

AN ABSTRACT OF THE THESIS OF

Max Waibler for the degree of Master of Science in
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Title : The Effects of Carbonated Beverages on Arterial Oxygen
Saturation, Serum Hemoglobin Concentration and Maximal Oxygen
Consumption

Abstract approved : *Redacted for Privacy*
Dr. Anthony R. Wilcox

Elite milers, Sir Roger Bannister and Joseph Falcon, have stated that the consumption of carbonated beverages hinders the performance of aerobic events. Oxygen transport is purportedly impaired by the consumption of carbonated beverages. The research on carbonated beverages has been limited to the effects on the digestive system, gastric emptying, and thermal heat stress in animals. The purpose of this study was to investigate the effects of consuming 28 ounces of carbonated beverages per day, for three weeks, on arterial oxygen saturation (SaO_2), serum hemoglobin concentrations (Hb), and maximal oxygen consumption (VO_{2max}) in experienced cyclists.

Nine competitive cyclists and triathletes (aged 19-24 years, $M = 21.67$ years), with average weights and percent body fat of 76.51 kg and 11.4 percent respectively, were randomly assigned to a three week period of consuming 28 ounces of carbonated water or a three week period of no carbonated beverages. At the end of each three week period, a 5 c.c. blood sample was taken for Hb determination and the subjects performed a test of maximal oxygen consumption on a cycle ergometer while SaO_2 was being monitored. The groups then crossed-over with respect to their treatment, and after another three week period, the same variables were measured.

The Student's t - statistic was used to compare SaO_2 , Hb, and VO_{2max} . The results showed no significant differences between the carbonated period (C) and the noncarbonated period (NC) in SaO_2 (94.00 vs 93.22 %, $p = 0.21$), Hb (13.71 vs 14.12 g/dl, $p = 0.11$), and VO_{2max} (4.63 vs 4.65 l·min, $p = 0.92$). From this study, it appears that the consumption of carbonated beverages does not

affect the variables associated with the oxygen carrying capacity of blood (SaO₂ and Hb) or the test of aerobic performance ($\dot{V}O_{2max}$).

The Effects of Carbonated Beverages on Arterial Oxygen Saturation, Serum
Hemoglobin Concentration and Maximal Oxygen Consumption

by

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The Effects of Carbonated Beverages on Arterial Oxygen Saturation, Serum Hemoglobin Concentration, and Maximal Oxygen Consumption

CHAPTER I

INTRODUCTION

Carbonated cola-type drinks are the most commonly ingested beverages in the United States (Bellet, 1968). Many athletes have the impression that carbonated beverages will detrimentally affect high intensity, aerobic performance. The famed milers, Roger Bannister and Joe Falcon, have been quoted in national newspapers and on television promoting this notion.

The purpose of this study is to investigate the claim that carbonated beverages impair oxygen transport and thereby affect maximal oxygen consumption. This will be accomplished by analyzing the affect that carbonated beverages have on maximal oxygen consumption, arterial oxygen saturation, and serum hemoglobin levels. Considering that there is no research which directly addresses this aspect of carbonated beverage consumption, the research question will be presented following a discussion of the transport of blood gases and the limited research on carbonated beverages.

Oxygen Transport Factors

Oxygen is transported in the blood by two mechanisms: a) in physical solution dissolved in the plasma portion of the blood, and b) bound loosely with hemoglobin, an iron-protein molecule in the red blood cell. Oxygen is relatively insoluble in blood plasma, which carries only about 3 ml of oxygen per liter compared to hemoglobin which captures 197 ml of oxygen per liter of blood at an alveolar partial pressure of oxygen (PO_2) of 100 mmHg (McArdle et al., 1991).

The oxygen carrying capacity of blood ($ml \cdot 100 ml^{-1} blood$) is dependant upon the quantity of hemoglobin ($grams \cdot 100 ml^{-1}$) and the oxygen binding capacity of hemoglobin ($ml O_2 \cdot g^{-1}$) (McArdle et al., 1991). In each 100 ml of blood, men have 15 to 16 grams of hemoglobin and women have 13-14 grams, and each gram of hemoglobin can combine with 1.34 ml of oxygen (1.34 ml

$\text{O}_2/\text{g Hb}$) (Brooks & Fahey, 1984). The higher $\dot{V}\text{O}_{2\text{max}}$ values seen in men is commonly attributed in part, to the difference in hemoglobin concentrations between the two sexes (McArdle et al., 1991). Oxygen delivery during exercise could be impaired by either a reduction in hemoglobin concentration or by a reduced oxygen saturation of hemoglobin.

