AN EXPLORATION OF THE CORRELATION BETWEEN LACTIC ACID LEVELS AND BODY TEMPERATURE IN HYPOTHERMIC, MODERATELY INJURED TRAUMA PATIENTS

by

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INTRODUCTION

Statement of Problem

Trauma care professionals thrive on the melodrama of a critically injured patient. The scene of a trauma resuscitation includes bright lights, instrument trays, loud noises, blood, and rushing doctors and nurses. The intensity of the situation is usually based on the patient's level of consciousness and amount of blood lost. During all phases of resuscitation, major attention is focused on the patient's "ABCs": airway, breathing, and circulation. Body temperature is a critical parameter that is frequently overlooked in all of the excitement; however, physiologic changes that occur in hypothermic patients may pose serious threats to the patient's survivability.

Curiously, a body temperature between 25-30 degrees centigrade is desirable in open-heart surgery patients because it slows metabolism, and delays the onset of anaerobic metabolism (Guzetta & Whitman, 1985; Rueler, 1978). In the severely injured trauma patient, however, hypothermia is associated with a high

mortality rate (Jurkovich, Greiser, & Luterman, 1987). How is it that hypothermia contributes to the survivability of one patient population and the mortality of another? Perhaps the answer is that hypothermia affects the oxygen-hemoglobin dissociation curve in such a way that cellular hypoxia and lactic acidosis result. The acidosis, then, is perhaps a reflection of cellular compromise that contributes to the morbidity of hypothermic trauma patients.

The subject of hypothermia has obvious clinical significance. Nurses need to know whether they should be comforted or concerned about the presence of hypothermia in trauma patients and how aggressively they should rewarm these patients to avoid the harmful consequences of hypothermia. They need to know if hypothermia does, in fact, lead to the increased production of lactic acid due to cellular hypoxia. The purpose of this study was to determine if there was a correlation between lactic acid levels and hypothermia in moderately injured trauma patients.

Definitions

The concept of hypothermia, or low body

temperature, can be divided into two separate categories: environmental and clinical. More familiar to the general public, environmental hypothermia is associated with over-exposure to the outside environment resulting in extreme drops in body temperature, coma, and death when untreated. Conversely, clinical hypothermia is a phenomenon well known in the emergency, peri-operative, and intensive care arenas. Feroe and Augustine (1991) define clinical hypothermia as an iatrogenic peri-operative complication resulting in a core body temperature of 31-36 degrees centigrade or 92-97 degrees farenheit. They add that it adversely affects physiologic processes and complicates the patient's recovery from anesthesia and major surgery. Clinical hypothermia is a well documented problem in the cardiac surgery and anesthesia literature and is now receiving increased attention in trauma research.

Review of the Literature

Much information is available about clinical hypothermia and its measurement in the nursing, surgical, and anesthesia literature. The mechanisms of heat loss predisposing the typical trauma patient to

clinical hypothermia are described below. Then, the pathophysiologic consequences of clinical hypothermia are reviewed, followed by an examination of its impact on trauma patients. Lastly, gaps in the available literature are identified.

Mechanisms of Body Heat Loss

There are four mechanisms by which the trauma patient's body reacts with the environment to lose heat: radiation, conduction, convection, and evaporation. Heat loss via radiation occurs when the uncovered trauma patient radiates body heat waves to the cooler environment. Up to 60% of the total heat lost can occur via this mechanism. Conduction of heat away from the body occurs as the patient lies on cold examination tables and receives infusions of cold intravenous fluids. Convection contributes to hypothermia as air currents around patients move heat away from the skin surface. Finally, evaporation causes heat loss as body fluids evaporate from the skin, as well as through insensible water loss, to the environment. Normally this last mechanism occurs at a rate of 12-16 calories per hour; but it is accelerated when oxygen is not humidified and moist body organs are

exposed to the dry environment (Guyton, 1981).

Heat loss is common in trauma patients as clothing is removed to evaluate the patient, fluids are administered for resuscitation, and internal organs are exposed to the environment during surgery. Any trauma patient will experience these mechanisms of heat loss, and can become vulnerable to the adverse physiologic consequences of a lowered body temperature.

Pathophysiologic Consequences

All of the major body systems are affected by temperatures between 32 and 36 degrees centigrade. The cardiovascular system demonstrates the most profound changes associated with clinical hypothermia. The blood pressure rises, initially in response to circulating catecholamines and vasoconstriction, but then hypotension occurs and cardiac output falls (Rueler, 1978). This rise in systemic vascular resistance puts obvious stress on the left heart by significantly increasing its workload. Pulmonary vascular resistance and central venous pressure also increase (Morrison, 1988). Significant life-threatening conduction changes also occur, as are reflected in the electrocardiogram, further

compromising cardiac function.

The electrocardiogram changes demonstrate a generalized slowing of the heart rate, T-wave inversion, and prolongation of the PR, QRS, and QT intervals. A slow, positive deflection at the end of the QRS complex, known as the Osborn or J wave, is frequently associated with hypothermia (Bessen, 1985). In addition, atrial fibrillation is not uncommon (Rueler, 1978). Fascicular and nodal blocks occur at temperatures less than 32 degrees centigrade (Morrison, 1988). Should hypothermia progress, fatal ventricular fibrillation and asystole are likely to occur (Rueler, 1978).

Changes in the hematopoeitic system are especially dangerous to the bleeding trauma patient. In the past, hypothermia-induced vasoconstriction was thought to inhibit serious bleeding. On the contrary, hypothermia only worsens the patient's propensity for hemorrhage (Feroe & Augustine, 1991). There is a loss of intravascular plasma proteins into the interstitial space, resulting in a falsely reassuring rise in hematocrit. Leukocyte and platelet counts decrease as they are sequestered in the spleen and liver (Rueler,

1978). This sequestering produces the referred disseminated intravascular coagulation that is frequently documented in the hypothermic trauma patient.

The exact etiology of the coagulation disorders in hypothermic trauma patients is unclear (Patt, McCroskey, & Moore, 1988). However, it is known that the significant bleeding problems noted in these patients are due to changes in platelet response time, structure, and function. Coagulation is an enzymatic chain of events dependent upon normothermia to function correctly. When slowed, platelet integration into the fibrin clot is delayed. Bleeding times, clotting times, and prothrombin consumption times are lengthened. The platelets are no longer disc shaped, become swollen with hypoplasma, develop pseudopods, and become "overly sticky." At hypothermic temperatures, the platelets then collect and form clumps so that platelet transfusions are of little use. Fibrinolytic activity is also increased. These changes in the coagulation cascade reverse with rewarming, but cellular damage from hypothermia releases large amounts of thromboplastin into the circulation as the patient

rewarms, resulting in additional clotting problems (Patt et al, 1988).

Mild changes in the respiratory system occur with clinical hypothermia. Research indicates that there is a decrease in the hypoxic ventilatory drive to breathe as the body temperature falls. The lungs become less compliant as bronchomotor tone increases. This expands dead space volume and worsens ventilation/perfusion mismatches. Hypoventilation and diaphragmatic dysfunction are due to the increased muscle tone that accompanies severe shivering (Morrison, 1988). Also the decreased level of consciousness associated with mild hypothermia leads to decreased cough and gag reflexes, predisposing the patient to aspiration (Rueler, 1978).

Of particular significance is the effect on the oxygen-hemoglobin dissociation curve. It has long been known that low body temperature causes a left shift of the oxygen-hemoglobin dissociation curve as hemoglobin increases its affinity for oxygen (Rueler, 1978). Although the hemoglobin molecule is well saturated with oxygen, it will not release oxygen to the tissues, causing tissue hypoxia. This occurs at a time when oxygen consumption is increased from shivering and

thermogenesis. Pulmonary reserve is minimal, and arterial desaturation is inevitable (Feroe & Augustine, 1991).

