.2	18.5	18.8	19.5	22.9	85.6	73.2	62.0	24.8
Dog 5	1.86	100	7.242	7.254	7.251	7.244	57.7	58.0	58.8	62.7	24.1	24.9	25.1	26.3	86.4	84.4	81.6	54.3
	1.74	93.6	7.355	7.268	7.273	7.305	39.6	54.0	52.4	51.1	21.4	23.9	23.5	24.7	115.2	78.5	91.1	48.9
	0.97	52.2	7.324	7.275	7.311	7.264	41.3	50.0	43.6	55.6	20.8	22.5	21.3	24.4	100.1	56.4	85.5	43.2
	0.89	47.9	7.238	7.160	7.186	7.166	46.1	62.2	54.3	65.9	19.1	21.5	19.9	23.1	91.0	46.2	74.4	37.8
	0.87	46.8	7.279	7.193	7.216	7.196	37	52.2	47.1	60.6	16.8	19.5	18.5	22.8	95.5	52.3	68.6	27.3
Dog 6	3.10	100	7.357	7.405	7.371	7.381	43.5	39.9	43.9	43.8	23.7	24.2	24.7	25.2	51.5	58.0	53.1	44.9
	2.26	72.9	7.375	7.367	7.347	7.327	41.0	40.2	44.2	48.3	23.3	22.4	23.5	24.5	58.1	54.8	57.3	41.8
	1.75	56.5	7.383	7.343	7.331	7.344	37.7	40.4	44.1	44.4	21.8	21.3	22.6	23.4	68.4	56.6	49.9	36.8
	0.76	24.5	7.303		7.146	7.236	30.9		61.6	48.7	14.8		20.6	20.1	78.5		18.6	19.4
Dog 7	3.12	100	7.269	7.272	7.270	7.260	50.1	51.1	51.4	53.8	22.3	22.9	22.9	23.4	64.2	61.8	63.2	48.8
	2.47	79.2	7.285	7.285	7.296	7.289	43.4	46.0	44.3	45.8	20.0	21.2	20.9	21.3	71.9	64.8	61.6	47.7
	1.86	59.6	7.347	7.323	7.274	7.317	34.5	38.8	51.0	42.2	18.3	19.5	22.9	20.9	91.8	64.3	30.4	44.5
	0.99	31.7	7.321	7.225	7.182	7.273	34.5	49.3	59.5	45.2	17.3	19.8	21.6	20.3	94.6	36.1	33.3	38.7
	0.87	27.9	7.365	7.175		7.276	25.4	57.2		42.0	14.1	20.5		19.0	97.5	25.7		30.1
Dog 8	2.00	100	7.272	7.274	7.251	7.251	59.0	59.7	62.7	65.4	26.4	26.8	26.7	27.9	74.7	63.9	63.5	49.8
	1.45	72.5	7.315	7.247	7.239	7.265	50.3	64.0	66.3	62.9	24.8	27.0	27.5	27.7	98.8	41.6	45.4	45.0
	1.22	61.0	7.252	7.195	7.187	7.249	55.5	73.4	76.9	62.8	23.7	27.5	28.3	26.6	85.4	38.5	36.4	38.2
	0.89	44.5	7.290	7.118	7.094	7.231	46.4	82.7	90.5	61.6	21.6	26.0	26.9	25.1	98.6	23.0	25.2	32.8
	0.73	36.5	7.323	7.086	7.021	7.216	37.3	83.6	96.6	57.4	18.8	24.4	24.2	22.6	103.6	19.2	21.3	24.6
Dog 9	2.95	100	7.367	7.334	7.371	7.341	49.0	54.3	49.6	55.2	27.3	28.0	27.9	28.9	83.0	54.7	60.9	50.5
	2.63	89.2	7.287	7.252	7.268	7.253	57.4	68.1	63.6	66.9	26.6	29.1	28.2	28.6	78.3	44.5	54.3	45.7
	2.29	77.6	7.274	7.244	7.235	7.263	55.3	70.5	70.3	68.7	24.9	29.6	28.9	30.1	74.1	40.1	39.1	42.8
	1.63	55.3	7.316	7.245	7.220	7.254	45.3	67.8	68.6	63.4	22.4	28.5	27.2	27.2	86.5	38.0	35.7	38.4
	1.36	46.1	7.382	7.182		7.265	33.9	67.9		55.4	19.5	24.7		24.4	94.5	36.2		32.2
Dog 10	3.73	100	7.345	7.335	7.341	7.334	43.3	46.7	43.7	46.8	22.9	24.2	22.9	24.1	82.5	62.2	69.3	61.8
	2.54	68.1	7.358	7.326	7.331	7.320	40.8	48.3	44.9	48.7	22.2	24.5	23.0	24.3	86.1	48.7	61.7	50.8
	1.89	50.7	7.371	7.322	7.308	7.324	38.8	47.2	50.1	48.6	21.8	23.7	24.3	24.5	89.7	51.6	50.8	47.5
	1.57	42.1	7.367	7.299	7.318	7.306	35.7	48.0	46.0	48.3	19.9	22.9	22.9	23.4	78.6	41.6	49.7	35.5
	1.22	32.7	7.311	7.267	7.237	7.247	39.8	49.5	54.3	53.7	19.5	21.9	22.4	22.7	73.3	42.1	37.9	26.5

Key: A = Arterial Blood
APV = Arterialized Peripheral Venous Blood
NAPV = Non-arterialized Peripheral Venous Blood
MV = Mixed Venous

Table 3

Ranges of Values for Heart Rate, Respiratory Rate and P. A. Blood Temperature

Cardiac Output	100% Control	Lowest % Control
Heart Rate		
Range	96-192	132-184
$\bar{x}$, SD	147±28.1	160±18.7
Respiratory Rate		
Range	4-20	10-40
$\bar{x}$, SD	10.2±5.4	27.4±11.4
P. A. Blood Temperature		
Range ^{o}C	35.1-38.4	35.4-39.7
$\bar{x}$, SD	36.8±1.3	37.4±1.5

Key: P. A. = Pulmonary Artery

Table 4

Summary of Mean A-V Differences

Cardiac Output Intervals % Control		pH		pCO_2		$[HCO_3^-]$		pO_2	
		APV	NAPV	APV	NAPV	APV	NAPV	APV	NAPV
80-100	$\bar{x}$	0.012	0.007	-3.900	-2.773	-1.182	-0.923	15.560	13.709
	SD	0.036	0.028	5.427	3.761	0.817	0.610	15.416	9.478
65-80	$\bar{x}$	0.036	0.030	-7.800	-6.925	-1.648	-1.561	27.533	26.513
	SD	0.032	0.025	6.186	5.959	1.895	1.428	20.129	16.451
50-65	$\bar{x}$	0.035	0.039	-8.044	-9.656	-1.880	-2.362	31.000	33.411
	SD	0.024	0.040	7.411	8.962	1.958	1.831	15.091	18.717
<50	$\bar{x}$	0.121	0.123	-21.960	-23.200	-3.470	-3.338	51.720	46.830
	SD	0.072	0.080	14.026	17.246	1.859	1.831	22.199	23.143

Key: APV = Arterialized Peripheral Venous Blood
NAPV = Non-arterialized Peripheral Venous Blood
A-V = Arteriovenous Differences

Table 5

Summary of Mean MV-PV Differences

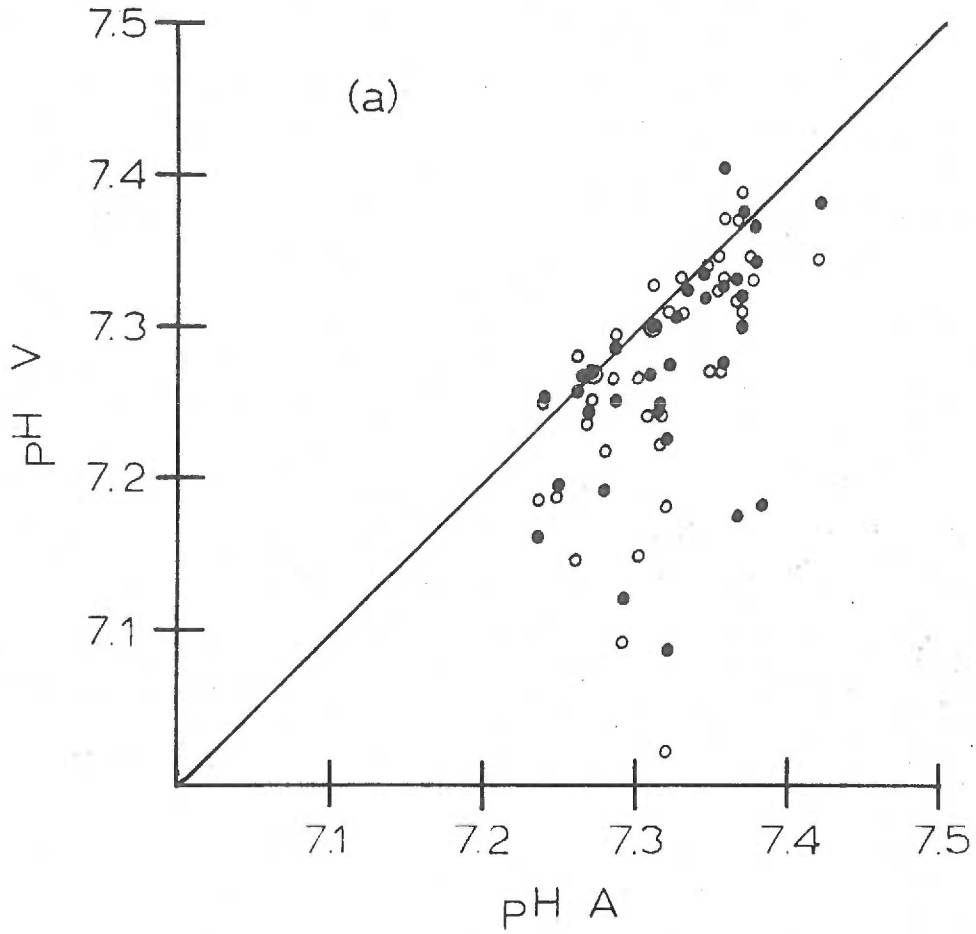
Cardiac Output Intervals % Control		pH		pCO_2		$\lvert HCO_3^- \rvert$		pO_2	
		APV	NAPV	APV	NAPV	APV	NAPV	APV	NAPV
80-100	$\bar{x}$	-0.005	-0.011	1.890	2.909	0.640	0.968	-13.490	-16.455
	SD	0.018	0.020	2.839	2.029	0.705	0.786	11.376	10.203
65-80	$\bar{x}$	0.001	-0.007	1.767	2.563	1.050	0.994	-6.583	-11.413
	SD	0.022	0.022	3.964	3.550	1.238	0.555	10.338	8.667
50-65	$\bar{x}$	-0.003	0.001	1.489	-0.122	0.763	0.281	-12.544	-10.133
	SD	0.027	0.042	5.646	8.317	1.197	1.526	8.901	16.409
<50	$\bar{x}$	0.040	0.048	-4.780	-6.160	0.634	0.707	-8.530	-13.260
	SD	0.066	0.077	13.439	17.624	1.948	2.218	17.507	18.803

Key: APV = Arterialized Peripheral Venous Blood

NAPV = Non-arterialized Peripheral Venous Blood

MV-PV = Mixed Venous-Peripheral Venous Differences

Figure 2. Identity relationship is shown for the pH of peripheral venous (V) blood [arterialized (APV) and non-arterialized (NAPV)] as a function of (a) arterial (A) pH (top figure) and (b) mixed venous (MV) pH (bottom figure). Each point represents paired samples. Open circles denote NAPV blood samples whereas closed circles denote APV blood samples.