The amount of oxyhemoglobin (HbO_2) depends on the partial pressure of oxygen (PO_2) (Stegemann, 1981). The oxyhemoglobin dissociation curve describes the relationship between PO_2 and the oxygen content of blood (Figure 1). At rest, the alveolar-capillary PO_2 is 100 mmHg and hemoglobin is 98 percent saturated with oxygen (McArdle et al., 1991). The PO_2 in the skeletal muscle tissue at rest is approximately 40 mmHg, and, as a result, hemoglobin in arterial blood releases it hold on oxygen to the extent that it is only about 75 percent saturated. However, during intense exercise, the PO_2 in active muscle tissue can fall as low as 3 mmHg and nearly all of the oxygen is released from the arterial blood (Brooks & Fahey, 1984).

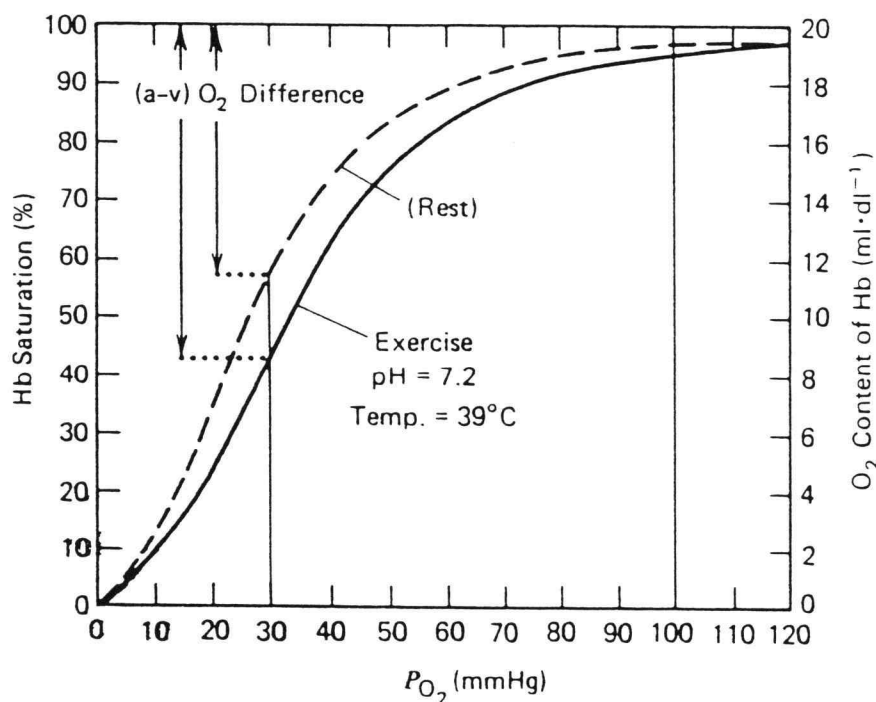


Figure 1 - Standard oxyhemoglobin dissociation curve. From Exercise Physiology by G.A. Brooks & T.D. Fahey. Copyright ©1984 by Macmillan Publishing company. Reprinted by permission of the publisher.

Exercise usually causes: a) an increase in acidity, from either carbonic acid (H_2CO_3) and/or lactic acid, which lowers the pH (Figure 2), b) increases in temperature (Figure 3), and c) in the partial pressure of CO_2 (PCO_2) in arterial blood (Figure 4), all of which cause the oxyhemoglobin dissociation curve to shift to the right (Astrand & Rodahl, 1986).