Neurologic compromise is most evident with severe hypothermia, but there is a 70% decrease in short term memory at temperatures of 34 degrees centigrade (Morrison, 1988). Altered mentation can also be explained by the 6-7% decrease in cerebral blood flow for every 1 degree centigrade decrease in body temperature (Rueler, 1978). The cold, anesthetized patient is slow to recover from anesthesia, further confusing the neurological assessment (Feroe & Augustine, 1991).

Abdominal and retroperitoneal organs are compromised by clinical hypothermia. Paralytic ileus occurs with temperatures of 34 degrees centigrade or less. Metabolism of medications by the liver is delayed, causing prolonged effects of drugs and anesthetics. Renal blood flow and urine output decrease with hypothermia as cardiac output falls and renal vasoconstriction occurs. Eventually, however, sodium and water reabsorption are impaired resulting in a "cold diuresis" in severely hypothermic patients

(Bessesn, 1985; Morrison, 1988; Rueler, 1978).

Lastly, overall body metabolism is adversely affected by even mild degrees of hypothermia. Hyperglycemia results from suppressed insulin release and inhibition of glucose uptake. Hypothermia provides a transient benefit by decreasing the basal metabolic rate (BMR) during hypoperfusion. Unfortunately, it exacerbates cellular hypoxia, thereby disrupting cell membranes, altering ion gradients, and starting a cascade of chain reactions resulting in cell death (Hochachka, 1986). Tissue hypoxia from insufficient tissue perfusion and carbon dioxide retention from respiratory failure and shivering result in acidosis. This acidosis is intensified by delayed hepatic clearance of lactate. Circulatory failure prevents renal buffering and excretion of organic acids (Rueler, 1978). Finally, protein catabolism and hyperkalemia develop, further impairing cardiac and renal function at a time when they are critical for survival (Feroe & Augustine, 1991).

The physiologic consequences of hypothermia, as described above, have the potential to affect any patient who becomes hypothermic in the clinical

setting. Unfortunately, trauma patients are particularly vulnerable to clinical hypothermia and its effects.

Impact of Clinical Hypothermia on Trauma Patients

Hypothermia has been documented in the majority of severely injured patients and it takes a long time to rewarm them (Luna et al, 1987). Severely traumatized cold patients have higher mortality rates than warm patients with identical injuries (Jurkovich, Greiser, & Luterman, 1987). Severely injured patients require large volumes of fluid resuscitation which can contribute to conductive heat loss. In two groups with similar injuries receiving large amounts of fluid, the warm patients had a lower mortality rate than the cold patients (Jurkovich et al., 1987).

In their 1991 study of 100 trauma patients undergoing operations, Gregory, Flancbaum, Townsend, Cloutier and Jonasson found that 92% of their patients were hypothermic in the emergency department and 43% were hypothermic in the operating room. Their data suggested that hypothermia in trauma patients has a multifactorial etiology related to the magnitude of

injury. Furthermore, research suggests that compensatory thermogenesis is impaired in the severely injured trauma patient (Steinemann et al, 1990).

The elderly trauma patient is at even more risk for developing clinical hypothermia. Physiologic changes associated with aging predispose the injured geriatric patient to clinical hypothermia. Heat loss is accelerated because decreases in subcutaneous fat fail to insulate the patient from conductive heat losses. Autonomic dysfunction causes venous pooling in cool extremities and impairs return to the warm core circulation (Rueler, 1978).

The elderly trauma patient is even less mobile than a younger patient, thereby generating less heat through activity. Peripheral sensation is diminished, prolonging exposure to cold objects (Luna et al, 1987). There is a greater ratio of surface area to body mass which increases convective, conductive, radiant, and evaporative heat losses (Morrison, 1988). In addition, metabolic changes and a high incidence of co-existing disease adversely affect the elderly trauma patient's ability to tolerate hypothermia as well as the younger patient (Morrison, 1988; Rueler, 1978).

Gaps in the Literature

The physiologic literature and trauma research cited above clearly demonstrate that clinical hypothermia is frequently documented in severely injured trauma patients. However, many questions about the role of clinical hypothermia in trauma remain unanswered.

While it is well known that clinical hypothermia exists with severe injury, there is no clarification as to whether it directly contributes to the mortality of the patient or merely coexists. There is no research investigating the point at which clinical hypothermia begins to influence physiologic well-being when associated with severe injuries. Such information would have obvious clinical significance: knowledge regarding the role of clinical hypothermia in patient outcome would surely expedite efforts to rewarm patients with low body temperatures.

Another limitation found in the studies is a lack of information about the incidence of hypothermia in the more common, moderately injured trauma patient. It is unknown if hypothermia occurs in all trauma patients, or only those who are seriously injured.

None of the studies assessed the physiologic parameters associated with hypothermia that might suggest altered oxygen delivery and consumption, such as shivering, oxygen desaturation, and elevated lactic acid levels.

The primary question, therefore, is whether or not the consequences of hypothermia directly contribute to the morbidity and mortality of trauma patients. It is accepted that cellular hypoxia occurs when hypothermia shifts the oxygen-hemoglobin dissociation curve to the left. This hypoxia causes inefficient anaerobic metabolism and the subsequent production of lactic acid. The presence of elevated lactic acid levels in the bloodstream, then, would suggest that significant metabolic compromise has occurred. If lactic acidosis occurs in patients who are cold but hemodynamically stable (thus eliminating hemorrhagic shock as the etiology of the acidosis), it could suggest that hypothermia itself causes adverse physiologic changes in trauma patients.

The purpose of this study, then, was to determine if there was a correlation between lactic acid levels and body temperature in the hypothermic, moderately injured trauma patient.

