# Week 2: Team C Template

 Mr. K.B. is age 81 and has had gastritis with severe vomiting for 3 days. He has a history of heart problems and is presently feeling dizzy and lethargic. His eyes appear sunken, his mouth is dry, he walks unsteadily, and he complains of muscle aching, particularly in the abdomen. He is thirsty but is unable to retain food or fluid. A neighbor has brought Mr. K.B. to the hospital, where examination shows that his blood pressure is low, and his pulse and respirations are rapid. Laboratory tests demonstrate elevated hematocrit, hypernatremia, decreased serum bicarbonate, serum pH 7.35, and urine of high specific gravity (highly concentrated).

*Part 1: Day 1 – Early Stage*

Initially, Mr. K.B. lost water, sodium in the mucus content, and hydrogen and chloride ions in the hydrochloric acid portion of the gastric secretions. Alkalosis develops for two reasons, the first being the direct loss of hydrogen ions and the second being the effects of chloride ion loss. When chloride ion is lost in the gastric secretions, it is replaced by chloride from the serum (see Fig. 2.9 in your text). To maintain equal numbers of cations and anions in the serum, chloride ion and bicarbonate ion can exchange places when needed. Therefore, more bicarbonate ions shift into the serum from storage sites in the erythrocytes to replace the lost chloride ions. More bicarbonate ions in the serum raise serum pH, and the result is hyperchloremic alkalosis.

*Part 2: Days 2 to 3 – Middle Stage*

As Mr. K.B. continues to vomit and is still unable to eat or drink any significant amounts, loss of the duodenal contents, which include intestinal, pancreatic, and biliary secretions, occurs. No digestion and absorption of any nutrients occurs. Losses at this stage include water, sodium ions, potassium ions, and bicarbonate ions. Also, intake of glucose and other nutrients is minimal. Mr. K.B. shows elevated serum sodium levels.

**Team C**

*Part 3: Day 3: Admission to the Hospital – Advanced Stage*

After a prolonged period of vomiting, metabolic acidosis develops. This change results from a number of factors:

* Loss of bicarbonate ions in duodenal secretions.
* Lack of nutrients leading to catabolism of stored fats and protein with production of excessive amounts of ketoacids.
* Dehydration and decreased blood volume leading to decreased excretion of acids by the kidney.
* Decreased blood volume leading to decreased tissue perfusion, less oxygen to cells, and increased anaerobic metabolism with increased lactic acid.
* Increased muscle activity and stress leading to increased metabolic acid production.

These factors lead to an increased amount of acids in the blood, which bind with bicarbonate buffer and result in decreased serum bicarbonate and decreased serum pH or metabolic acidosis.

1. List several reasons why Mr. K.B. is lethargic and weak.
2. Predict the serum level of carbon dioxide or carbonic acid in this case. Explain your prediction.
3. If Mr. K.B. continues to lose body fluid, why might serum pH decrease below 7.35?
4. If serum pH drops below 7.35, what signs would be observed in Mr. K.B.?
5. If serum pH drops below 7.35, would this be considered compensated or decompensated? Explain the pathophysiology that contributes to this?
6. What are the very slow, shallow respirations that occur with metabolic acidosis called? How are they likely to affect the following?
	1. PCO2
	2. Serum pH
7. Describe the effect of acidosis on serum potassium levels.
8. Mr. K.B. will be given replacement fluid therapy. Why is it important that sodium and potassium be given as well as water?