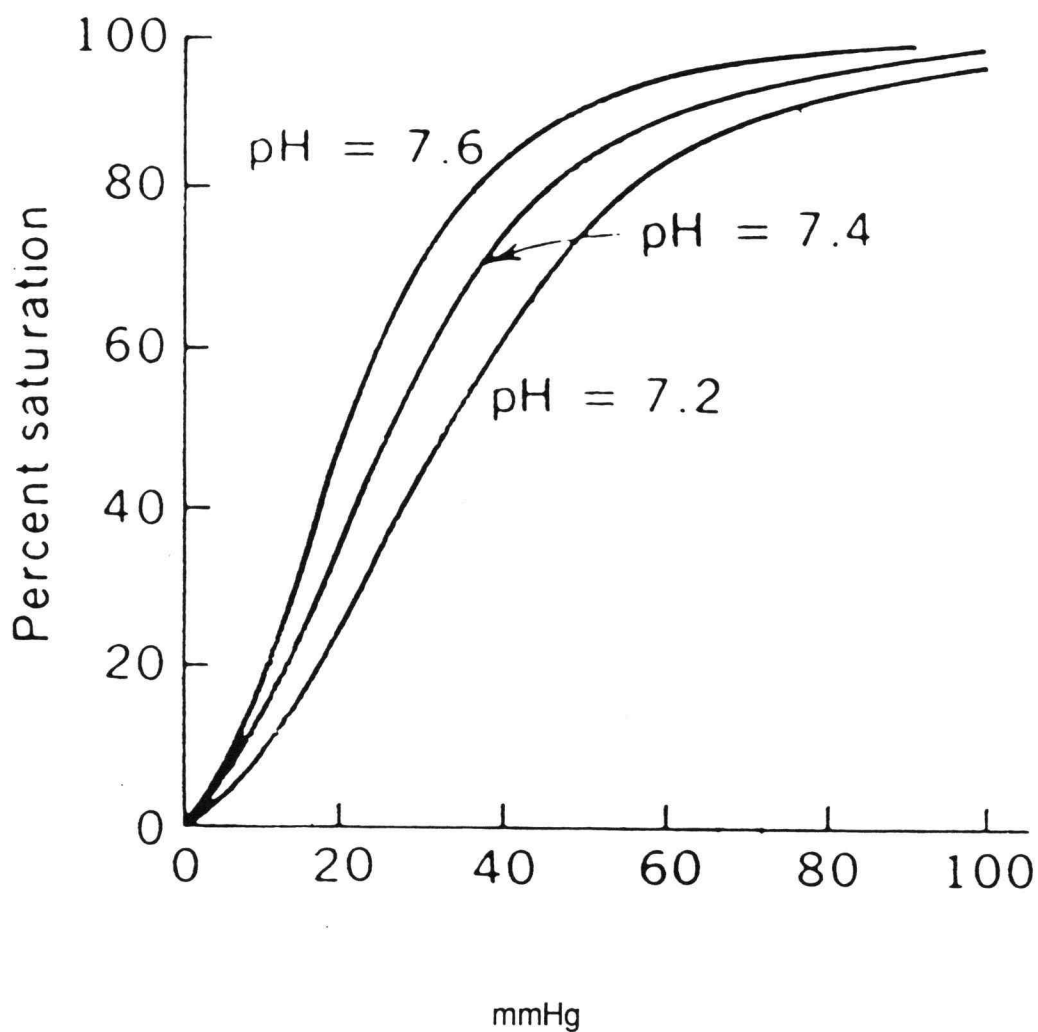


Figure 2 - The O_2 dissociation curve and the effect of different pH levels. From Text book of Work Physiology, 3E by P. Astrand & K. Rodahl. Copyright ©1986 by McGraw-Hill Publishing company. Reprinted by permission of the publisher.