(a)
7.5
7.4
7.3
7.2
7.1
pH V
7.1
7.2
7.3
7.4
7.5
pH A

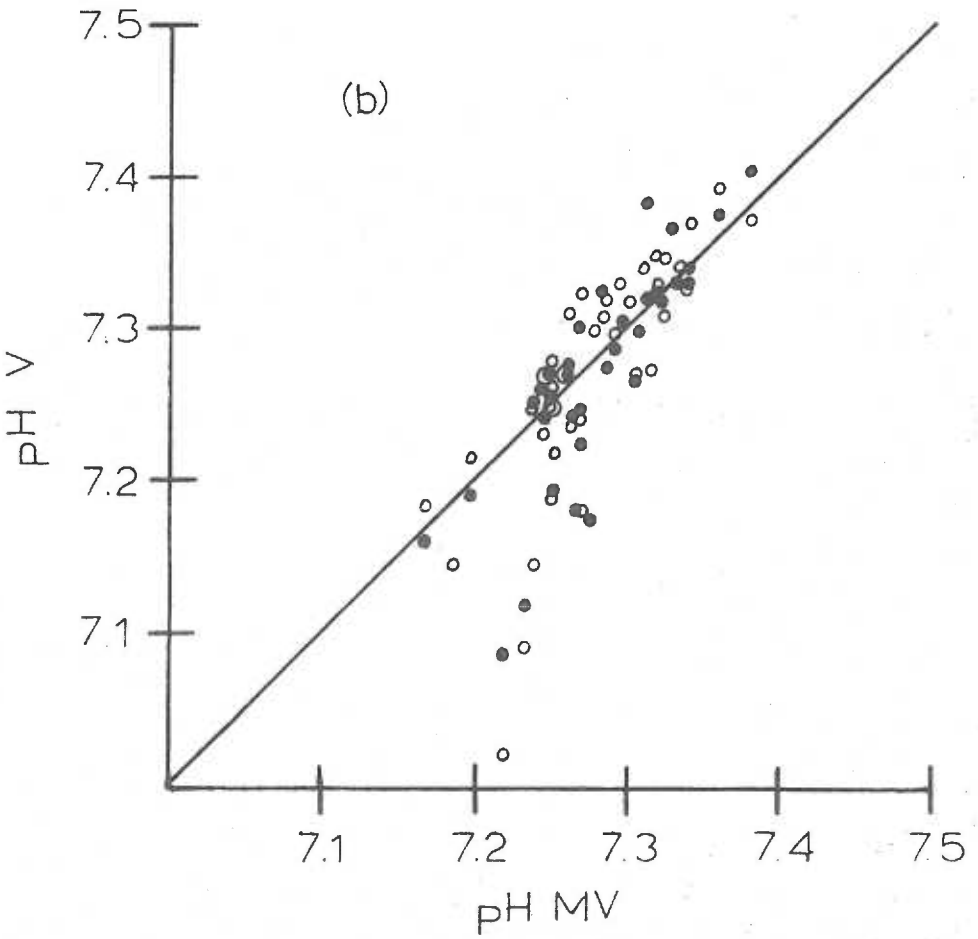
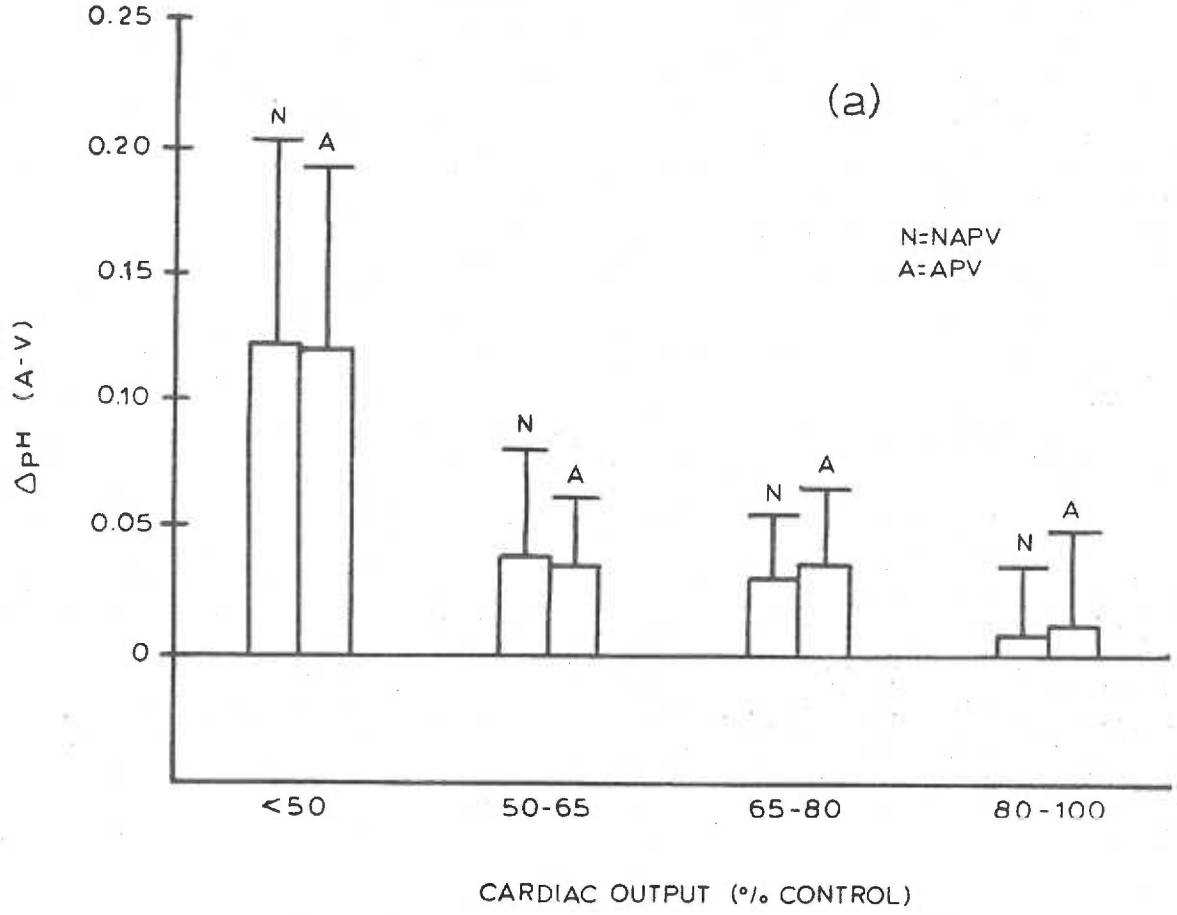

(b)
7.5
7.4
7.3
7.2
7.1
pH V
7.1
7.2
7.3
7.4
7.5
pH MV

Figure 3. (a) Mean arteriovenous (A-V) differences for pH and (b) mean mixed venous-peripheral venous (MV-PV) differences for pH as a function of ranges of percent control cardiac output. Venous samples included arterialized (APV) and non-arterialized (NAPV) peripheral venous blood.


(a)
N=NAPV
A=APV
0.25
0.20
0.15
0.10
0.05
0
ΔpH (A-V)
N
A
<50
50-65
65-80
80-100
CARDIAC OUTPUT (% CONTROL)

