ON LINE IN VIVO MEASUREMENT OF TISSUE AND BLOOD GASES IN PATIENTS WITH SEVERE TRAUMA

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SURFACE pH of skeletal muscle has been shown by Dmochowski and Couch (2) to be a sensitive, indirect indicator of peripheral blood flow. This is in accordance with the concept that decreased tissue perfusion causes increased hydrogen ion activity as a consequence of anaerobic metabolism with lactic acid production.

The medical mass spectrometer has made possible continuous in vivo measurement of pO_2 and pCO_2 in blood, tissue, and respired air.

The objective of this study is to determine normal tissue fluid values for pO_2-pCO_2 in vivo; to determine the status of peripheral perfusion by use of the tissue pO_2-pCO_2 levels; and to study the effects of changes in ventilation and oxygenation of the arterial blood on the tissue pO_2-pCO_2 levels.

MATERIALS AND METHODS

The criteria for admission to the study included shock of any cause; trauma, secondary to stab wound; gunshot wound or blunt trauma; and severe injury associated with a major surgical procedure. Nine consecutive patients admitted to the Shock-Trauma Unit were studied. A total of ten studies were conducted, one patient having been studied at two separate times. Total patient study time was 527 hours, with a range of 12 to 96 hours and a mean of 52.7 hours.

Vital signs were monitored at least hourly and more frequently if it was believed necessary. Blood pressures, electrocardiograms, temperature, and pulmonary artery pressure were monitored continuously. Pulmonary wedge and central venous pressures were monitored hourly. Pulmonary artery and wedge pressures were obtained by use of an indwelling catheter connected to a strain gauge transducter. Arterial pressure was monitored by use of an indwelling radial artery catheter connected to a strain gauge transducer. Temperature was monitored by means of a rectal probe connected to a temperature monitor. Electrocardiogram was monitored by an oscilloscope connected to a cardiotachometer. All patients had indwelling catheters for the measurement of hourly urine output.

Muscle tissue pO_2-pCO_2 ($P_TO_2-P_TCO_2$) determinations were made by means of a special, Teflon® (polytetrafluoroethylene)—covered, nonthrombogenic catheter with a sampling rate of 10^{-6} cubic centimeters of gas per second, which was inserted into the right or left deltoid muscle by a cutdown technique in four studies and percutaneous technique in six studies. These catheters were connected to the medical mass spectrometer. In practice, tissue gases diffused across the Teflon covering, and a small quantity of the gases was transmitted through a 22 gauge stainless steel tubing to the medical mass spectrometer by means of machine-generated vacuum of 10^{-5} millimeters of mercury. Equilibration time with this system was 15 minutes, while the sampling delay time was less than two minutes. The theory of the mass spectrometer operation is explained by Dardik and his associates (1) but basically involves separation according to atomic mass numbers.

Arterial and venous blood gas readings were obtained at least every six hours. Lactate and pyruvate levels were obtained daily. Blood volume determinations were done on admission to the study group by the Risa® (radioiodinated serum albumin) method. Hematocrit and hemoglobin determinations and electrolytes were obtained serially. Cardiac output measurements were made for all patients by dye dilution techniques, using a cardiac output computer.

Of the ten studies, six were used to establish control values. Two of the six patients who served as controls were studied both preoperatively and postoperatively, while the remaining four were studied only postoperatively until stabilization of tissue gas levels was observed. These six patients also formed the postoperative study. All of the patients underwent extensive abdominal operations with general inhalation anesthesia.

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Supported in part by Merck, Sharpe, and Dohme Research Laboratories.

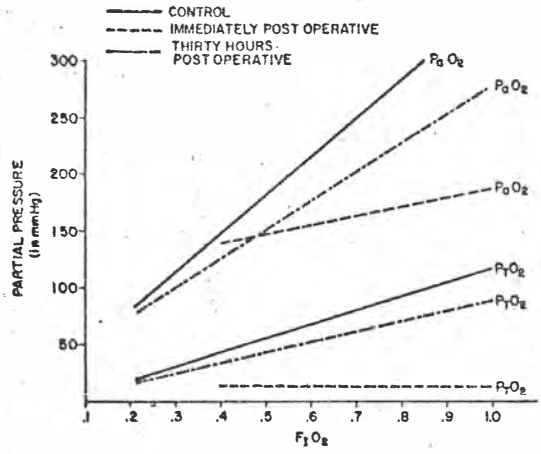

FIG. 1.