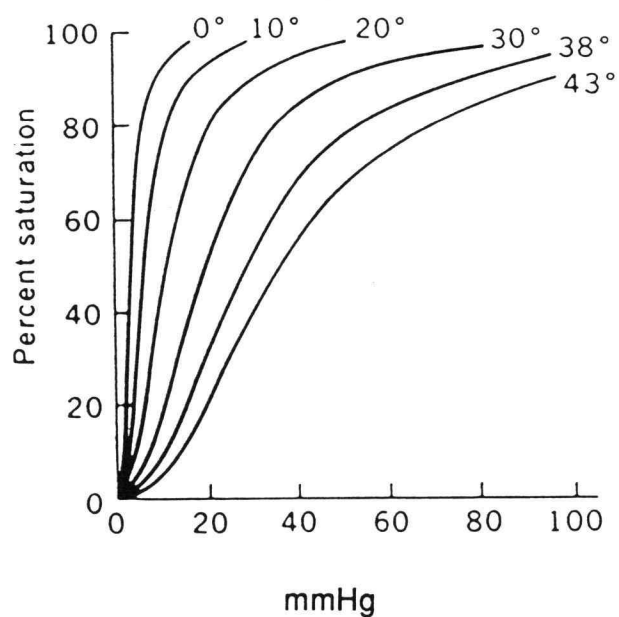


Figure 3 - The O_2 dissociation curve and the effect of various temperatures. From Text book of Work Physiology, 3E by P. Astrand & K. Rodahl. Copyright ©1986 by McGraw-Hill Publishing company. Reprinted by permission of the publisher.

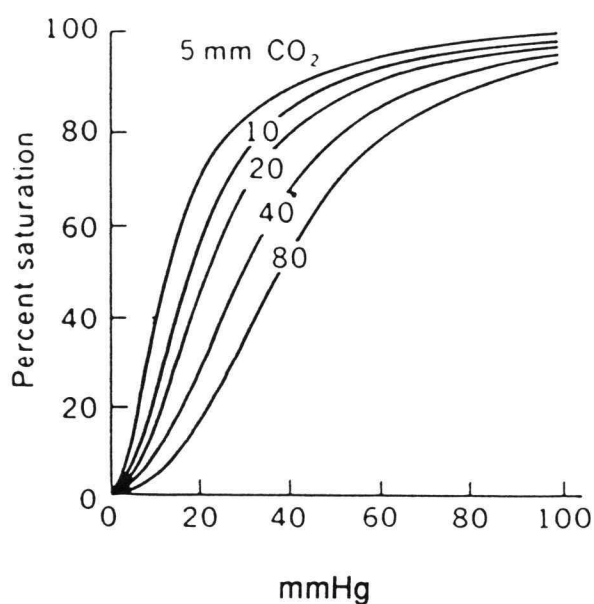


Figure 4 - The O_2 dissociation curve and the effect of PCO_2 . From Textbook of Work Physiology, 3E by P. Astrand & K. Rodahl. Copyright ©1986 by McGraw-Hill Publishing company. Reprinted permission of the publisher.

This shift of the dissociation curve is called the Bohr effect and allows for more oxygen to be released to the tissues during vigorous exercise (Adams, 1983). Conceivably, the CO_2 injected into beverages could increase the PCO_2 and the acidity of the arterial blood and cause the dissociation curve to shift to the right. If this were to occur at the level of the lungs, the oxygen saturation of hemoglobin in the arterial blood could be reduced. However, at the tissue level, a further shift in the dissociation curve would allow more oxygen to be released from hemoglobin. Considering that the PO_2 in arterial blood is only 3 mmHg during maximal exercise, a further shift in the dissociation curve would be of little advantage.

Carbon Dioxide Transport

Carbon dioxide is the end product of aerobic metabolism and is transported in the blood by three mechanisms (figure 5): a) 10 percent is diffused across the cell membrane into the tissue fluid and then across the capillary wall and carried as free carbon dioxide in the blood plasma, b) 70 percent is transported in chemical combination with water to form carbonic acid (H_2CO_3), and c) 20 percent combines with blood proteins to form carbaminohemoglobin (McArdle et al., 1991).