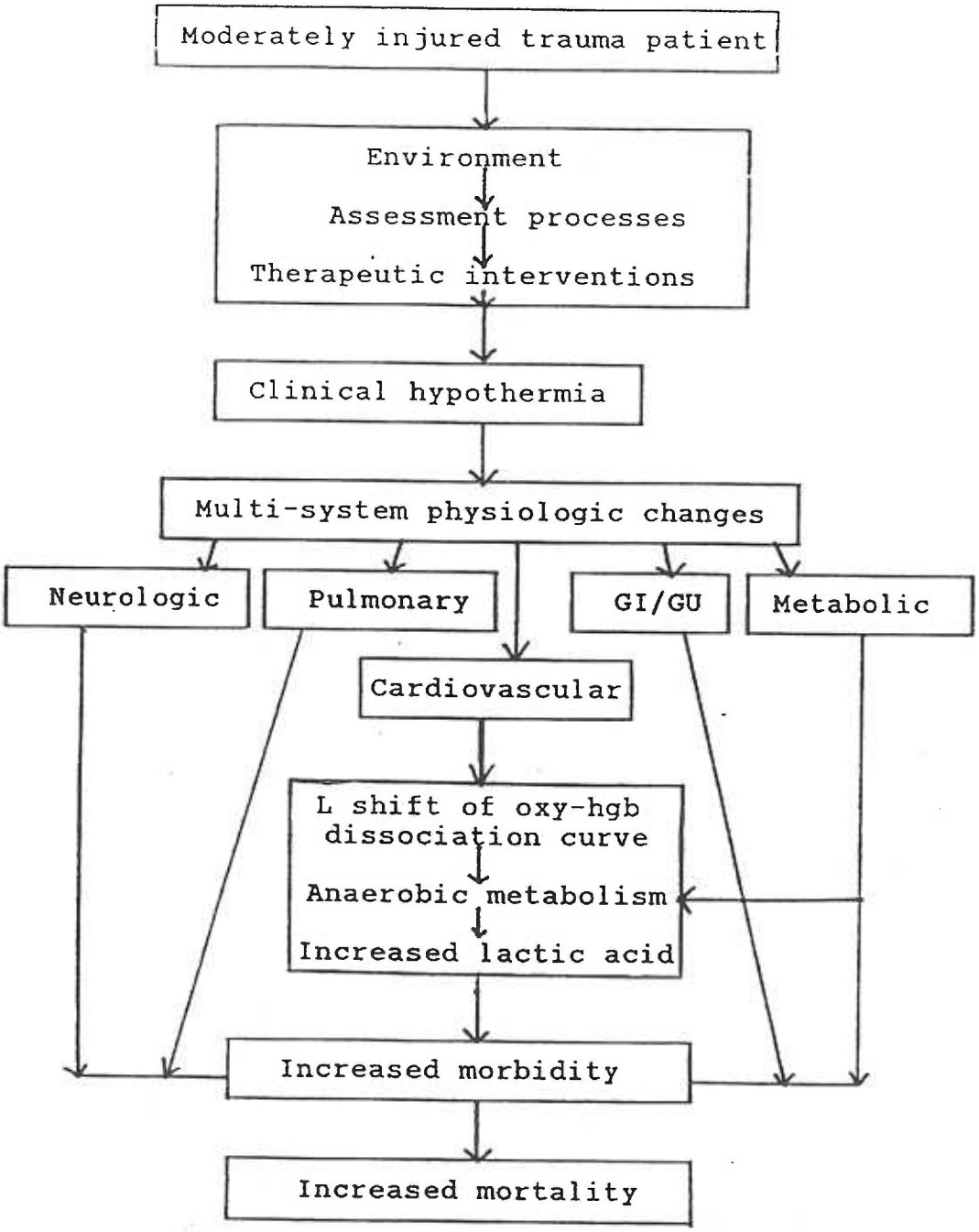
Conceptual Framework

Given the above review of the physiologic and surgical literature, the topic of clinical hypothermia in moderately injured trauma patients appears to be multi-faceted. The conceptual framework, therefore, is also intricate, as is illustrated on page 17. The following discussion describes the specific framework guiding this particular study. The dependent and independent variables and their relationships to each other are identified and operationally defined.

The moderately injured trauma patient may become hypothermic as a consequence of the environment at the time of injury, the assessment process, and the treatment of the injuries. Conduction, evaporation, radiation, and convection all contribute to the lowering of body temperature to 31-36 degrees centigrade (clincal hypothermia). When hypothermia is left untreated, physiologic changes occur throughout the major body systems that contribute to the morbidity and mortality of the patient.

All systems of the body are affected when the patient develops clinical hypothermia. Well-documented alterations of neurologic, pulmonary, gastrointestinal,

CONCEPTUAL FRAMEWORK



genitourinary, metabolic, and cardiovascular function occur as the body cools. The cardiovascular system becomes significantly compromised in the hypothermic patient in many ways. One of them is of particular concern: the development of a left shift of the oxygen-hemoglobin dissociation curve.

In the face of clinical hypothermia, the oxygen-hemoglobin dissociation curve shifts in such a way that the hemoglobin molecule increases its affinity for oxygen, and will not release it to the cells. This results in cellular hypoxia and anaerobic metabolism. An end-product of this metabolic pathway is lactic acid, and elevated levels of this organic acid indicate that generalized cellular compromise has occured in the patient as a direct consequence of hypothermia. This phenomenon, in combination with the other pathophysiologic changes occuring elsewhere in the body, contributes to the overall morbidity and mortality of the trauma patient.

This study focused specifically on the relationship between clinical hypothermia and lactic acid levels. It identified lactic acid as the dependent variable. The independent variable was body

temperature. The co-existing variable was the moderately injured trauma patient. Each of these variables was defined before their relationships to each other were described.

Lactic acid is the end product of insufficient cellular oxygenation resulting in a shift from aerobic to anaerobic metabolic pathways. This inefficent process leads to an accumulation of lactic acid that is released into the circulation (Guyton, 1981). The presence of elevated lactic acid levels implies that insufficient oxygenation has occurred, and a threat to patient survivability exists if the process is not reversed. Lactic acid levels of greater than 2.0 meq/L are considered to be elevated (normal range 0.5-1.5 meq/l) (Ragland, 1985).

Hypothermic body temperature is defined as a patient core temperature of 31-36 degrees centigrade. This definition is operationalized by measurement with a urinary bladder or a tympanic membrane temperature probe, as research suggests that these are appropriate sites for accurate core body temperature measurements in trauma patients (Benzinger, 1969; Mravinac, Dracup, & Clochesy, 1989; Nicholson & Iserson, 1991).

Core temperature refers to the temperature of the central circulation and reflects hypothalamic temperature (Rueler, 1978). Core temperatures can be measured at the tympanic membrane, urinary bladder, pulmonary artery, central vein, nasopharynx, esophagus, and rectum (Cork, Vaughan, & Humphrey, 1983). Urinary bladder sites are more reliable core measurements than rectal sites (Mravinac, Dracup, & Clochesy, 1989). Benzinger (1969) states that tympanic membrane measurements are the most indicative of true core temperature because they are taken at the site closest to the hypothalamus. Nicholson and Iserson (1991) agree that tympanic membrane measurements represent brain temperature. They believe that urinary bladder and tympanic measurements are the most appropriate sites for temperature measurement when resuscitating trauma patients.

Moderate injury is the co-existing variable, operationally defined using the physiological parameters of blood pressure, heart rate, and level of consciousness. The moderately injured trauma patient has injuries associated with moderate mortality levels. This patient is expected to survive, and interventions

are not extreme in nature. For the purpose of this study, moderate injury implied that no hypotension below a systolic pressure of 90mm Hg occurred, that minimal pulmonary or cardiac injury occurred to influence oxygen supply and delivery, and that any loss of consciousness was less than 5 minutes.

The traumatized patient who is moderately injured becomes hypothermic via convective, radiative, evaporative, and conductive heat loss. As a result the oxygen-hemoglobin dissociation curve shifts to the left preventing oxygen release to the cells for energy production. The cells convert to anaerobic metabolism, increasing the levels of lactic acid in the boodstream. The elevated lactic acid levels specifically due to hypothermia may contribute to the overall morbidity and mortality of the trauma patient.

The preceeding discussion of the interrelationships between the dependent and independent variables within the conceptual framework allows for the generation of a research question and identification of the purpose of the study.

Purpose of the Study

The purpose of this investigation was to determine if there was a correlation between lactic acid levels and body temperature in hypothermic, moderately injured trauma patients.

CHAPTER II

METHODS

Design

In order to describe the incidence and prevalence of lactic acid levels in moderately injured hypothermic patients, a correlative design was used. The study involved the prospective collection of quantitative data from adult trauma patients entering the Oregon Health Sciences University Hospital, a Level I Trauma Center in Portland, Oregon. This 360-bed teaching hospital receives 1100-1200 trauma patients per year. Its high volume and range of trauma patients provided an accessible sample for the target population.

Subjects

Following approval by the Institutional Review Board (IRB), a convenience sample of adult trauma patients admitted to the Emergency Department between February 8, 1993 and May 7, 1993 was used. The need for patient consent was waived by the IRB because the blood for lactic acid testing was collected simultaneously with labs drawn for the standard trauma admission protocol. No additional invasive procedure was performed for purposes of this study. Nine

subjects were included in this investigation. Inclusion criteria were: moderately injured trauma patients over the age of 18 with systolic blood pressures greater than 90mm Hg, heart rates greater than 60/minute but less than 160/minute, rousable levels of consciousness, and urinary bladder or tympanic membrane temperatures of 31-36 degrees centigrade within 15 minutes of admission.

Initially, categorization of patients into the moderately injured range was to be done using the Revised Trauma Score (RTS)(Appendix B). A highly reliable predictor of trauma patient mortality (97.2%), this physiologic scoring tool is used extensively in trauma research (Champion et al, 1989). For the purposes of this study, however, it did not serve as an effective screening tool for the identification of moderately injured subjects. Calculated RTS scores for patients who met the required blood pressure and heart rate parameters were too high to allow for subject entry into the study. In addition, integration of level of consciousness into the RTS score did not affect the score so that clinically appropriate patients met the entry criteria. Therefore, only the

physiologic parameters of blood pressure, heart rate, and level of consciousness were used as the screening parameters for subject entry. The RTS was not used as a screening measure but was calculated as a subject descriptor.