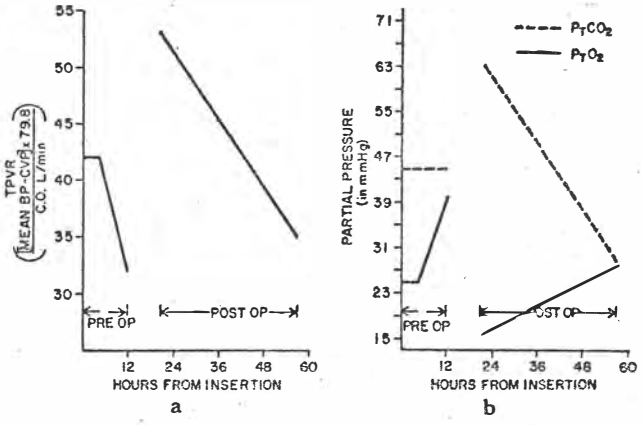

FIG. 2.

FIG. 1. Partial pressure and F_IO_2.

FIG. 2. a, Total peripheral vascular resistance and hours from insertion. b, Partial pressure and hours from insertion.

Three patients in a state of shock were studied, each representing a different cause of shock—cardiogenic, septic, and hemorrhagic. The diagnoses of shock were made if the patients became cold, became clammy, tachycardia developed, the mean arterial blood pressure was below 80 millimeters of mercury, and if they became oliguric or anuric. All patients in this group were treated aggressively with fluids, colloids, and 5 milligrams per kilogram of Decadron® (dexamethasone). They also were treated with Dibenzyline® (phenoxybenzamine hydrochloride), digitalis, Aramine® (metaraminol), and Levophed® (levarterenol) as indicated.

RESULTS

Control study. Recorded values were plotted for six consecutive hours after stabilization, and a mean value was derived for each patient. These individual mean values were summed to obtain a mean value and standard deviation for $P_TO_2 - P_TCO_2$ for the group. During this period blood pressure was maintained between 100 to 140 millimeters of mercury. Blood pressures remained stable in all six patients who served as controls.

Postoperative study. Immediately postoperatively, the P_TCO_2 values were found to be greater than normal, ranging from 38 to 65, with a mean of 51.7 millimeters of mercury; P_TO_2 values, on the other hand, were lower than normal, ranging from 14 to 32 millimeters of mercury, with a mean value of 20 millimeters of mercury. Again, the PaO_2 was maintained in this phase of the study over 100 millimeters of mercury, with a range of 100 to 140 millimeters of mercury. All six patients studied had oxygen supplementation by mask, cannula, or respirator. Stabilization with return to normal $P_TO_2 - P_TCO_2$ values was found to occur between 12 and 24 hours postoperatively. In both patients studied preoperatively, values returned to normal during this postoperative period (Fig. 1).

It also was found that in the early postoperative period, prior to tissue value stabilization, increased fraction of inspired oxygen had no affect on P_TO_2 values (Fig. 2a and b). After $P_TO_2 - P_TCO_2$ values had stabilized, increases in F_IO_2 increased the P_TO_2 in a linear manner, while P_TCO_2 values remained unchanged.

Periods of hyperventilation, however, lowered the PCO_2 in both blood and tissue, while periods of hypoventilation were noted to increase both arterial and tissue P_TCO_2 levels.

Lactate levels immediately postoperatively were elevated to a mean of 21.1 milligrams per cent with a range of 16 to 24 milligrams per cent, while the normal value was 6 to 16 milligrams per cent. Twenty-four hours postoperatively, lactate levels decreased to a mean of 9.2 milligrams per cent, with a range of 5.9 to 12.9 milligrams per cent.

Shock study. The patients in states of septic and hemorrhagic shock responded to therapy but, subsequently, both died from massive pulmonary emboli proved at autopsy. They were maintained for 48 and eight hours, respectively, after the diagnosis of shock was made. The sole patient in a state of cardiogenic shock died after 36 hours of therapy.

PaO_2 was maintained between 200 to 300 millimeters of mercury and $PaCO_2$, 36 to 40 millimeters of mercury, with all three patients being connected to volume-cycled respirators.