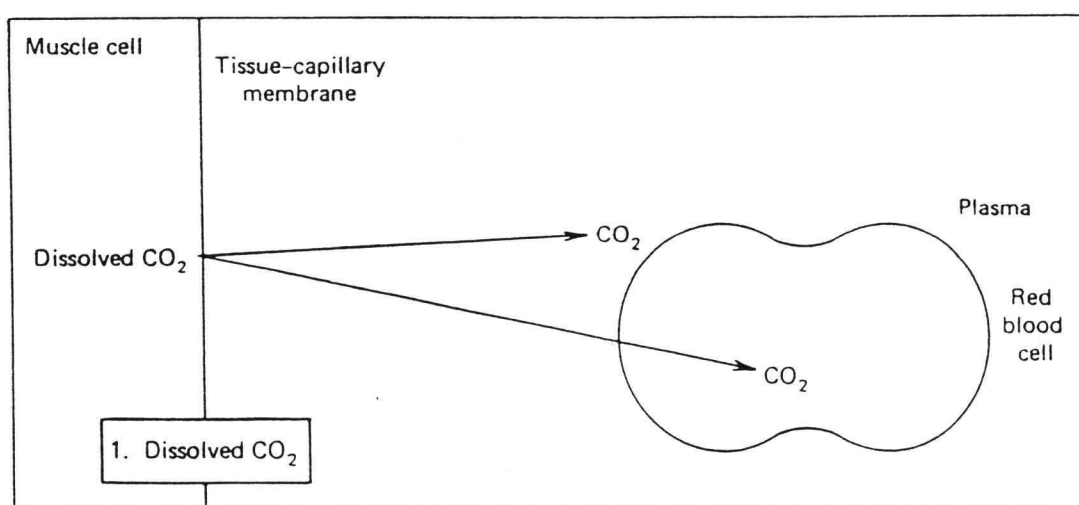


Figure 5 - Schematic representation of the transport mechanisms of CO_2 .
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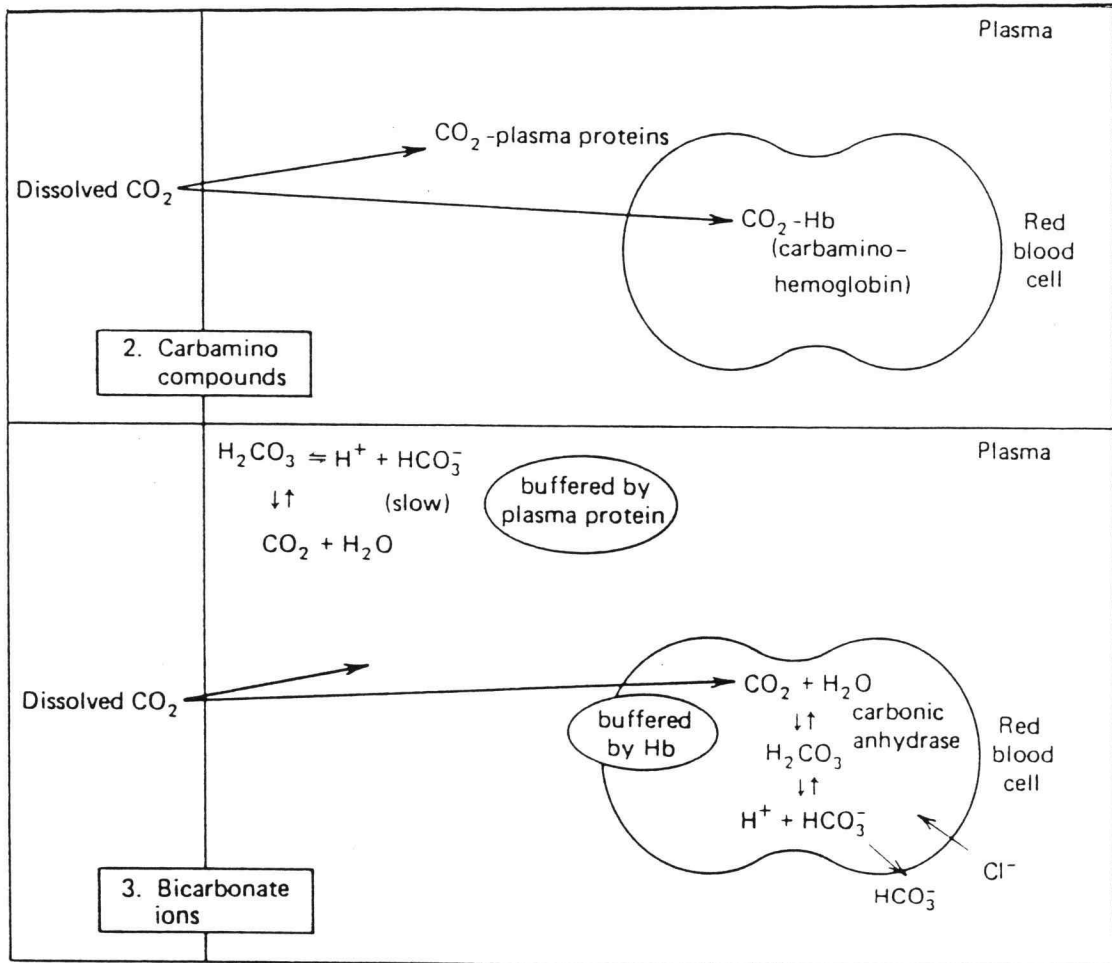


Figure 5 (cont.) - Schematic representation of the transport mechanisms of CO₂. From Fundamentals of Human Performance by G.A. Brooks & T.D. Fahey. Copyright ©1987 by Macmillan Publishing company. Reprinted by permission of the publisher.