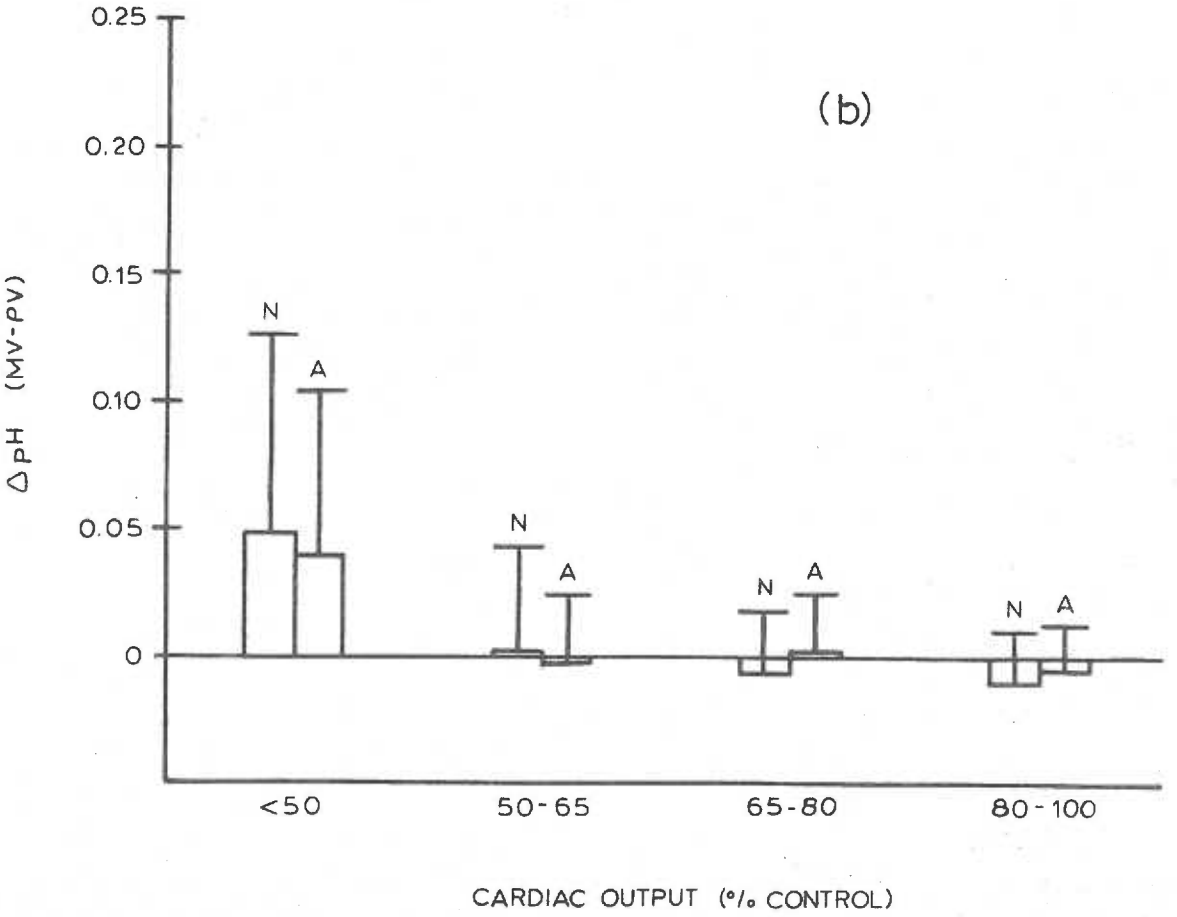
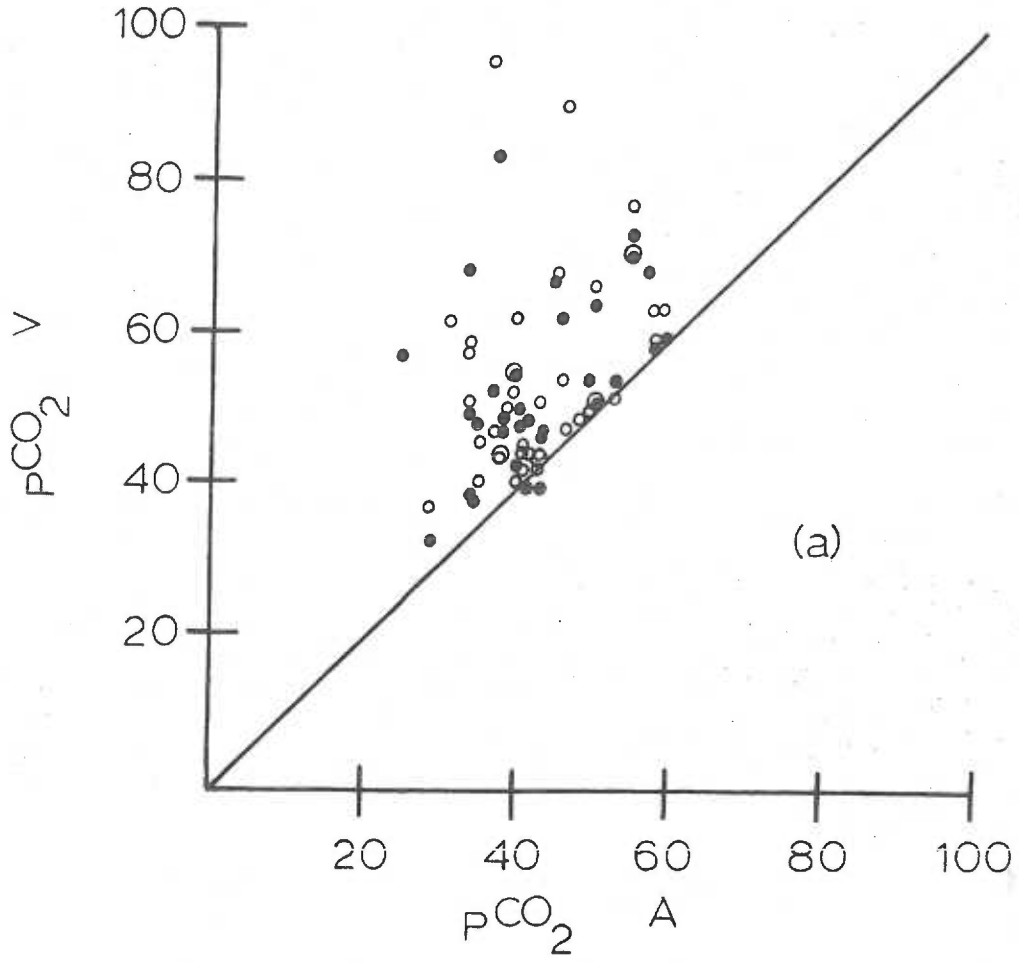

(b)
0.25
0.20
0.15
0.10
0.05
0
ΔpH (MV-PV)
N
A
<50
50-65
65-80
80-100
CARDIAC OUTPUT (% CONTROL)

Figure 4. Identity relationship is shown for the pCO_2 of peripheral venous (V) blood [arterialized (APV) and non-arterialized (NAPV)] as a function of (a) arterial (A) pCO_2 (top figure) and (b) mixed venous (MV) pCO_2 (bottom figure). Each point represents paired samples. Open circles denote NAPV blood samples whereas closed circles denote APV blood samples. Units are in mmHg.


100
80
60
40
20
P^{CO_2} V
(a)
20
40
60
80
100
P^{CO_2} A

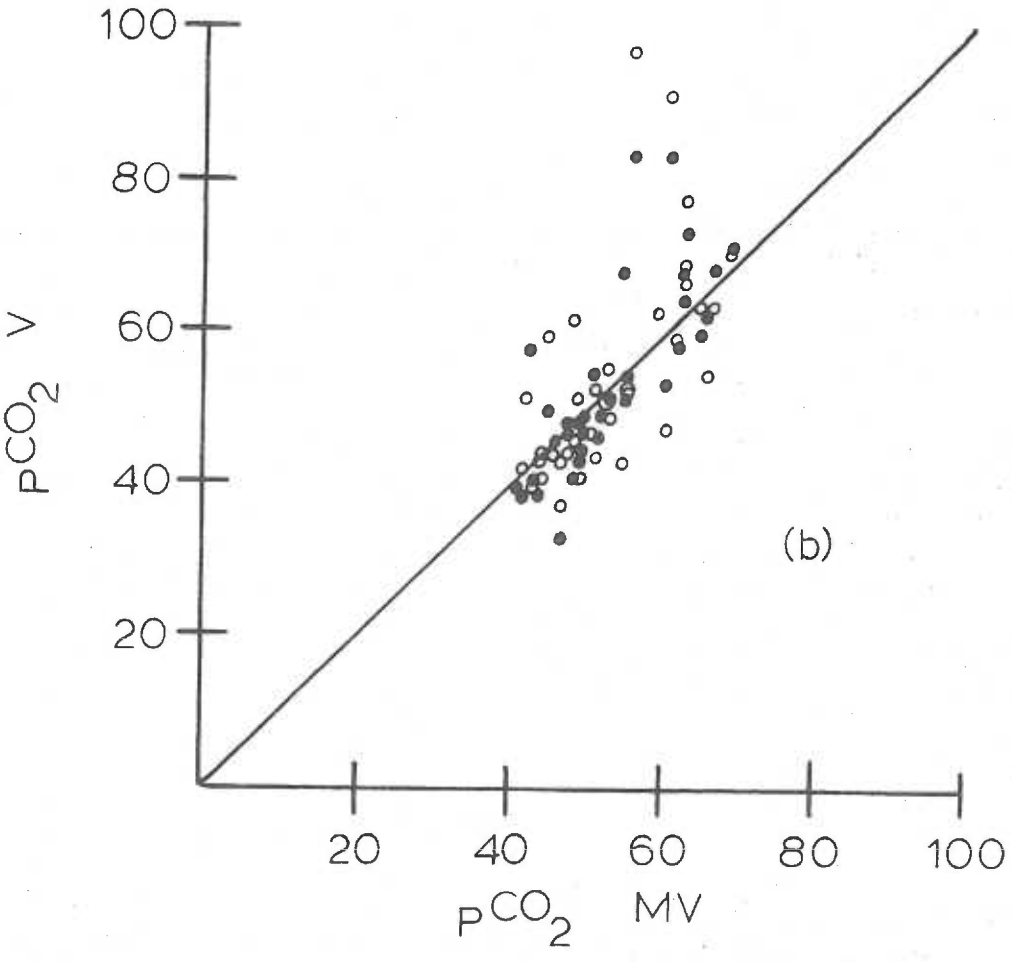
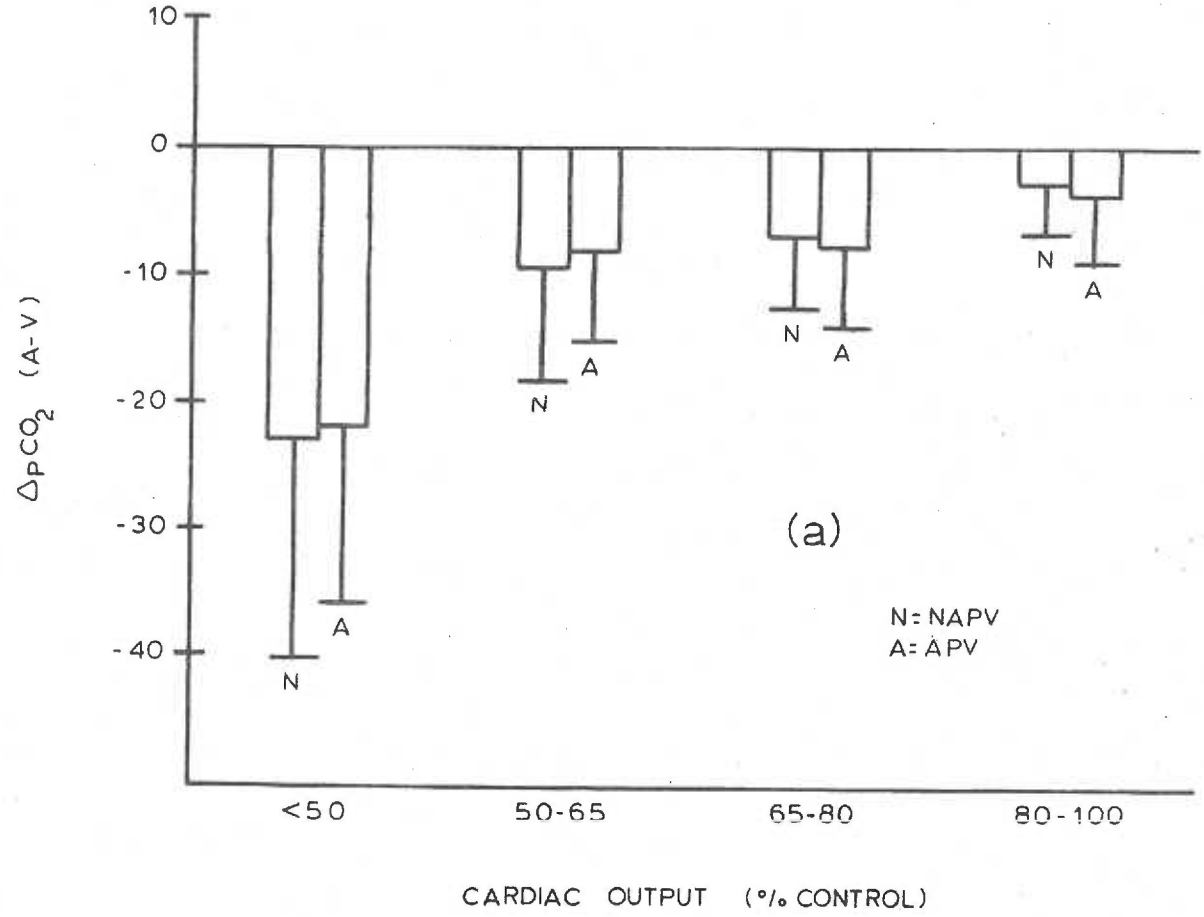