The patients' blood pressure, heart rate, and level of consciousness were measured by the Emergency Department (ED) nurses, who then entered patients into the study. Individual variances in professional skill posed a threat to validity due to interrater reliability. This threat, however, was minimized because the ED nurses were experienced critical care and emergency practitioners with special education in trauma care. Additionally, an inservice to the nursing staff at the beginning of the study described the physiologic parameters needed to enter a patient into this investigation. Initially, daily informal inservices were to done to familiarize the nurses with the study and inspire them to enter appropriate patients. Weekly rounds by the principal investigator reinforced the study protocol and prompted patient inclusion. The principal investigator, with 12 years of emergency, trauma and critical care experience,

retrospectively reviewed the physiological parameters for all patients before entering the subjects into the final data base.

The exclusion criteria excluded: 1) patients younger than 18 years of age, because of patient consent issues and differences in pediatric metabolic rates; 2) unconscious, posturing, or intoxicated patients with blood alcohol levels greater than 120 mg%, because of the potential influence of these clinical states on basal metabolic demands; 3) patients with evidence of significant cardiac or pulmonary injury or pre-existing cardiac or pulmonary disease, to eliminate those influences on ventilation and oxygen delivery; and 4) patients with systolic blood pressures less than 90 mm Hg, to prevent any influence of hypotension on lactic acid levels.

Data collection instruments

Body temperature was measured using either the urinary bladder or tympanic membrane route. The bladder temperatures were obtained using the Synergy Temperature Monitor (American Medical Systems, Division of American Hospital Supply Company; Cinncinati, Ohio). It has a range of 34-44 degrees centigrade with an

accuracy of +/- 0.2 degrees centigrade and resolution of .1 degrees centigrade (Appendix C). After replacement of a faulty connector cable, calibration of this monitor was performed by the principal investigator and a member of the hospital bioengineering department using the Fogg Temperature Probe Simulator (Fogg System Company; Denver, Colorado), and a warm water bath using a glass therometer guaranteed by the National Institute of Standards and Technology.

Bardex Lubricath Temperature Sensing Urotrack Plus Foley Catheters (C.R. Bard, Incorporated; Covington, Georgia) were used for the urinary bladder temperature measurements. Their temperature range is 7.9-43.0 degrees centigrade with an accuracy of +/- .1 degrees centigrade (Appendix C). Accuracy and consistency were tested on two randomly selected catheters connected to the Synergy Temperature Monitor and a water bath; differences of .1 degree centigrade with the first catheter and .4 degree centigrade with the second catheter were observed. In both instances the Synergic Temperature Monitor read higher than the water bath.

The tympanic membrane sites were measured using

FirstTemp devices (Intelligent Medical Systems; Carlsbad, California). These devices have a temperature range of 21.1-43.3 degrees centigrade, an accuracy of +/- 0.1 degrees centigrade, and a resolution of 0.1 degrees centigrade (Appendix C). Calibration of these devices performed by the product representative at the beginning and end of data collection demonstrated negligible drift in measurements (Appendix C).

Both the foley probe monitor and the tympanic membrane monitors were tested by the nursing staff using standardized procedures immediately prior to obtaining patient temperatures. All devices had met the hospital requirements for patient safety and accuracy within the calendar year.

Lactic acid levels were measured using the Beckman Synchron CX5 System (Diagnostic Systems Group; Brea, California). Its range is 0.2-11.0 mmol/L and it has a within-run precision of one standard deviation equal to 0.13 mmol/L (Appendix D). Calibration occurred once a shift by laboratory personnel according to department policy.

Data collection

Vital signs including blood pressures, pulses, respirations, level of consciousness assessments, and temperature measurements were done within 15 minutes of the trauma patient's admission by the ED nurse and recorded in the chart. Because this information was part of the patient's chart, confidentiality was maintained. Those patients who met the study criteria had an extra 5 cc of venous blood drawn, along with the standard trauma bloodwork, and placed on ice. This specimen was immediately hand-carried to the lab within 5 minutes of draw time, and analyzed within 10 minutes of arrival in the lab.

The steps of the research protocol were posted above the recording desk in the trauma resuscitation room and in the chemistry lab. They were entitled the "Clinical Hypothermia and Lactic Acid Level Research Protocol for Trauma Patients." They included the following:

Emergency Department Nurse:

1. The Emergency Department Nurse measures the patient's blood pressure, heart rate, and level of consciousness and records the

information on the trauma flow sheet.

2. The Emergency Department Nurse measures the patient's temperature using either the foley catheter temperature monitor or tympanic membrane thermometer after calibrating the equipment according to the standard procedure. The nurse sets the tympanic membrane probe in the "core temperature" mode. The temperature is measured and recorded within 15 minutes of the patient's admission.

3. The Emergency Department Nurse enters the patient into the study if the systolic blood pressure is greater than 90 mm hg, the heart rate is greater than 60/minute but less than 160/minute, the level of consciousness is rousable, the body temperature is between 32-36 degrees centigrade, and the patient is at least 18 years old.

4. The Emergency Department Nurse places 5cc of venous blood in a grey-top tube within 15 minutes of admission. The tube is placed on ice and sent to the lab with a transportation

aide within 5 minutes of the time drawn.

Laboratory Personnel:

1. The laboratory personnel analyze the lactic acid level within 10 minutes of specimen arrival time.
2. The laboratory personnel record the test results in the lab computer.

Analysis

The interval level data were analyzed using the Crunch Statistical Package, Version 4 (Crunch Software Corporation; Oakland, California). To describe the sample, frequency distributions were done on the dependent and independent variables for all 9 subjects. A two-tailed, pairwise deletion Pearson's *r* test was done on all variables to determine if any correlations were present. Because hypothermia and diastolic blood pressure were both noted to correlate strongly with lactic acid, Y on X regression scatterplots were done to illustrate those relationships. No tests were performed to determine the incidence of lactic acidosis in hypothermic elderly patients because only one subject was older than 65 years of age.

CHAPTER III

RESULTS

The sample size of 9 included 8 men and 1 woman from 18-77 years of age with a mean age of 35.5. Their admission temperatures ranged from 34.0-36.0 degrees centigrade with a mean of 35.2 degrees centigrade. Lactic acid levels ranged from 1.0-6.1 mmol/L with a mean of 3.32mmol/L. Diastolic blood pressures ranged from 64-106 mm Hg with a mean of 82 mm Hg. Data reflecting the independent variables are summarized in Table 1. Patient #3 was an outlier because he had the highest blood alchol level and was the only patient with a bladder temperature measurement (Appendix A). The diagnosed injuries were predominantly those of orthopedic fractures and simple closed head injuries. All subjects were considered "hemodynamically stable" in the Emergency Department and aggressive fluid resuscitation was not required for any patient.

A correlation of $-.81$ ($p < .007$) was noted between body temperature and lactic acid. The distribution along the Y on X regression line clearly demonstrated that those patients with the lowest body temperatures had the highest lactic acid levels (Figure 1).