Initially, the P_TO_2 levels of all three patients were

extremely low, with a mean of 15 millimeters of mercury. P_TCO_2 levels were elevated to 63 millimeters of mercury. In the septic and hemorrhagic shock groups of patients, the P_TCO_2 values began to decline with improvement in clinical condition but P_TCO_2 values remained low with a mean of 20 millimeters of mercury (Fig. 1). In all patients, serial lactate levels were above normal, with a mean of 33.4 milligrams per cent, ranging from 22.3 to 54 milligrams per cent.

In the patient in the cardiogenic shock group, P_TCO_2 levels declined with therapy but not to normal values. P_TO_2 values did not increase until therapy was begun with alpha adrenergic blockers. After this, the P_TO_2 rose dramatically and P_TCO_2 decreased. In Figure 3, the increased P_TO_2 and decreased P_TCO_2 are shown, with decreased peripheral resistance in the patient in a state of cardiogenic shock after alpha adrenergic blockade with Dibenzyline. Fluctuation of P_TO_2 versus total peripheral vascular resistance in other types of shock currently is being evaluated in our laboratory.

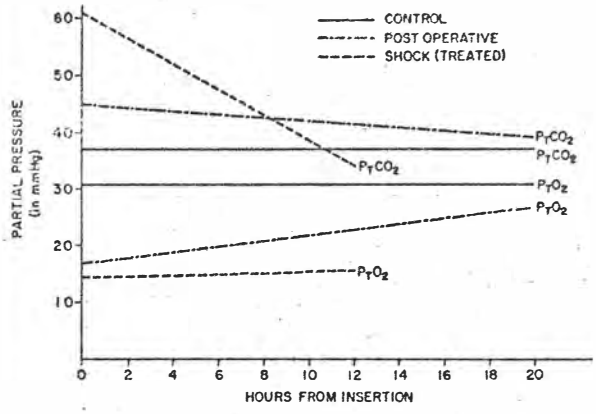

FIG. 3. Partial pressure and hours from insertion.

DISCUSSION

The results of this study indicate that patients both in a state of shock and those in the immediate postoperative state exhibit marked peripheral vasoconstriction, greater in degree in the shock state. The effect of this peripheral vasoconstriction is the redistribution of blood flow from the peripheral tissues to the central circulation, resulting in local tissue hypoxia, hypercarbia, and anaerobic metabolism, with the resultant accumulation of lactic acid.

Arteriovenous shunts may be created in the peripheral circulation which then bypasses the tissue capillary flow. In certain periods, increasing total peripheral vascular resistance, secondary to increased catecholamine production, is concomitant with increased intra-arterial pressure or a decrease in cardiac output. In an attempt to maintain adequate intra-arterial pressure, resistance continues to rise. If resistance at the capillary level becomes too great, blood is shunted away from the tissue to the venous side of the circulation. Postoperatively, the vasoconstriction peripherally is not as intense as in the patient in a state of shock; therefore, arteriovenous shunting is not exhibited to the same degree as it is in the patient in a state of shock.

In both types of patients, increased PaO_2 has little effect in increasing P_TO_2. After 12 to 24 hours in the postoperative state, the P_TO_2 returns to normal values. Additionally, it responds to increases in arterial oxygenation. The explanation is believed to be related to improved tissue perfusion and decreased shunting at the microcirculatory level. Decreasing lactate levels also tend to support this increased perfusion.

The patients in a state of shock exhibited a severe decrease in peripheral perfusion to a greater degree than did those after operation. With a decrease in peripheral resistance after alpha blockade, an increase in P_TO_2 and a decrease in P_TCO_2 occur as a consequence of the increased perfusion, despite the continued low cardiac output. The continuing low P_TO_2 noted in the patients in a state of shock as clinical improvement occurred and, as evidenced by the decreased P_TCO_2, may represent an increase in oxygen consumption, with slightly improved perfusion.

The medical mass spectrometer used to measure $P_TO_2 - P_TCO_2$ levels provides an accurate, rapid, and clinically useful indirect method for the estimation of the status of peripheral blood flow at the bedside. Evaluating treatment and planning future therapy of acutely ill patients thus are facilitated.

SUMMARY

A medical mass spectrometer was used in the evaluation of peripheral blood flow in critically ill patients. Normal values for tissue $PO_2 - PCO_2$ have been obtained by use of this instrument, and the relationship of tissue $PO_2 - PCO_2$ to perfusion status was determined. The effects of ventilation and arterial oxygenation on tissue $PO_2 - PCO_2$ have been demonstrated in both postoperative and shock states and, thus, facilitates the evaluation of therapy in acutely ill patients.

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