100
80
60
40
20
P^{CO_2} V
(b)
20
40
60
80
100
P^{CO_2} MV

Figure 5. (a) Mean arteriovenous (A-V) differences for pCO_2 and (b) mean mixed venous-peripheral venous (MV-PV) differences for pCO_2 as a function of ranges of percent control cardiac output. Venous samples included arterialized (APV) and non-arterialized (NAPV) peripheral venous blood. Units are in mmHg.


10
0
-10
-20
-30
-40
ΔpCO_2 (A-V)
N
A
(a)
N: NAPV
A: APV
<50
50-65
65-80
80-100
CARDIAC OUTPUT (% CONTROL)

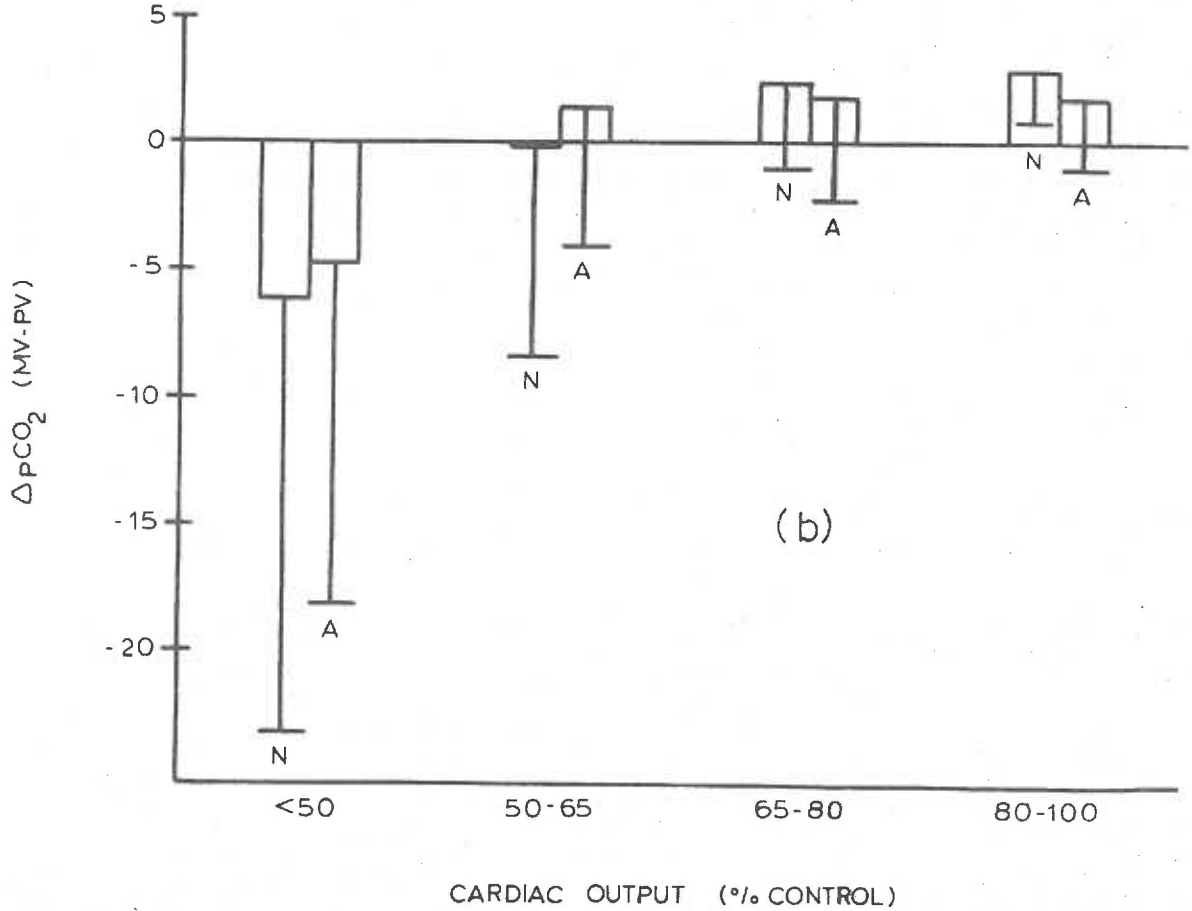
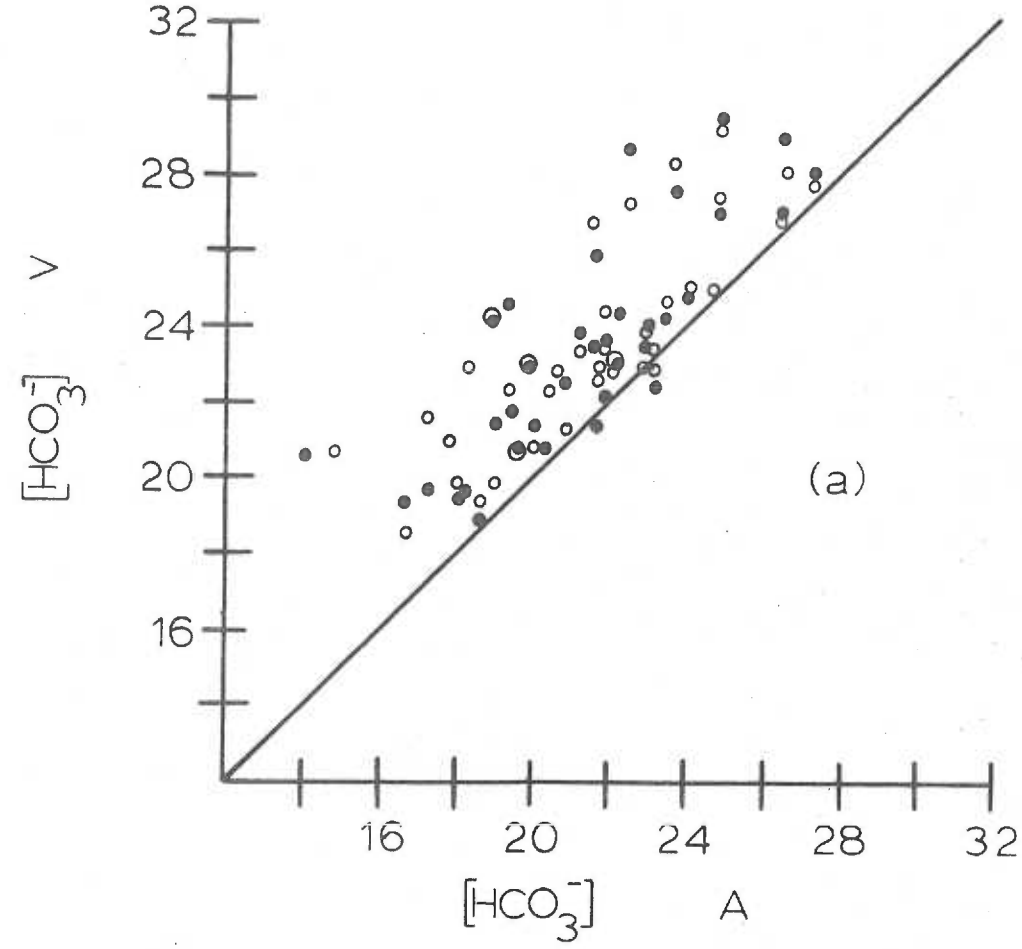

5
0
-5
-10
-15
-20
ΔpCO_2 (MV-PV)
N
A
(b)
<50
50-65
65-80
80-100
CARDIAC OUTPUT (% CONTROL)

Figure 6. Identity relationship is shown for the $[HCO_3^-]$ of peripheral venous (V) blood [arterialized (APV) and non-arterialized (NAPV)] as a function of (a) arterial (A) $[HCO_3^-]$ (top figure) and (b) mixed venous (MV) $[HCO_3^-]$ (bottom figure). Each point represents paired samples. Open circles denote NAPV blood samples whereas closed circles denote APV blood samples. Units are in Meq/L.