Table 1

Frequency Distributions of the Study Variables

Variable	Mean	Range	Standard Deviation
Age (years)	35.5	59	21.20
Serum Lactate	3.32	5.1	1.99
Systolic Blood Pressure (mm Hg)	140	93	24.89
Diastolic Blood Pressure (in mm Hg)	82.4	42	15.87
Temperature (centigrade)	35.2	2.0	.86
Respiratory Rate	18.6	6.0	2.23
Glascow Coma Score (Normal = 15)	14.0	1.0	.33

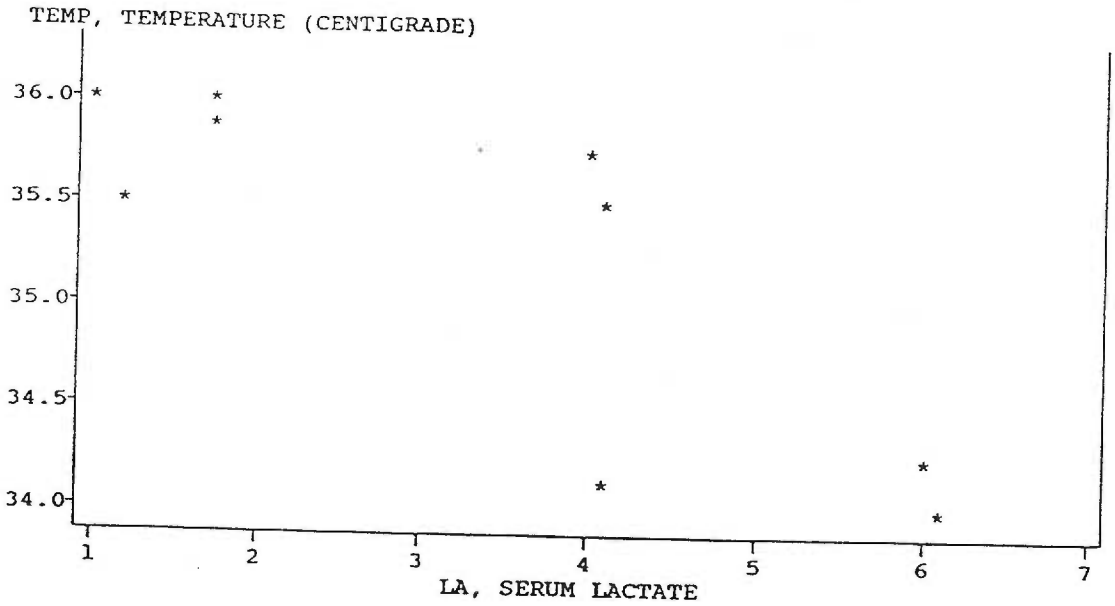


Figure 1. Regression scatterplot of hypothermia and serum lactate level.

An unexpected finding was the even stronger negative correlation between diastolic blood pressure and lactic acid levels ($r = -.89$, $p < .007$). The distribution of this correlation is illustrated in the scatterplot in Figure 2. An additional finding was that those patients with the lowest body temperatures also had the lowest diastolic blood pressures ($r = .78$). No correlations were noted between lactic acid and hematocrit, systolic blood pressure, or the Glascow coma score (Table 2).

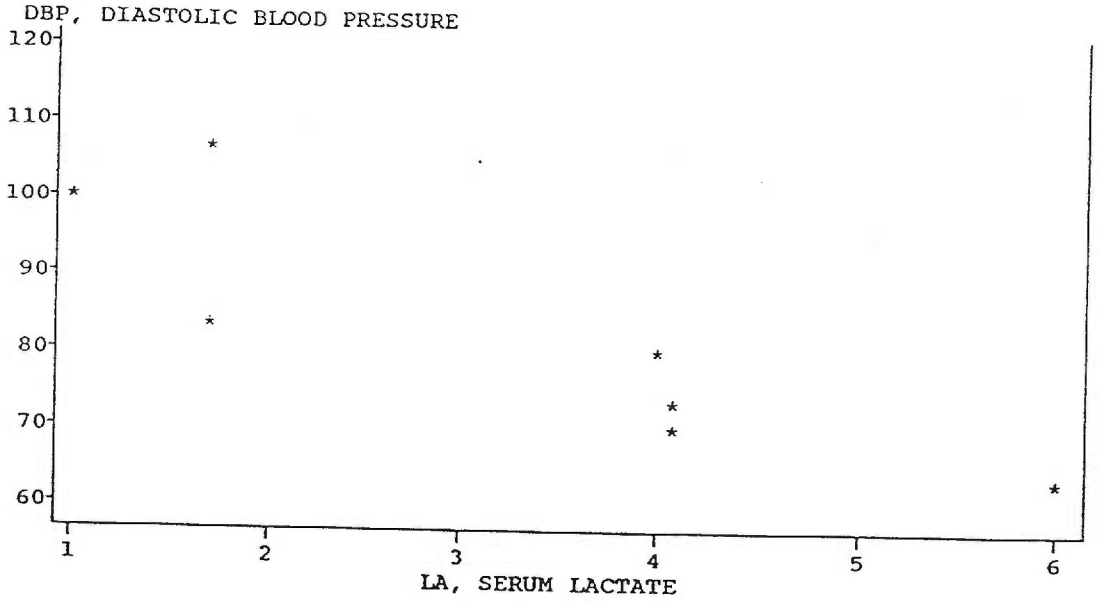


Figure 2. Regression scatterplot of diastolic blood pressure and serum lactate level.

Table 2

Multiple Correlations Between Variables Using the Two-tailed Pearson's *r* Statistical Test

	AGE	LA	SBP	DBP	HR	RR	GCS	TEMP	HCT
AGE	1.0000 0.0000 9	-0.3658 0.3329 9	0.7462 0.0209 9	0.4518 0.3089 7	0.1530 0.6944 9	-0.2092 0.5890 9	0.5032 0.1673 9	0.6007 0.0871 9	-0.3059 0.4234 9
LA	-0.3658 0.3329 9	1.0000 0.0000 9	-0.1430 0.7136 9	-0.8908 0.0071 7	-0.1488 0.7023 9	-0.5147 0.1562 9	-0.3255 0.3926 9	-0.8116 0.0079 9	-0.1650 0.6714 9
SBP	0.7462 0.0209 9	-0.1430 0.7136 9	1.0000 0.0000 9	0.6285 0.1306 7	-0.1096 0.7790 9	-0.3789 0.3146 9	0.1752 0.6520 9	0.3715 0.3249 9	-0.2121 0.5837 9
DBP	0.4518 0.3089 7	-0.8908 0.0071 7	0.6285 0.1306 7	1.0000 0.0000 7	-0.2127 0.6470 7	0.0992 0.8324 7	0.5260 0.2252 7	0.7770 0.0399 7	0.3419 0.4529 7
HR	0.1530 0.6944 9	-0.1488 0.7023 9	-0.1096 0.7790 9	-0.2127 0.6470 7	1.0000 0.0000 9	-0.2924 0.4451 9	-0.2223 0.5654 9	0.3437 0.3651 9	-0.6933 0.0384 9
RR	-0.2092 0.5890 9	-0.5147 0.1562 9	-0.3789 0.3146 9	0.0992 0.8324 7	-0.2924 0.4451 9	1.0000 0.0000 9	-0.1581 0.6845 9	0.1857 0.6323 9	0.2973 0.4373 9
GCS	0.5032 0.1673 9	-0.3255 0.3926 9	0.1752 0.6520 9	0.5260 0.2252 7	-0.2223 0.5654 9	-0.1581 0.6845 9	1.0000 0.0000 9	0.2937 0.4431 9	0.4445 0.2307 9
TEMP	0.6007 0.0871 9	-0.8116 0.0079 9	0.3715 0.3249 9	0.7770 0.0399 7	0.3437 0.3651 9	0.1857 0.6323 9	0.2937 0.4431 9	1.0000 0.0000 9	-0.3128 0.4124 9
HCT	-0.3059 0.4234 9	-0.1650 0.6714 9	-0.2121 0.5837 9	0.3419 0.4529 7	-0.6933 0.0384 9	0.2973 0.4373 9	0.4445 0.2307 9	-0.3128 0.4124 9	1.0000 0.0000 9

CHAPTER IV

DISCUSSION

This investigation clearly demonstrated that elevated lactic acid levels were present in the hypothermic, moderately injured trauma patients included in the sample. Despite the small sample size, this fact is likely to be reflected in the general population because of the strength of the correlation ($r = -.81$) .