Carbon Dioxide Dissociation Curve

The relationship between the quantity of carbon dioxide and the partial pressure of carbon dioxide (PCO_2) is represented by the CO_2 dissociation curve (Figure 6). An increase in PCO_2 causes the total amount of CO_2 binding with the protein fraction (globin) of the hemoglobin molecule to increase (Stegemann, 1981).

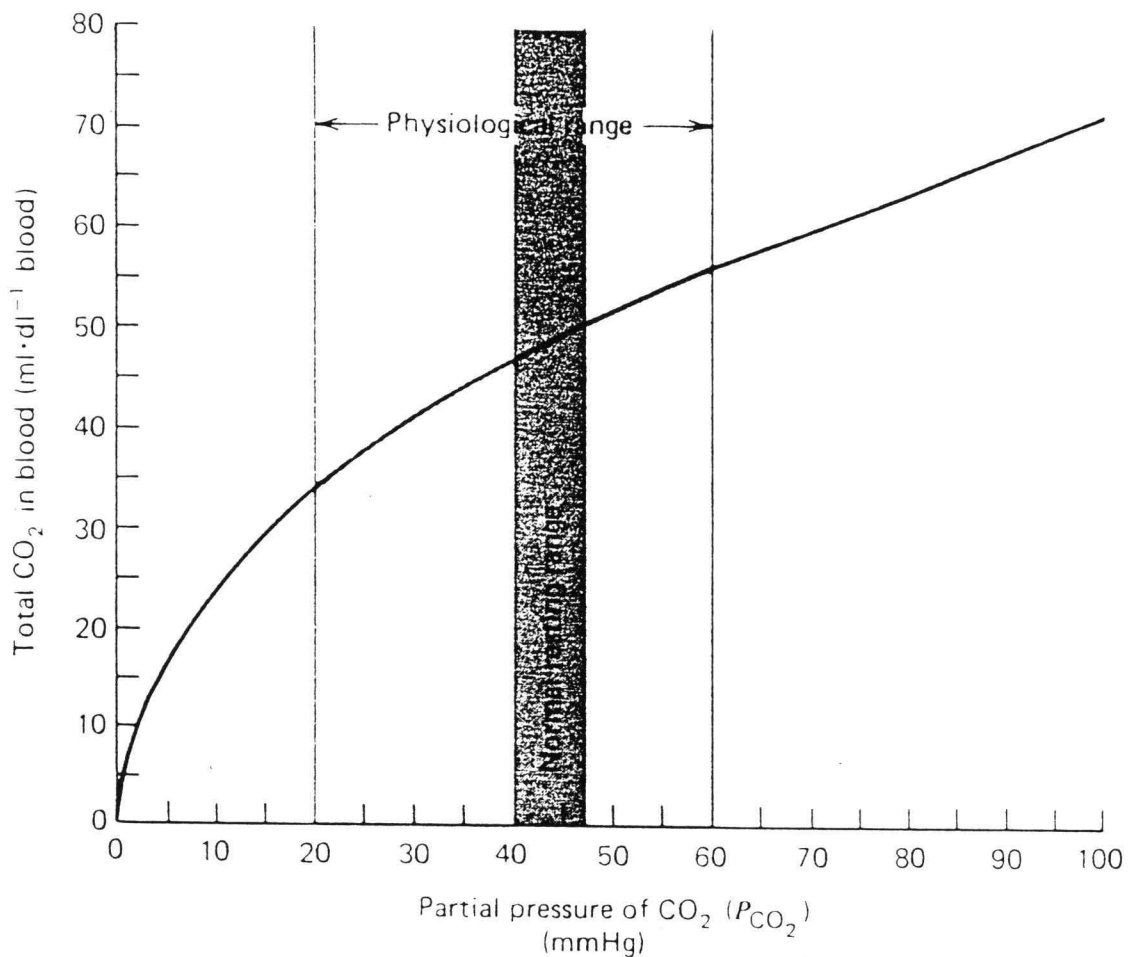


Figure 6 - The carbon dioxide dissociation curve. From Fundamentals of Human Performance by G.A. Brooks & T.D. Fahey. Copyright ©1987 by Macmillan Publishing company. Reprinted by permission of the publisher.