32
28
24
20
16
V
[HCO3-]
(a)
16
20
24
28
32
[HCO3-]
A

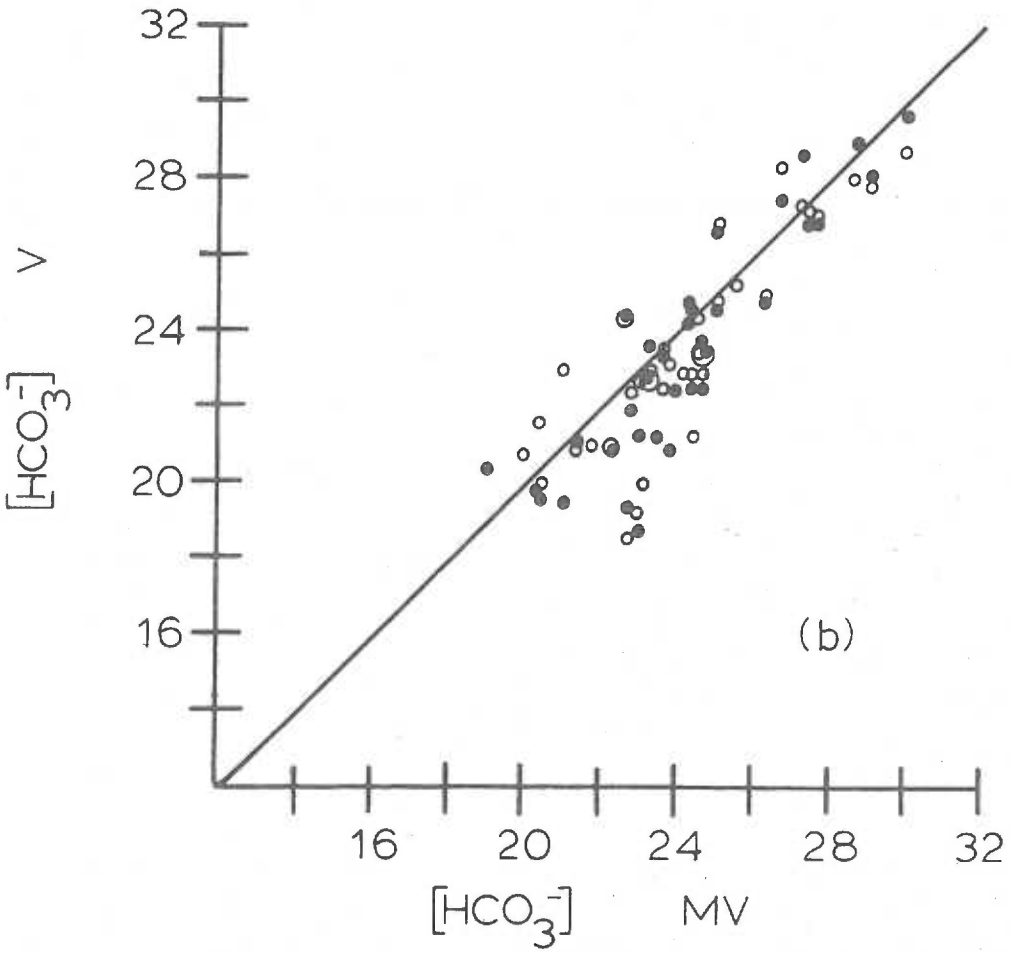
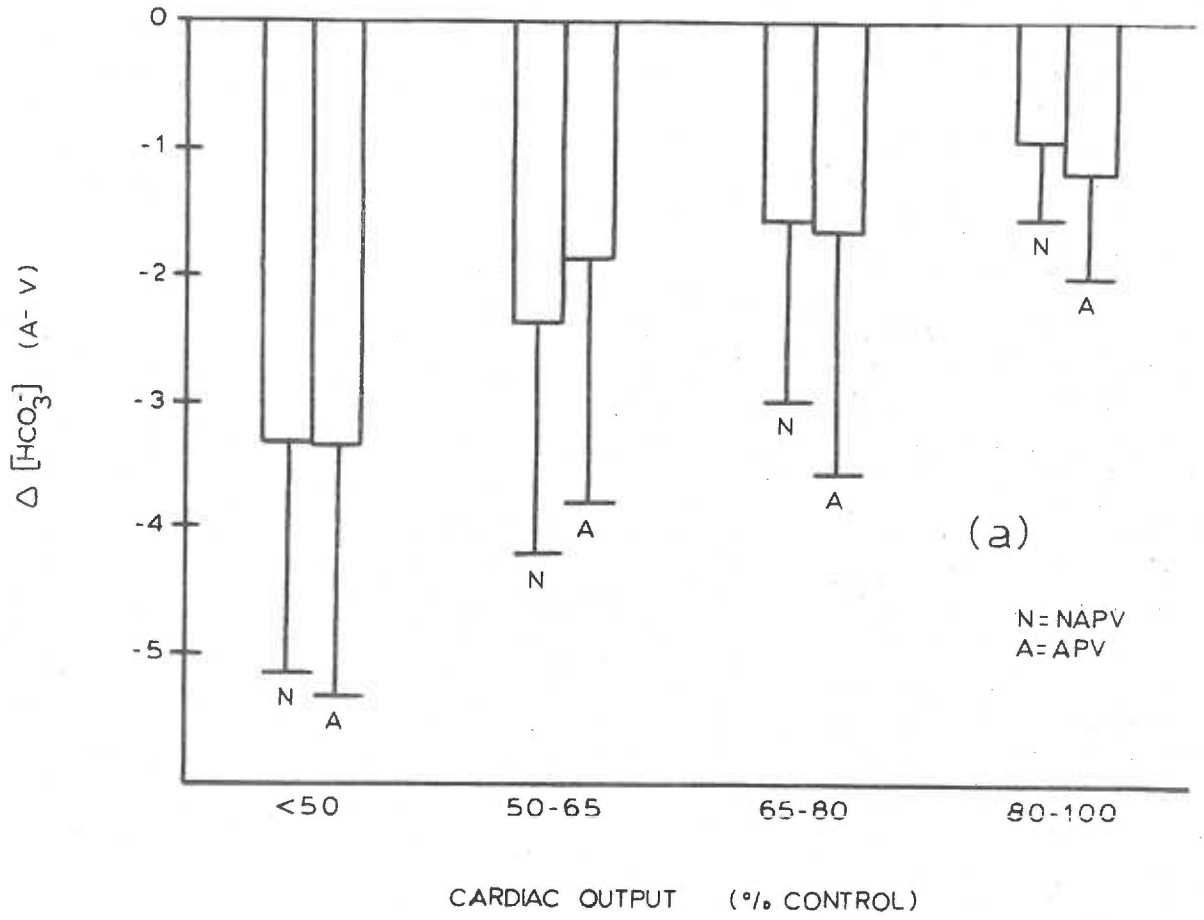

32
28
24
20
16
V
[HCO3-]
(b)
16
20
24
28
32
[HCO3-]
MV

Figure 7. (a) Mean arteriovenous (A-V) differences for $[HCO_3^-]$ and (b) mean mixed venous-peripheral venous (MV-PV) differences for $[HCO_3^-]$ as a function of ranges of percent control cardiac output. Venous samples included arterialized (APV) and non-arterialized (NAPV) peripheral venous blood. Units are in Meq/L.


0
-1
-2
-3
-4
-5
Δ [HCO3-] (A-V)
N
A
(a)
N=NAPV
A=APV
<50
50-65
65-80
80-100
CARDIAC OUTPUT (% CONTROL)