The relationship of this correlation is significant. The lowest body temperatures were associated with the highest lactic acid levels, suggesting that the more hypothermic patients had increasingly significant cellular compromise and hypoxia.

High lactic acid levels existed in stable patients despite acceptable systolic blood pressures, heart rates, levels of consciousness, hematocrits and respiratory rates. It is interesting to note that these typically desirable ranges for the variables did not prevent the development of increased lactic acid levels.

The strong inverse relationship between lactic

acid and diastolic blood pressure in hypothermic patients was an unexpected finding ($r = -.89$). One explanation for this may lie with the specific role of diastolic blood pressures in maintaining adequate systemic circulation. Systolic pressures merely reflect circulatory resistance; more importantly, diastolic pressures represent actual cardiac and systemic perfusion.

Compensatory mechanisms in response to hypothermia seen in trauma patients lead to vasoconstriction and vascular shunting to the major organs. Measured systolic pressures may be acceptable in a cold, hypovolemic patient because of this vasoconstriction. However, this resistance is not felt during diastole and diastolic pressure falls, thus demonstrating that actual hypoperfusion is present. When diastolic hypotension occurs in a hypothermic, hypovolemic trauma patient, general perfusion may already be compromised. This subsequent decrease in peripheral circulation during hypothermia could lead to cellular hypoxia, anaerobic metabolism, and the unavoidable buildup of serum lactate levels. For the hypothermic patient with diastolic hypotension, the presence of elevated lactic

acid levels could be a clinically ominous finding.

The results of this study demonstrated that lactic acid levels were increased in clinically stable patients with low body temperature. The results did not, however, identify the mechanism by which this phenomenon occurred. It may have been due to the left shift of the oxygen-hemoglobin dissociation curve during hypothermia. Or, it could have been due to the hypothermia-induced diastolic hypotension compromising perfusion and oxygen delivery to the cells. Further research is needed to determine if the lactic acid level elevations are caused by increased lactic acid production or decreased clearance from the circulation.

Limitations

There are several limitations to this study, the most significant of which is the small sample size. Although power analysis indicated that 67 patients were required for statistical significance, only 9 patients were entered into the investigation. Low numbers of eligible subjects were enrolled due to dependency by the principal investigator on the nursing staff to adhere strictly to the research protocols. This was

demonstrated in a number of situations.

First, retrospective chart review revealed that many appropriate patients were not entered into the study; this was most likely due to simple oversight by the nursing staff in a busy Emergency Department where patient care took priority over clinical research. Second, chart review also demonstrated instances where temperatures were not measured within designated time frames or were not taken at all; perhaps this was because temperature correction is not yet considered an immediate priority in the stabilization of critically injured patients. Third, the tympanic thermometers were missing for six days during a record cold weather spell when patients were highly likely to arrive hypothermic in the Emergency Department, thus preventing subject entries until the probes were located and recalibrated. Lastly, several patients initially entered into the study were subsequently dropped because their blood alcohol levels were too high, their head injuries were too severe, or they were less than 18 years of age.

Another limitation of the study was the physical instability of a blood sample being tested for lactic

acid. Metabolic changes occur immediately in the unoxygenated sample and are merely delayed when the specimen is placed on ice. If this was not done promptly after being drawn or transportation to the lab was delayed, the lactic acid levels could be elevated because of degradation of the sample, rather than hypothermia. Data from samples that were not on ice when they arrived in the lab were not included in the study.

An additional drawback to this study was the discovery of two alternative explanations for the presence of elevated lactic acid levels that were not controlled for. Extreme combativeness and agitation, common in intoxicated or head-injured patients, could lead to a build up of lactic acid levels as a result of profound, sustained muscle activity. Secondly, while compensatory vasoconstriction during trauma may prevent hypotension, circulatory shunting could still lead to hepatic hypoperfusion; one result of poor perfusion to this organ is decreased lactic acid clearance from the circulation (D.D. Trunkey, M.D., personal communication, March 2, 1993). This investigation did not control for the extremely combative patient or the

normotensive, but hypoperfused patient.

The study is also limited because there were no normothermic controls to determine whether lactic acidosis was present in all moderately injured patients or only in those who were hypothermic. Information regarding the incidence of elevated lactic levels in all moderately injured patients would give greater significance to its occurrence in hypothermic patients.

The final limitation of the study concerns the methodology for screening potential patients. The original intention to use the RTS as a screening tool proved faulty because tabulated patient scores consistently eliminated patients who physiologically met the inclusion criteria. Because of this problem, the nurses were re-educated to use hemodynamic and neurological parameters to enter patients into the study. In addition to these parameters, they were encouraged to use their professional nursing judgement to confirm their identification of the "moderately injured patient." While the specific physiologic parameters were effective screening guidelines, the opportunity to use arbitrary "intuitive sense" probably contributed to the inconsistent level of injury noted

in patients who were initially entered into the study but ultimately dropped.

Clinical Implications

There are multiple causes for lactic acid build up in any trauma patient: hypotension, hypoperfusion, agitation, hypovolemia, and respiratory arrest. This study demonstrated that hypothermia was an additional contributor to the total increase of lactic acid in moderately injured patients as they arrived in the Emergency Department. With the knowledge that low body temperature played a significant role in the development of cellular hypoxia, caregivers should now give priority to normalizing patient temperature.

This should begin early in the resuscitation phase of trauma care in the Emergency Department and continue until normothermia is achieved. The nurse who assesses the low body temperature should immediately alert the physician to the finding, and prompt measures to warm the patient as well as prevent additional heat loss should be initiated.

A further clinical implication of this study is that the trauma team should follow the diastolic blood

pressure as a more accurate indicator of tissue perfusion in the hypothermic, vasoconstricted patient. A drop in diastolic blood pressure in a cold trauma patient could signal the onset serious cellular hypoxia that may ultimately impact patient outcomes. Efforts to warm the patient and restore circulating blood volume should be aggressive.

Additionally, the presence of elevated lactic acid levels in patients who appeared clinically stable implies that blood pressure, respiratory rate, hematocrit and heart rate inadequately reflect the patient's oxygenation status at the cellular level. The addition of lactic acid blood tests to the standard trauma lab panel would allow more reliable evaluation of resuscitative efforts. Many east coast trauma centers use serial blood lactate levels as well as physiologic parameters to guide interventions aimed at correcting cellular compromise (Kathryn von Reuden, RN; personal communication, April, 1993). Careful attention in handling the specimen would be imperative, however, to minimize the sample degradation that results in falsely high lactate levels.

Recommendations for Future Study

Replication of this study is justified because an increase in the number of subjects would contribute significantly to the statistical power of the study. Additionally, reliability and validity issues would be minimized if a full-time investigator were present at all trauma admissions in the Emergency Department to assure strict adherence to study protocols. Use of normothermic controls would give insight into the presence and clinical significance of elevated lactic acid levels in moderately injured trauma patients. Studies to determine the temperature range at which lactic acid levels increase occurs and to clarify the mechanism of the increase would have obvious clinical significance.

Other questions needing answers include: Are morbidity and mortality rates of moderately injured patients influenced by cold-induced elevated lactic acid levels? Do increases in serum lactate levels occur because of increased production or because of decreased clearance from the circulation? How does pulmonary disease or injury affect the levels of lactic acid in the hypothermic patient? Are cold elderly

trauma patients more susceptible to elevations in lactic acid than younger trauma patients? And finally, do oxygen saturations accurately reflect lactic acid levels as an indicator of cellular hypoxia in hypothermic patients?

Summary

The role of hypothermia in trauma patients is an exciting new topic in need of thorough investigation. This study demonstrated that elevated lactic acids levels were present in moderately injured trauma patients who were hypothermic. This finding is significant to trauma nurses in terms of both assessment and intervention. Efforts to reverse hypothermia and prevent additional heat loss should occur simultaneously with the immediate resuscitation of the "ABCs" in all trauma patients.