Gas Stores

Gas stores in the body assist the heart and lungs in transporting oxygen and carbon dioxide to meet the metabolic demands of intense exercise. If increases in cardiac output and ventilation do not meet the oxidation demands, oxygen stores in muscle and in arterial blood are drawn upon to meet the oxygen deficit. Similarly, CO₂ that cannot be removed immediately, is shuttled into body stores so that the effects on venous PCO₂ and pH are minimized. Ventilation during exercise would be three-to-four times higher if no CO₂ was stored and tissue and venous PCO₂ were controlled at resting levels. Carbon dioxide stores are large; the bicarbonate in metabolically active tissues can store about .05 ml/kg/mmHg during rest, and 2-3 ml/kg/mmHg in moderate exercise bouts. Carbon dioxide originates in the muscles, however, the muscles become much better perfused with blood during exercise and thereby extract much of the excess CO₂ produced. The relationship between changes in mixed venous PCO₂ and the volume of CO₂ stored during exercise follows the same pattern of the blood CO₂ dissociation curve, which "levels off" as PCO₂ increases. Carbon dioxide stores are particularly important when discussing the respiratory exchange ratio, which will not reflect the exchange at the tissue level if CO₂ storage is taking place.

Related Research

Ruhling and Williams (1984) investigated the acute effect of consuming 1.8 +/- .05 liter of degassed water within 12 hours of a test for maximal oxygen consumption using the Balke Treadmill Protocol. The altered state of water structure due to the removal of gas was believed to influence plant and animal growth and enzymatic activity. The results showed that consuming degassed water does not affect the work performance of normal, healthy, young men and women.

Although caution must be exercised when generalizing from case studies, Bell et al. (1987) claimed that a sixty-nine year old male was having right ventricular failure because of wet beriberi. The ingestion of excessive quantities of carbonated beverages was presumed to have reduced his thiamine levels, which, in turn, caused the right ventricular failure. The

coenzyme of thiamine (thiamine pyrophosphate) is involved in the reactions to remove carbon dioxide. Intake of carbonated beverages to the extent that thiamine levels are lowered could hinder the ability to rid the body of CO_2 and thereby decrease aerobic performance.

Hallberg & Rossander (1982) served composite meals (hamburger, string beans, and mashed potatoes) with different drinks to clarify if the beverages had any significant influence on the absorption of non-heme iron. A reduction in iron absorption may affect the oxygen carrying capacity of the blood. However, the carbonated beverages (beer and Coca-Cola) actually showed a tendency for increased non-heme iron absorption as compared to water.

Zachweija et al. (1991) examined whether adding carbonation to either water or a low calorie sport drink would affect gastric emptying. After five minutes of cycling at 55 % of $\dot{V}\text{O}_{2\text{max}}$, subjects ingested 5.5 ml/kg body weight of carbonated or non-carbonated beverages and continued to cycle for another fifteen minutes. The results indicated that carbonation had no effect on gastric emptying.

In the past, exercise-induced hypoxemia (EIH) at sea-level was thought to occur only in sufferers of cardiopulmonary disease. Powers et al. (1988) found, using a pulse oximeter, that arterial oxygen saturation was significantly lower ($90.0 \pm .88\%$) in a group of elite runners and cyclists as compared to an untrained group ($95.1 \pm .88\%$). Possible explanations for EIH include: a) diffusion limitations and/or ventilation-perfusion inequality due to decreases in PO_2 and b) very short red blood cell transit times in the pulmonary capillary bed.