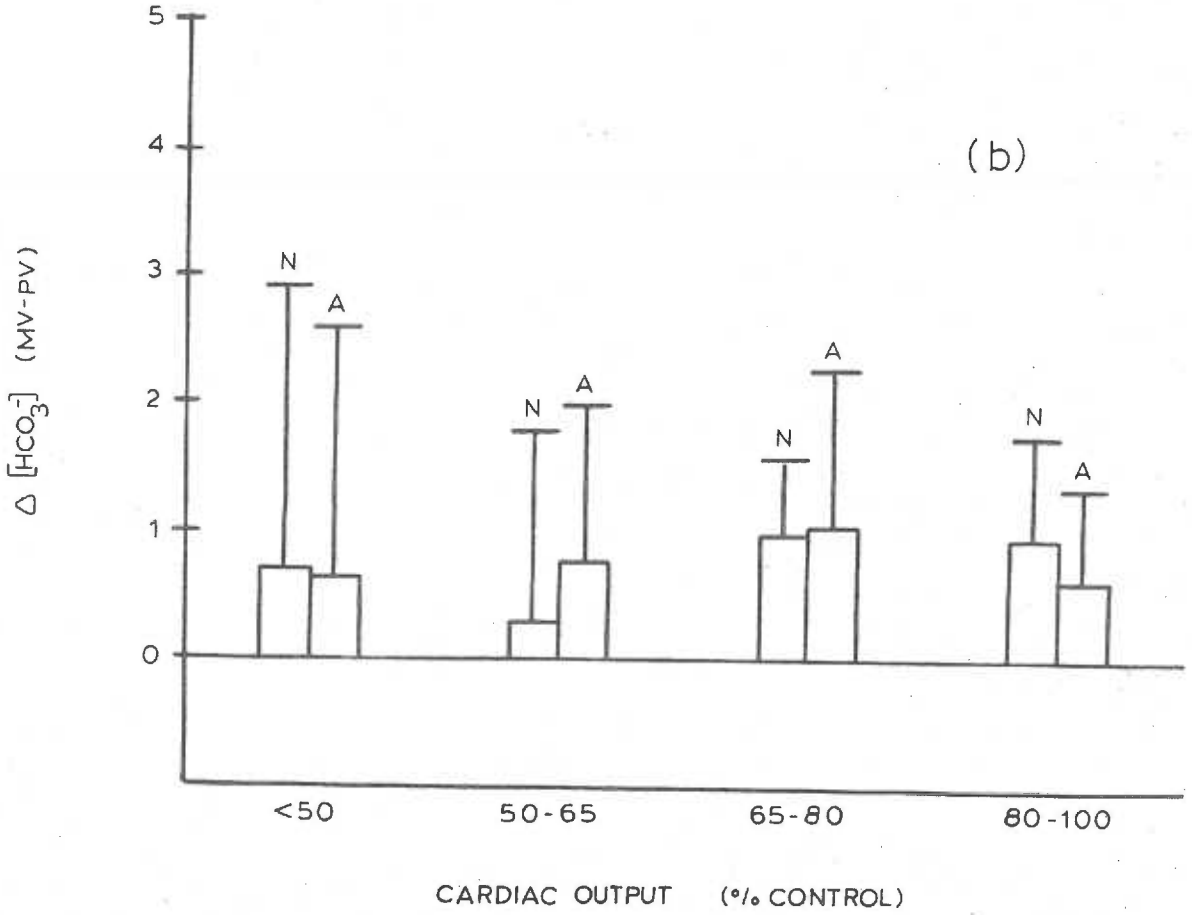
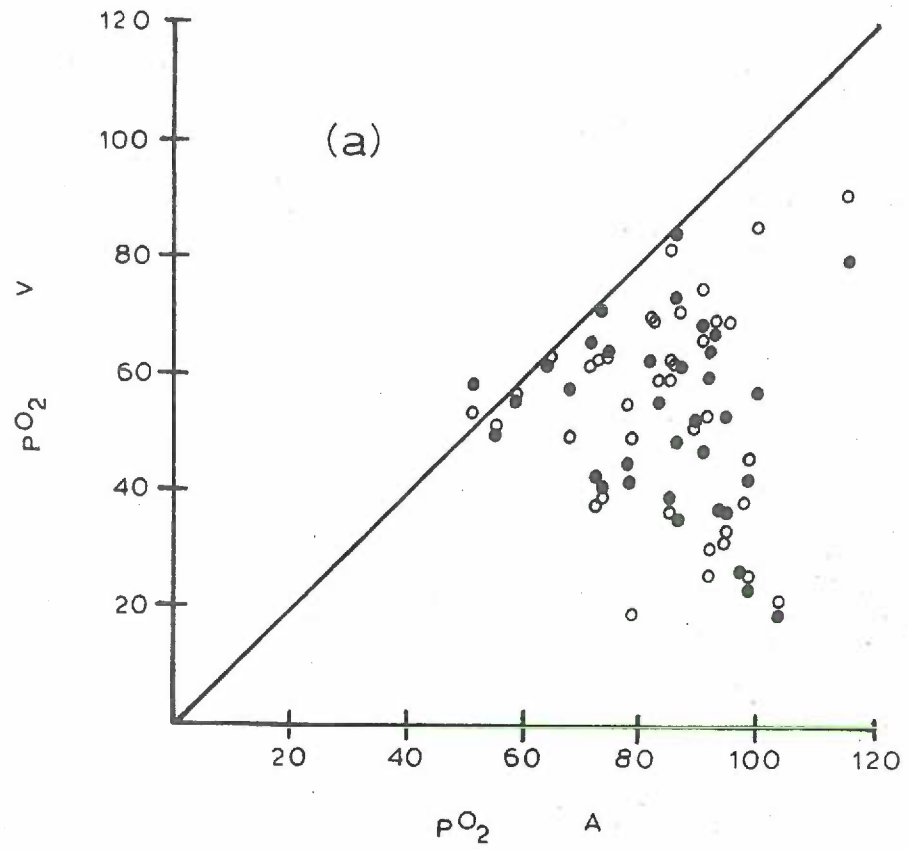
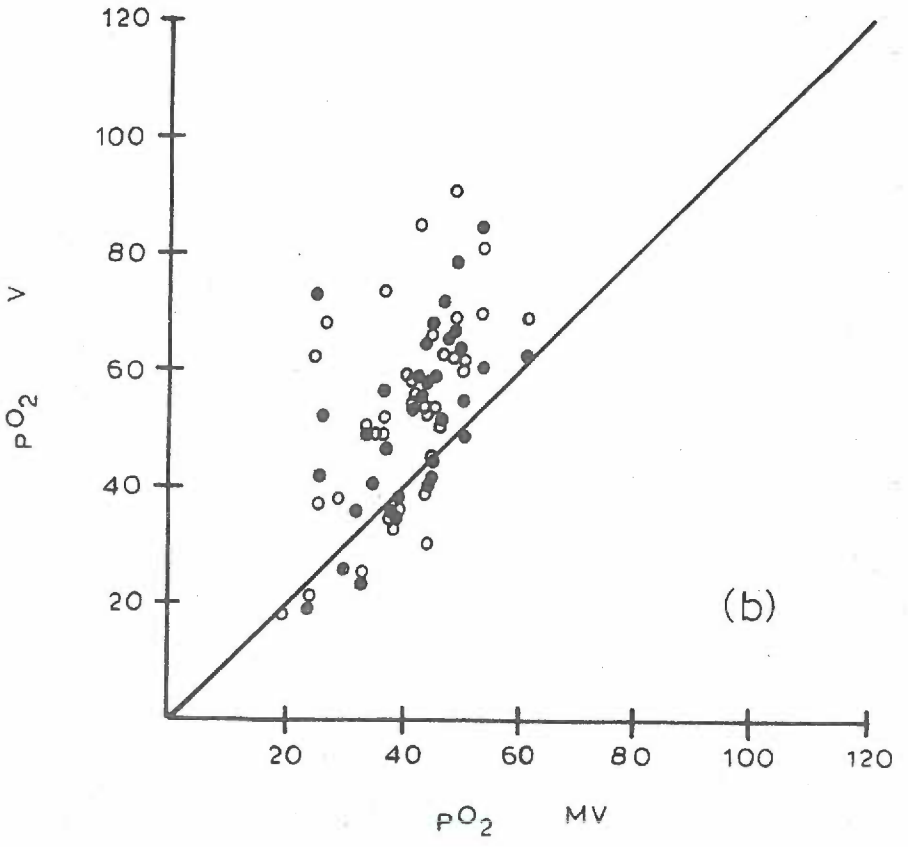
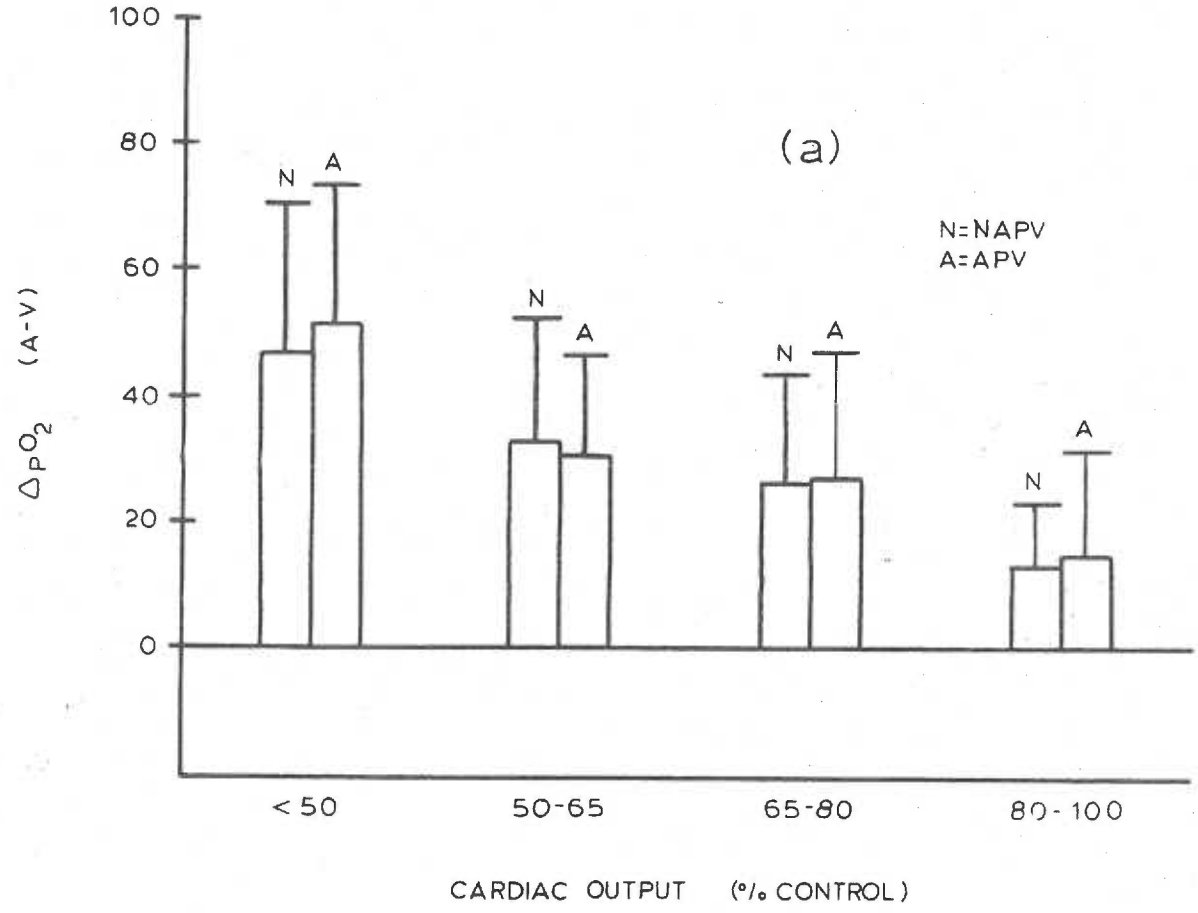

5
4
3
2
1
0
Δ [HCO3-] (MV-PV)
N
A
(b)
<50
50-65
65-80
80-100
CARDIAC OUTPUT (% CONTROL)

Figure 8. Identity relationship is shown for the pO_2 of peripheral venous (V) blood [arterialized (APV) and non-arterialized (NAPV)] as a function of (a) arterial (A) pO_2 (top figure) and (b) mixed venous (MV) pO_2 (bottom figure). Each point represents paired samples. Open circles denote NAPV blood samples whereas closed circles denote APV blood samples. Units are in mmHg.