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Appendix A

DATABASE

OBS	AGE	SEX	LA	SBP	DBP	HR	RR	GCS	RTS	TEMP	ETOH	HCT
1	18	M	6.0	137	64	130	16	13	11	34.2	0	34.7
2	49	M	4.1	142	72	102	20	14	12	35.5	0	34.0
3	18	M	4.1	120	70	74	22	14	12	34.1	95	46.5
4	32	M	1.7	145	106	99	20	14	12	36.0	0	40.8
5	58	M	4.0	150	80	117	16	15	12	35.8	0	36.0
6	29	M	1.2	100	.	123	20	15	12	35.5	0	43.7
7	77	F	1.0	193	101	98	18	15	12	36.0	0	42.1
8	21	M	6.1	140	.	72	16	15	12	34.0	29	47.0
9	18	M	1.7	137	84	100	20	14	12	35.9	0	41.0

OBS= observation
AGE= age in years
LA= lactic acid (mmol/l)
SBP= systolic BP (mm Hg)
DBP= diastolic BP (mm Hg)
HR= heart rate/min
. = palpable blood pressure

RR= respiratory rate/min
GCS= Glasgow Coma Score
RTS= Revised Trauma Score
TEMP= Temperature in degrees centigrade
ETOH= serum alcohol (mg%)
HCT= hematocrit (mg%)

Appendix B

Emanuel IV Antibiotic Guidelines for Trauma Patients

		Adult	Pediatrics
Severe trauma, shock, major operations	Claforan	2gm q8h x 24h	50mg/kg q8h x 24h
If perforated bowel treat for 5 days with	Claforan	2gm q8h	50mg/kg q8h
If open fractures treat for 5 days with	Ancef	2gm q8h	25mg/kg q8h
(See full prescribing information)	Gentamicin	2.5mg/kg q12h	2.5mg/kg q8h

Revised Trauma Score (RTS)

Respiratory Rate (RR)	10 - 29 min.	4
	> 29 min.	3
	6 - 9 min.	2
	1 - 5 min.	1
RR = ____	None	0
Systolic Blood Pressure (SBP)	> 89 mmHG	4
	76 - 89 mmHG	3
	50 - 75 mmHG	2
	1 - 49 mmHG	1
SBP = ____	No pulse	0

Glasgow Coma Scale (GCS)

Eyes Opening	Spontaneous	4
	To speech	3
	To pain	2
	None	1
Best Verbal	Oriented	5
	Confused	4
	Inappropriate words	3
	Incomprehensible sounds	2
	None	1
Best Motor	Obeys command	6
	Localizes pain	5
	Withdraws to pain	4
	Flexion to pain (decorticate)	3
	Extension to pain (decerebrate)	2
GCS = ____ (convert below)	None	1

Glasgow Coma Conversion Scale (GCCS)

13 - 15 = 4
9 - 12 = 3
6 - 8 = 2
4 - 5 = 1
3 = 0

GCCS = ____

RR ____ + SBP ____ + GCCS ____ = RTS ____

Appendix C

ACCURACIES FOR THE
SYNERGY TEMPERATURE MONITOR MODEL 672N310

Accuracy	+/- .2 'C or +/- .4 'F
Range	34-44 'C or 93-111 'F

ACCURACIES FOR THE FIRST-TEMP
TYMPANIC THERMOMETER MODEL 2000A

Accuracy	+/- .1 'C or +/- .2 'F
Range	21.1-43.3 'C or 60.0-110.0 'F

FIRST-TEMP TYMPANIC THERMOMETER
CALIBRATION

FEBRUARY 8, 1993
MODEL # 2000A, SERIAL #33258
CORE EQUIVALENCY

LOW ('C)	HIGH ('C)
98.2	102.1
98.2	102.1
98.2	102.1

MAY 1, 1993
MODEL #2000A, SERIAL #33258
CORE EQUIVALENCY

LOW ('C)	HIGH ('C)
98.3	101.9
98.3	101.9
98.2	101.9

BARD UROTRACK
FOLEY CATHETERS

ACCURACY	+/- .2 'C at 37 'C (Urotrack System) +/- .1 'C at 37 'C (Catheters only)
RANGE	7.9-43.0 'C

BARD UROTRACK FOLEY CATHETER
ACCURACY VERIFICATION

FOLEY #1

H_2O BATH	37.5 'C
SYNERGY MONITOR	37.6 'C
RANGE	0.1 'C

FOLEY #2

H_2O BATH	37.0 'C
SYNERGY MONITOR	37.4 'C
RANGE	0.4 'C

Synergy Temperature Monitor
Model #67N310 Serial #001002
Service date 9/92

Glass Mercury Therometer
Guaranteed by National Institute of
Standards and Technology

Appendix D

BECKMAN

Made in U.S.A.

SYNCHRON CX® SYSTEMS CHEMISTRY INFORMATION

LACTATE REAGENT (LAC)

KIT REORDER #445875

For In Vitro Diagnostic Use

INTENDED USE / METHODOLOGY

The Lactate Reagent in conjunction with SYNCHRON CX System MULTI ™ Calibrator is intended for the quantitative determination of lactate concentration in plasma on SYNCHRON CX Clinical Systems.

In the assay reaction, lactate oxidase converts lactate to pyruvate with the concomitant generation of hydrogen peroxide (H_2O_2). The H_2O_2 formed reacts with dichlorobenzenesulfonic acid (DCBSA) and 4-aminoantipyrine (4-AAP) in a reaction catalyzed by peroxidase to form a chromophore. The lactic acid concentration is determined by measuring the absorbance due to the chromophore using endpoint technique.

The SYNCHRON CX Clinical System automatically proportions the appropriate sample and reagent volumes into a cuvette. The ratio used is 1:110. The system monitors the change in absorbance at 520 nanometers for a fixed-time interval. This change in absorbance is directly proportional to the concentration of lactic acid in the sample and is used by the SYNCHRON CX Clinical System to calculate and express the lactate concentration.

CHEMICAL REACTION SCHEME

$$\text{L-Lactate} + O_2 \xrightarrow{\text{Lactate Oxidase}} \text{Pyruvate} + H_2O_2$$

$$H_2O_2 + \text{DCBSA} + \text{4-AAP} \xrightarrow{\text{Peroxidase}} \text{chromophore}$$

CONTENTS

Each kit contains the following items:

Two (2) Lactate reagent bottles	Lactate (LAC) Reagent (granular). Each 15 mL when reconstituted as directed.
Two (2) empty reagent cartridges	Lactate Reagent Cartridges (empty). Each contains a minimum of 35 tests when filled as directed.
One (1) Package Insert	

REACTIVE INGREDIENTS

CONSTITUENTS	CONCENTRATION
Lactate oxidase	700 U/L
Peroxidase	508 U/L
DCBSA	2.0 mmol/L
4-Aminoantipryrine	1.16 mmol/L

Non-reactive chemicals necessary for optimum system operation.

REAGENT PREPARATION

CAUTION

Due to the presence of lactate in skin, avoid contact with reagent.

1. Add 15.0 mL of distilled or deionized water to one of the Reagent vials.
2. Mix vial until contents are dissolved.
3. Let stand ten (10) minutes at room temperature.
4. Transfer all of the reconstituted Reagent to compartment B (middle) of the reagent cartridge.
5. Once in the cartridge, the reagents are stable for 14 days at +2°C to +8°C unless the expiration date is exceeded.
6. When needed, repeat steps 1 and 2 to prepare second cartridge.

NOTE

Contamination of reagent must be avoided. The LAC Reagent solution should appear colorless to very pale pink. A deep purple color indicates that the reagent has been contaminated.

