PRACTICE GUIDELINES

Effective Date: **2/18/2011** Manual Reference: **Deaconess Trauma Services**

TITLE: GUIDELINES FOR MONITORING BASE DEFICIT TO DETERMINE SEVERITY OF SHOCK STATE IN RESUSCITATION OF TRAUMA PATIENTS

PURPOSE: The initial step in the metabolism of carbohydrate is the conversion of stored glycogen to pyruvate which can be accomplished without oxygen and is anaerobic. All carbohydrate metabolism after pyruvate requires oxygen. In the absence of oxygen, pyruvate accumulates and instantly is converted to lactate or lactic acid. Trauma with blood loss creates hypovolemia and hypoperfusion. Hypoperfusion results in an oxygen deficit, anaerobic metabolism and the accumulation of lactic acid. The acid load causes a base deficit in order to keep pH within normal range. Thus the accumulated lactic acid and the resultant base deficit reflect hypoperfusion, and several studies have documented the ability of these metabolic markers to predict outcome in trauma. These two parameters, blood lactate and base deficit reflect hypoperfusion, metabolic acidosis, and oxygen debt. The amount of base required to return pH to normal is the base deficit.

PATHOPHYSIOLOGY:

I. Metabolic acidosis is a pH abnormality in which the body has accumulated acid and does not have enough base to effectively neutralize the acid.
   A. Metabolic acidosis is classified according to the anion gap
      1. The anion gap is determined by the difference between routinely measured concentration of serum cations sodium and potassium (Na^+ and K^+) and the routinely measured concentration of anions chloride and bicarbonate (Cl^- and HCO_3^- [measured as CO_2])
      2. In most laboratories the range is 12 ± 4mEq/L if potassium is considered, and 8 ± 4 mEq/L if potassium is not considered (potassium is sometimes not considered because of the small amount involved)
      3. There are two types of metabolic acidosis: Normal-anion gap metabolic acidosis and positive-anion gap acidosis
   B. Normal-anion gap metabolic acidosis the causes of which include:
      1. Renal tubular acidosis (poor urinary acidification in otherwise well-functioning kidneys).
      2. Gastrointestinal condition
         a. Diarrhea
         b. Pancreatic or small bowel drainage
      3. Iatrogenic
         a. Parenteral nutrition
         b. Saline administration

C. Positive-anion gap metabolic acidosis the usual cause of which is the presence of excess acid with a resultant low bicarbonate level:
    1. Lactic acidosis (including hypovolemia, tissue ischemia, and hypoperfusion)
    2. Ketoacidosis
    3. Poisoning (included here are alcohol, cocaine, methamphetamine, phencyclidine, epinephrine, metformin)
    4. Renal failure
    5. Sepsis

II. Base deficit occurs in the presence of excess acid and can be quantified by bicarbonate level, lactic acid level, and base deficit
    A. Bicarbonate and base deficit may be used to calculate the degree of excess acid, metabolic acidosis
        1. Bicarbonate from electrolytes (measured as CO_2): venous blood is an inferior evaluation of systemic circulation
        2. Bicarbonate from arterial blood gases: the result is a nomographic calculation (not a measurement)
    B. Blood lactate levels are helpful if the excess acid is lactic acid
        1. Blood lactate levels are measured as lactic acid
        2. Arterial blood reflects systemic circulation
        3. Venous blood lactate is an inferior evaluation of systemic condition
    C. Base deficit is routinely calculated by arterial blood gas analysis

III. The value of base deficit
    A. Consider these
        1. Class III: Lactate and base deficit are independent predictors of mortality, ICU and Hospital length of stay.[1-2]
        2. Base deficit correction reflects increasing oxygen delivery and is most useful predicting physiologic normalization in patients with sufficient reserve to respond to resuscitation.
        3. Class II-III: Several studies have correlated the base deficit with survival, development of organ failure, and the need for blood transfusion in trauma patients.[3-7]
        4. Class II-III: Base deficit can be stratified into mild (3- 5), moderate (6- 14) and severe (> 14mMol/L) and these values correlate with resuscitation requirements and mortality.[3-4]
        5. Patients with markedly severe base deficit after traumatic injury can have a reasonable survival rate if they can survive the first several hours after injury. [8]
        6. Magnitude of base deficit does not equate to therapeutic futility
        7. Class III: Minor abnormalities in base deficit may still be associated with significant injuries.[9]
        8. Class III: Failure to normalize the base deficit with ongoing resuscitation is associated with greater mortality, increased risk of multiple organ failure, and the potential for ongoing undetected blood loss.[4,10]
        9. Class II: Factors that can influence base deficit besides metabolic acidosis

from shock include hypothermia, alcohol, drugs (bicarbonate rich or chloride rich resuscitation fluid), epinephrine and hypoperfusion (cardiomyopathy).[11]

10. Class III: Base deficit continues to be widely used to stratify severity of illness after injury.[12]
11. Serum bicarbonate has been postulated to reflect base deficit and to be easier to obtain.[13,14]

B. The hypoperfusion and oxygen debt of hypovolemia create a base deficit indicating an accumulation of an acid, in this instance lactic acid. The accumulation of lactic acid is an increased anion gap and the type of metabolic acidosis an increased anion gap metabolic acidosis with a base deficit.

GUIDELINES:

A. Initial base deficit levels and time to normalization of these levels correlate well with need for transfusion and risk of MODS and death. Persistently high or worsening base deficit levels may be an early indicator of complications, e.g., ongoing hemorrhage or the abdominal compartment syndrome (altered preload with low cardiac output). There are currently no data to suggest using base deficit as an endpoint for resuscitation improves survival.[4,12]

B. Use of base deficit requires:
   1. With metabolic acidosis we need more than blood pressure, pulse, urine output
   2. Base deficit -4 or greater requires attention
      a. Arterial blood gases every one to two hours until BD cleared or adequately resuscitated.
      b. Consider A-line, CVP, pulmonary artery catheter
      c. Correct hypoxemia, hypovolemia, ongoing bleeding
      d. Cardiomyopathy may interfere with correction of the base deficit
      e. This is an emergency over the age of 65
      f. During resuscitation oxygen delivery should be increased to normalize base deficit and lactate during the first 24 hours. The optimal algorithms for fluid resuscitation, blood product replacement, and the use of inotropes and/or vasopressors had not been determined.

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REVIEWED DATE	REVISED DATE
FEB 2011	
AUG 2014	
AUG 2016	
JAN 2017	
JAN 2018	
JAN 2019	
AUG 2020	
JUNE 2021	