120
100
80
60
40
20
(a)
P_{O_2} V
20
40
60
80
100
120
P_{O_2} A


120
100
80
60
40
20
P_{O_2} V
(b)
20
40
60
80
100
120
P_{O_2} MV


100
80
60
40
20
0
(a)
N=NAPV
A=APV
N
A
ΔpO_2 (A-V)
< 50
50-65
65-80
80-100
CARDIAC OUTPUT (% CONTROL)

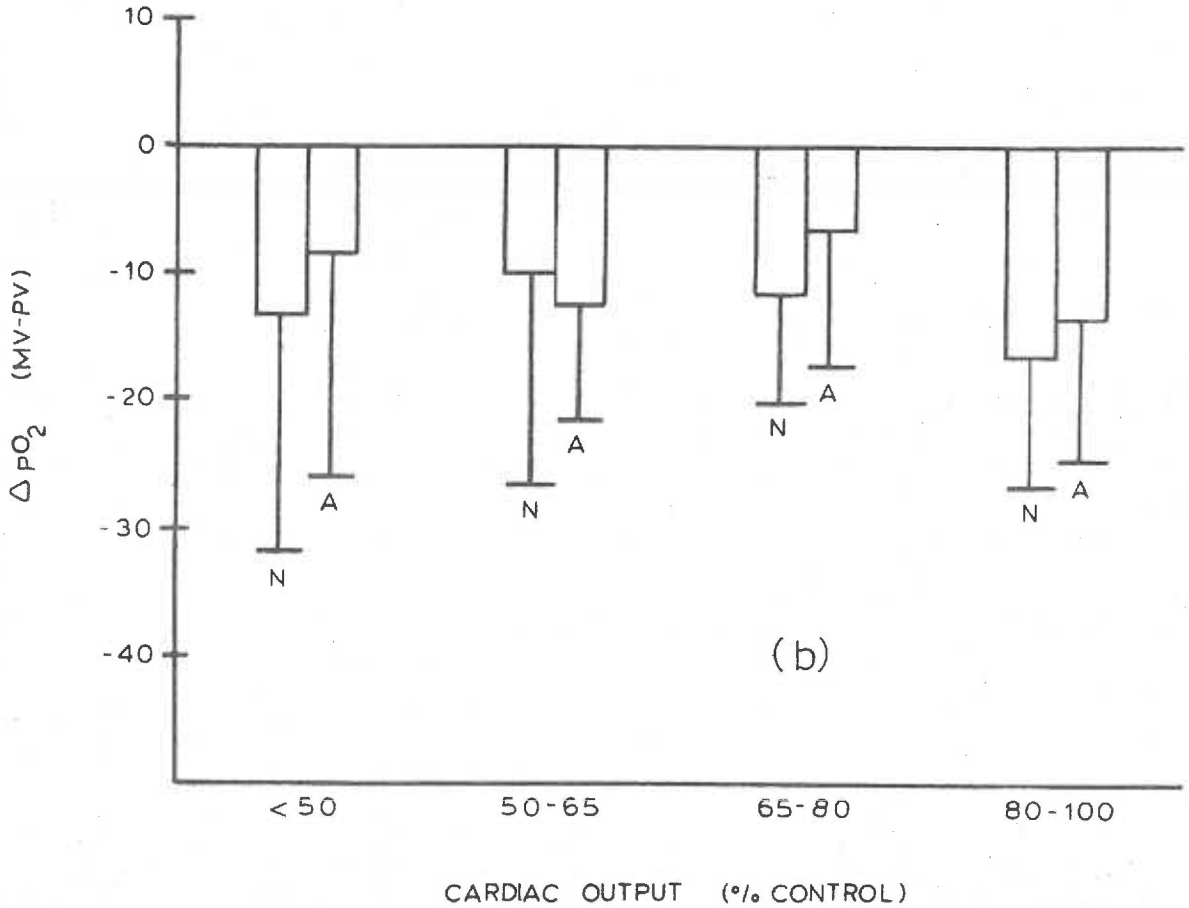

10
0
-10
-20
-30
-40
(b)
N
A
ΔpO_2 (MV-PV)
< 50
50-65
65-80
80-100
CARDIAC OUTPUT (% CONTROL)

Figure 9. (a) Mean arteriovenous (A-V) differences for pO_2 and (b) mean mixed venous-peripheral venous (MV-PV) differences for pO_2 as a function of ranges of percent control cardiac output. Venous samples included arterialized (APV) and non-arterialized (NAPV) peripheral venous blood. Units are in mmHg.

CHAPTER IV

DISCUSSION

Discussion of the results is organized under 3 headings:

1. APV and NAPV blood-gas composition compared to that of arterial blood when cardiac output is reduced.
2. APV and NAPV blood-gas composition compared to that of mixed venous blood when cardiac output is reduced.
3. Clinical implications.

Since pH, pCO_2 and $[HCO_3^-]$ are the blood-gas parameters used to assess acid-base status, they are discussed as a group under the first two headings, while the pO_2 results are discussed separately under the same headings.

1. <u>APV and NAPV Blood-Gas Composition Compared to that of Arterial Blood When Cardiac Output is Reduced</u>.

<u>pH, pCO_2 and $[HCO_3^-]$</u> Inspection of A-V identity plots for pH, pCO_2 and $[HCO_3^-]$ (Figures 2a, 4a, 6a) shows that APV and NAPV blood-gas composition correlated poorly with that of arterial blood. This is in sharp contrast to the strong correlation for these A-V identity relationships reported by other researchers (Brooks & Wynn, 1959; Paine et al., 1961; Collis & Neaverson, 1967; Schriver, 1981). However, cardiac output was not an identified variable in these studies. In this present study little change in mean A-V differences was found until cardiac output was decreased to or below 50% of control (Figures 3a, 5a, 7a). While Schriver (1981) must have reduced the cardiac output of her canine subjects when they received

mechanical ventilation, the high A-V correlation coefficients she obtained for pH, pCO_2 and $[HCO_3^-]$ suggest that cardiac output was not reduced below 50% of control in her animals.

The mean A-V differences for pH, pCO_2 and $[HCO_3^-]$ increased markedly when cardiac output was reduced to or below 50% of control. These findings are compatible with the situation of decreased peripheral venous blood flow resulting from increased vasoconstriction following blood loss. The magnitude of mean A-V differences for APV blood-gas composition was slightly more constant than that of NAPV blood when cardiac output was greater than 50% of control (Figures 3a, 5a, 7a). The process of arterialization most likely counteracted some of the vasoconstriction in peripheral venous blood vessels. Therefore, flow was artificially maintained at higher levels than flow in NAPV blood vessels until cardiac output was less than 50% of control. At or below that cardiac output arterialization was ineffective in maintaining constant A-V differences.

Hence, results of this study show that peripheral venous blood can be used to assess acid-base status when cardiac output is 50 to 100% of control. Furthermore, APV blood-gas composition as opposed to that of NAPV blood is a more suitable indicator of arterial pH, pCO_2 and $[HCO_3^-]$ over this range of cardiac output.

pO_2 In these experiments, the pO_2 values obtained from APV and NAPV blood proved unreliable for estimating arterial pO_2. This finding is similar to that reported by Schriver (1981). The magnitude of mean A-V differences noticeably increased as cardiac output was reduced (Figure 9a).

Interpretation of A-V differences. It was previously noted that blood-gas parameters for APV and NAPV blood did not all change in the same direction as those of arterial blood with reduction of blood volume. The pH and $[HCO_3^-]$ of both arterial and peripheral venous blood decreased when cardiac output was reduced. However, the pCO_2 of peripheral venous blood increased with progressive blood loss whereas that of arterial blood decreased. Conversely, the pO_2 of peripheral venous blood decreased with blood loss whereas that of arterial blood increased.

The increased pCO_2 and decreased pO_2 in APV and NAPV blood simply reflect decreased blood flow through systemic tissues. Reduced flow of oxygenated blood generates tissue hypoxia. Subsequently, anerobic metabolism ensues resulting in increased lactic acid production which is buffered with HCO_3^-. Thus, pH and $[HCO_3^-]$ decreased in both arterial and peripheral venous blood (APV and NAPV) due to systemic lactic acidosis. The acid-base status presented by APV and NAPV blood at low cardiac outputs ($\leq$ 50% of control) is consistent with a mixed acidosis, i.e., lower than normal pH and $[HCO_3^-]$, higher than control pCO_2 (Table 2).

The decreased pCO_2 and increased pO_2 in arterial blood reflects increased alveolar ventilation which can be caused by at least 2 mechanisms. These are (1) stimulation of respiratory neurons in the brain stem due to decreased blood pressure, and (2) stimulation of peripheral and possibly central chemoreceptors by H^+ from the lactic acidosis (Slonim & Hamilton, 1976). These combined effects appeared to be strongest when cardiac output was decreased below approximately

50% of control values. Thus, arterial blood-gas composition associated with these low values of cardiac output was consistent with a compensated metabolic acidosis i.e., pH, $[HCO_3^-]$ and pCO_2 less than control (Table 2).

Thus, this study clearly shows that the blood-gas composition of peripheral venous blood (APV and NAPV) indicates a different acid-base status (mixed acidosis) than that of arterial blood (compensated acidosis) when cardiac output is reduced. However, arterial blood is the recognized standard for determining acid-base status. The question must then be asked, is arterial blood the appropriate standard for determining acid-base status in all situations? If one accepts the argument that blood equilibrates with interstitial fluid (internal milieu) in systemic capillaries, then peripheral venous blood must describe systemic acid-base status more accurately than arterial blood.

Clearly, the pO_2 of arterial blood is essential for determining pulmonary function. However, a high arterial pO_2 does not guarantee adequate oxygenation of systemic tissues and organs. The pO_2 of peripheral venous blood seems the more appropriate indicator for assessment of tissue oxygenation.