STORAGE AND STABILITY

The Lactate Reagent when stored unopened at +2°C to +8°C will remain stable until the indicated expiration date stamped on the label. Once in the cartridge, the reagent is stable for 14 days at +2°C to +8°C (unless the expiration date is exceeded). DO NOT FREEZE.

SPECIMEN COLLECTION, PREPARATION, AND STORAGE

Biological fluid samples should be collected in the same manner normally used for any laboratory test.[1] Freshly drawn plasma collected in a tube containing sodium fluoride and potassium oxalate (10 mg of each anticoagulant per 5 ml of blood) is the specimen of choice. As soon as possible after collection, preferably within 15 minutes, centrifuge the sample and remove from the cells. Blood should be drawn without stasis because venous stasis may cause lactate elevation.[2] Perform the analysis on red cell free plasma. Lactate in separated plasma is stable up to 2 hours at room temperature or two days when stored at +2°C to +8°C. Serum and urine are not recommended for use as samples. Only plastic or borosilicate glass containers should be used to store samples.

LIMITATIONS

1. Only plasma obtained using sodium fluoride/potassium oxalate collection tubes is suitable for use with the Lactate Reagent.

   Based on a study of samples obtained from 20 healthy volunteers, the following anticoagulants were found to be incompatible with this method:

ANTICOAGULANT	LEVEL TESTED FOR IN VITRO INTERFERENCE	PLASMA-SERUM BIAS (mg/dL) 37°C	30°C
Ammonium Heparin	29 units/mL	≤ +0.7	≤ + 0.6
Lithium Heparin	29 units/mL	≤ +0.9	≤ + 0.9
Sodium Heparin	29 units/mL	≤ +0.9	≤ + 1.0
EDTA	3.0 mg/mL	≤ ±0.8	≤ ± 0.8
Sodium Citrate	1.7 mg/mL	≤ −0.9	≤ − 0.8

2. Sample values greater than 11.0 mmol/L (99.0 mg/dL) should be diluted with saline and reanalyzed.

3. Sample results which are below the analytical range lower limit of ~~0.3~~ 0.2 mmol/L (2.7 mg/dL) should be reported as "< ~~0.3~~ 0.2 mmol/L" ("< 2.7 mg/dL"). 9-10-92 cw

INTERFERENCES

1. The following substances were tested with this methodology for interference. The results are listed below:

SUBSTANCE	SOURCE	LEVEL	OBSERVED EFFECT*
Hemoglobin	RBC Hemolysate	375 mg/dL (3+)	+ 0.2 mmol/L or + 4.8%
Bilirubin	Bovine	9 mg/dL	− 0.2 mmol/L or −4.8%
Lipemia	Intralipid®**	2+	+ 0.2 mmol/L or ± 4.8%
Ascorbic Acid	NA	3 mg/dL	≤ ± 0.2 mmol/L or ± 4.8%
Pyruvate	NA	6 mg/dL	≤ ± 0.2 mmol/L or ± 4.8%
Lactate Dehydrogenase	NA	2000 IU/L	≤ ± 0.2 mmol/L or ± 4.8%

* Plus (+) or minus (−) signs in this column signify positive or negative interference.
** Intralipid is a trademark of Kabivitrum, Inc.

2. Refer to Bibliography for other interferences caused by drugs.[3]

OPERATIONAL PROCEDURE

1. Prepare LAC cartridge as described above in the Reagent Preparation section.
2. Remove all the cartridge caps prior to installation of the reagent on the system.
3. Load the reagent as directed in the SYNCHRON CX Clinical System CX4CE and CX7 Operating Instructions Manual.
4. After reagent load is completed, calibration may be required (SYNCHRON CX System Multical P/N 442600). Refer to Operating Instructions Manual for details of the calibration procedure.
5. Program samples for analysis as directed in the Operating Instructions for sample programming.
6. After transfer of samples and controls to sample cups in the assigned sectors follow the protocols for system operation as directed in the Operating Instructions manual.

PERFORMANCE CHARACTERISTICS

Analytic Range

The SYNCHRON CX4CE and CX7 Systems method for the determination of lactate provides the following analytic range:

SAMPLE	CONVENTIONAL UNITS	S.I. UNITS
Plasma	0.3 - 11.1 mmol/L	2.7 - 99 mg/dL

Samples exceeding the high end of the analytic range should be diluted with saline and reanalyzed.

Reference Values

The values given as reference ranges[4] are intended to act only as a guide. No adjustments have been made for age, sex, or dietary differences. Each laboratory should establish a reference range based upon its patient population.

SAMPLE	CONVENTIONAL UNITS	S.I. UNITS
Plasma	0.5 - 2.2 mmol/L	4.5 - 19.8 mg/dL

Equivalency

Equivalency was assessed by correlation analysis of serum samples to accepted clinical methods.

37°C

Y (SYNCHRON CX4CE and CX7)	=	0.94X + 0.1
N	=	53
MEAN (SYNCHRON CX4CE and CX7)	=	3.8
MEAN (Du Pont ACA)	=	3.9
CORRELATION COEFFICIENT (r)	=	0.9986

30°C

Y (SYNCHRON CX4CE and CX7)	=	0.91X + 0.1
N	=	53
MEAN (SYNCHRON CX4CE and CX7)	=	3.8
MEAN (DuPont ACA)	=	3.9
CORRELATION COEFFICIENT (r)	=	0.9989

Precision

A SYNCHRON CX4CE or CX7 System will typically exhibit a within-run precision of one standard deviation (S.D.) equal to 1.2 mg/dL (0.13 mmol/L), or a 3.0% coefficient of variation (C.V.).

NOTE

These degrees of precision and equivalency were obtained in typical testing procedures on the SYNCHRON CX Systems and are not intended to represent performance specifications for this reagent.

CLINICAL SIGNIFICANCE

Measurements of lactic acid are useful in the diagnosis and treatment of lactic acidosis (abnormally high acidity of the blood). Typically, patients with lactic acidosis and congestive heart failure or severe anemia show poor tissue oxygenation (hypoxia). Patients with lactic acidosis and diabetes mellitus, renal failure or liver disease usually do not show evidence of tissue hypoxia.

ADDITIONAL INFORMATION

For more detailed information on the operation of SYNCHRON CX Clincial Systems, refer to the Beckman SYNCHRON Clinical Systems CX4CE and CX7 Operating Instructions manual. Copies of this manual are available for Beckman Instruments, Inc., Brea, CA 92621-6209.

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ABSTRACT

AN EXPLORATION OF THE CORRELATION BETWEEN LACTIC ACID LEVELS AND BODY TEMPERATURE IN HYPOTHERMIC, MODERATELY INJURED TRAUMA PATIENTS

Christine L. Smith

The purpose of this correlational study was to determine if a relationship between lactic acid levels and body temperature existed in hypothermic, moderately injured trauma patients. A convenience sample of 9 moderately injured trauma patients entering the Emergency Department of a Level I Trauma Center in a university hospital was used. Subject entry criteria included patients with core body temperatures of 31-36 degrees centigrade, systolic blood pressures greater than 90 mm Hg, heart rates greater than 60/min but less than 160/min, and rousable levels of consciousness at the time of admission. Five cc of venous blood was sent on ice to the laboratory for analysis within 15 minutes of draw time.

Frequency distributions were performed for all variables. Pearson's *r* was used to determine the relationship of hypothermia and serum lactic acid ($r = -.81$) and diastolic blood pressure and serum lactic acid ($r = -.89$).

The study has significant clinical implications. Because it clearly demonstrated a strong correlation between hypothermia and lactic acid elevation, nurses should be quick to assess patients for lowered body temperature and integrate rewarming interventions into their resuscitative care in the Emergency Department. In addition, because of the correlation between diastolic blood pressure and serum lactate levels, a falling diasystolic pressure may well indicate profound visceral hypoperfusion in a cold trauma patients necessitating aggressive temperature correction and volume replacement.