2. APV and NAPV Blood-Gas Composition Compared to that of Mixed Venous Blood When Cardiac Output is Reduced.

Mixed venous blood. Up to this point it has been suggested that peripheral venous blood-gas composition more precisely reflects systemic acid-base status than that of arterial blood. As was previously discussed, a similar and earlier challenge was made by Tung et al. (1976) regarding mixed venous blood-gas composition as opposed

to that of arterial blood for systemic acid-base assessment. Mixed venous blood-gas composition, unlike that of peripheral venous blood, represents a flow-weighted average of output from all systemic tissues and organs. Therefore, the blood-gas composition of mixed venous blood should provide a more representative indication of systemic (total body) acid-base status. However, acquisition of mixed venous blood can result in undesirable and life-threatening complications such as endocarditis and arrhythmias. If the blood-gas composition of peripheral venous blood can be shown to resemble that of mixed venous blood, then peripheral venous blood sampling may prove a less invasive, more complication-free means of evaluating systemic acid-base status.

pH, pCO_2 and $[HCO_3^-]$ Schriver (1981) reported that the pH, pCO_2 and $[HCO_3^-]$ of mixed venous blood correlated closely with that of peripheral venous blood. In the present study, a close correlation is not apparent from inspection of MV-PV identity plots for these blood-gas parameters (Figures 2b, 4b, 6b). However mean MV-PV differences, like A-V differences, remained fairly constant until cardiac output was at or below 50% of control (Figures 3b, 5b, 7b). Therefore, as previously suggested, the cardiac output of Schriver's animals must not have decreased below this percent of control cardiac output.

In general, the magnitude of mean MV-PV differences was markedly less than that of mean A-V differences. Furthermore, the magnitude of mean MV-PV differences remained slightly more constant for APV blood than that of NAPV when cardiac output was greater than 50% of control. Warming the peripheral venous sampling site artificially maintained

flow at a higher rate whereby the pH, pCO_2 and $[HCO_3^-]$ of APV blood more closely resembled that of flow-weighted venous blood i.e., mixed venous blood in the pulmonary artery.

The pH, pCO_2 and $[HCO_3^-]$ of peripheral venous and mixed venous blood all changed in the same direction when cardiac output was reduced i.e., pH and $[HCO_3^-]$ decreased, pCO_2 increased. As a result, mixed venous and peripheral venous blood-gas compositions reflected the same acid-base status (mixed acidosis) at low values of cardiac output. This is in sharp contrast to the lack of agreement found when comparisons of peripheral venous and arterial blood-gas compositions showed that the pCO_2 values changed in opposite directions as cardiac output was reduced.

$\underline{pO_2}$ Peripheral venous pO_2 values did not correlate as closely with those of mixed venous blood as did other blood-gas parameters. (See Figure 8b). This finding is similar to that reported by Schriver (1981). In the majority of dogs, mixed venous pO_2 values decreased more than those of peripheral venous blood in the extremities (Table 2; Figure 9b). Certainly there is reduced flow to the extremities, but these tissues have a lower metabolic activity and, hence, lower O_2 consumption than vital organs. Therefore, venous blood draining the extremities may not show a dramatic decrease in pO_2 until flow is more severely restricted. Conversely, mixed venous blood contains venous drainage from tissues with both low and high O_2 consumption. Since the vital organs have higher O_2 consumptions than extremities, the mixed venous blood will show a more marked decrease in

pO_2 than peripheral venous blood in the extremities as cardiac output (flow) is decreased.

In some cases the pO_2 values for APV and NAPV blood were less than those of mixed venous blood. This was not observed only at low ($\leq$ 50% of control) values for cardiac output, but occurred at higher values of cardiac output as well. The reason for this finding is uncertain. One possible explanation, however, is that sympathetic stimulation causing vasoconstriction was predominant over autoregulation of arterioles in the extremities in these cases.

The magnitude of mean MV-PV differences for pO_2 remained fairly constant over a wide range of cardiac output (50 to 100% of control) Figure 9b). Furthermore, NAPV blood seemed to provide more constant mean MV-PV differences for pO_2 than APV blood. Hence, the pO_2 of NAPV blood was a more reliable indicator of mixed venous pO_2 when cardiac output was reduced.

3. Clinical Implications

This study shows that peripheral venous blood-gas composition resembles that of mixed-venous blood over a wide range of changes in cardiac output (50 to 100% of control). Mixed venous blood-gas composition may eventually become the recognized standard for determining systemic tissue oxygenation and acid-base status. In that eventuality, peripheral venous blood-gas composition may be used in lieu of mixed venous blood to make these assessments. This would obviate the need for routine insertion of Swan-Ganz catheters for sampling mixed venous blood. Hence, an invasive, dangerous and painful procedure can be supplanted by a simple venipuncture or

aspiration from an indwelling venous catheter for some patients.

It is recognized that some patients will require insertion of Swan-Ganz catheters for mixed venous sampling. These individuals would have a severely reduced cardiac output which would (1) increase the difficulty of obtaining peripheral venous blood samples, (2) reduce the reliability of peripheral venous blood-gas composition as an index of systemic tissue oxygenation and acid-base status, and (3) require constant monitoring for evaluating efficacy of therapies. However, measurement of the pO_2 of peripheral venous blood to assess and monitor blood flow, hence cardiac output, could reduce the need for prolonged catheterization following a crisis period. Therefore, the increased risk of complications associated with prolonged Swan-Ganz catheterization could be reduced.

Arterial blood-gas composition remains the recognized standard for acid-base assessment. Unquestionably, arterial blood samples are necessary for determining pulmonary function. However, if pulmonary assessment is not indicated, then peripheral venous blood-gas composition can be used to assess acid-base status providing that cardiac output is not severely reduced. Peripheral venous blood sampling via a simple venipuncture would reduce and/or eliminate unnecessary risk and discomfort to patients that is incurred by arterial sampling.

Caution must be employed by practitioners when interpreting peripheral venous blood-gas composition. The unwary practitioner may incorrectly assume that a low pH and high pCO_2 in peripheral venous blood indicates <u>only</u> systemic acid-base status. A high pCO_2 in peripheral venous blood could be indicative of pulmonary dysfunction.

Therefore, arterial blood-gas composition would have to be evaluated to rule out such a possibility.

Peripheral venous blood samples were difficult to obtain when cardiac output was reduced. In the clinical setting, peripheral venous blood samples are routinely obtained from arm veins. A reduced cardiac output and/or pre-existing peripheral vascular disease can make these samples difficult if not impossible to obtain in some patients. Therefore, it is suggested that this study be replicated using a larger caliber venous vessel such as the femoral vein.

It is recognized that the results of this study were obtained using an animal model. Qualitatively, a similar pattern of change in arterial, peripheral venous and mixed venous blood-gas compositions would be expected in human subjects. Quantitatively, it is not known if peripheral venous blood-gas composition can be used to assess acid-base status in humans over the same range of percent change in cardiac output observed in dogs. Furthermore, the effect of anesthetic and canine splenic contraction on these results is not known. A similar study involving human subjects with low values of cardiac output is needed to establish the clinical utility of peripheral venous samples for assessing acid-base status.

Additionally, some comments must be made regarding the observed standard deviations. These dogs did not start the experiment with precisely the same blood-gas composition, but displayed the kinds of variations which would be present had the study been done with critically ill human subjects. Therefore, while the pattern of change in blood-gas composition was largely the same for all dogs, the

deviation from mean MV-PV and A-V differences primarily reflects variation within the group. It must also be noted that the ordinate scale is expanded further magnifying what are actually small variations. Furthermore, averaging MV-PV and A-V differences in blood-gas parameters for ranges of percent cardiac output contributed towards increasing the standard deviation. Hence, it is unlikely that this amount of variance would be observed in samples obtained from single subjects.

CHAPTER V

SUMMARY AND CONCLUSIONS

Arterial blood is the recognized standard for determining pulmonary function and acid-base status. More recently, however, it has been challenged that mixed venous blood is a more representative index of systemic acid-base status. Since both arterial and mixed venous blood sampling involve painful procedures associated with multiple complications, several investigators have explored the possibility of using peripheral venous blood for acid-base assessment. None of these investigations were conducted with cardiac output as an identified variable.

In the present study, the blood-gas composition of peripheral venous blood (APV and NAPV) was compared to that of mixed venous and arterial blood when cardiac output was acutely reduced.

Successive decreases in the cardiac output of 10 canine subjects was achieved by reducing blood volume. Subsequently, cardiac output was measured using thermodilution technique and a cardiac output computer. Samples of arterial, mixed venous and peripheral venous (APV and NAPV) blood were obtained simultaneously and the blood-gas composition determined with each change in cardiac output.

The results of these experiments show that the pH, pCO_2 and $[HCO_3^-]$ of peripheral venous blood can be used to assess systemic acid-base status when cardiac output is at or greater than 50% of control. Additionally, the pO_2 of peripheral venous blood does not reliably indicate that of arterial blood. Therefore, the pO_2 of peripheral venous blood cannot be used for assessment of pulmonary function.

However, the pO_2 of peripheral venous blood was shown to more closely reflect that of mixed venous blood when cardiac output was at or greater than 50% of control. Hence, tissue oxygenation may be assessed from peripheral venous blood pO_2.

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AN ABSTRACT OF THE THESIS OF

PAULA MAURER FELDEN

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Title: THE USE OF PERIPHERAL VENOUS BLOOD TO ASSESS ACID-BASE STATUS WHEN CARDIAC OUTPUT IS REDUCED

Approved: [redacted]
Jack L. Keyes, Ph.D., Thesis Advisor

Arterial blood has traditionally been used to assess pulmonary function and acid-base status. More recently, however, it has been challenged that mixed venous blood-gas composition provides a more representative indication of systemic acid-base status. The many disadvantages and complications associated with arterial and mixed venous sampling led to investigations concerning the reliability of using peripheral venous blood to assess systemic acid-base status. None of these investigations were conducted with cardiac output as an identified variable. In the present study the blood-gas composition of arterialized peripheral venous (APV) and non-arterialized peripheral venous (NAPV) blood was compared to that of arterial and mixed venous blood when cardiac output was acutely reduced.

The cardiac output of 10 experimental animals was decreased by progressively reducing their blood volume. Cardiac output was subsequently measured using thermodilution technique. Arterial, mixed venous, and peripheral venous (APV and NAPV) blood samples were

obtained simultaneously after each change in cardiac output.

The results of these experiments show that the pH, pCO_2 and $[HCO_3^-]$ of peripheral venous blood can be used to assess systemic acid-base status when cardiac output is at or greater than 50% of control. Additionally, the pO_2 of peripheral venous blood does not reliably indicate that of arterial blood. Therefore, the pO_2 of peripheral venous blood cannot be used for assessment of pulmonary function. However, the pO_2 of peripheral venous blood was shown to more closely reflect that of mixed venous blood when cardiac output was at or greater than 50% of control. Hence, tissue oxygenation may be assessed from peripheral venous blood pO_2.