A follow-up study by Powers et al. (1989) had highly trained athletes who exhibited EIH ($\% \text{SaO}_2 < 92\%$) perform two cycle ergometer exercise tests to determine maximal oxygen consumption under normoxic (21 % O_2) and mild hyperoxia conditions (26 % O_2). The results showed $\dot{V}\text{O}_{2\text{max}}$ during hyperoxia to be significantly elevated ($P < 0.05$) as compared to normoxia (74.7 vs. $70.1 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$). The prevention of hypoxemia and arterial O_2 desaturation increased mean capillary PO_2 in skeletal muscle and, in turn, allowed for a larger arteriovenous O_2 difference. The relevance of the work of Powers and co-workers to the present investigation lies in their demonstration that inadequate oxygen saturation of arterial blood can limit maximal oxygen consumption. If carbonated beverages were to affect oxygen saturation of

hemoglobin, or reduce hemoglobin levels, then aerobic capacity could be compromised.

OBJECTIVES

The objectives of this study were:

- 1) To determine the difference in arterial oxygen saturation of hemoglobin during the performance of a maximal oxygen consumption test after three weeks of carbonated beverage consumption (C) and after three weeks of consuming no carbonated beverages (NC).
- 2) To determine the differences in serum hemoglobin concentrations after C and after NC.
- 3) To determine the differences in maximal oxygen consumption after C and after NC.

ASSUMPTIONS

Assumptions applicable in this study were:

- 1) Twenty-eight ounces of carbonated beverages per day for three weeks was a sufficient volume and time period to identify a carbonated beverage effect, if one exists.
- 2) The subjects followed their treatment regimen.
- 3) The subjects did not change their eating, drinking (other than as directed for the treatment), or exercise habits during the course of the study.

DELIMITATIONS

The delimitations of the study are that results can be generalized to well-trained triathletes and bicyclists between the ages of 19-24 and to those

consuming 28 ounces of carbonated beverages per day for a period of three weeks.

LIMITATIONS

The limitations of the study are the quantity of carbon dioxide in the Calistoga water beverage and the quantity of carbon dioxide in the beverage that was actually consumed. Diet and training volumes during the six weeks of the study and fluid intake during the noncarbonated period were not monitored or controlled.

VARIABLES

The dependant variables were:

- percent saturation of arterial blood with oxygen - SaO_2
- serum hemoglobin concentration - Hb
- maximal oxygen consumption - $\dot{V}O_{2max}$

The independant variable was the consumption of 28 ounces of carbonated beverages.

RESEARCH HYPOTHESIS

The research hypothesis employed was that the consumption of moderate quantities of carbonated beverages (28 ounces/day) will not effect arterial oxygen saturation of hemoglobin (SaO_2), hemoglobin (Hb) concentrations, or maximal oxygen consumption ($\dot{V}O_{2max}$) during maximal cycle ergometry.

STATISTICAL HYPOTHESIS

The statistical hypothesis used SaO_2 , Hb concentration, and $\dot{V}O_{2max}$ as variables.

Across the two treatment periods (three weeks of carbonated beverage consumption vs three weeks of no carbonated beverage consumption), there will be no significant difference in:

- a) SaO_2
- b) Hb concentrations
- c) $\dot{V}O_{2max}$

OPERATIONAL DEFINITIONS

Arterial Oxygen Saturation (SaO_2) - percentage of oxygen-binding sites of hemoglobin occupied by a molecule of oxygen. At the lungs, Hb is approximately 97 percent saturated with oxygen whereas in the capillary beds of the muscle tissue, some of the oxygen is released, and therefore the venous blood is only 75 percent saturated with oxygen at rest (Brooks & Fahey, 1984).

Hemoglobin (Hb) - a protein molecule in the red blood cell that has the ability to take up oxygen at the lungs and release oxygen to the muscle tissues (Solomon & Davis, 1983). The four atoms in the hemoglobin molecule can transfer one molecule of oxygen. Men typically have 15 to 16 grams of hemoglobin per 100 ml of blood (g/dl blood) whereas women have 14 g/dl of blood (McArdle et al., 1991). Each gram of hemoglobin can carry 1.34 ml of oxygen (Brooks & Fahey, 1984).

Maximal oxygen consumption ($\dot{V}O_{2max}$) - the rate of oxygen utilized by the working tissues during heavy exercise. Maximal oxygen consumption is reached during a progressively increasing exercise test when the oxygen consumption plateaus, or increases minimally, despite a further increase in the workload (McArdle et al., 1991).

Carbonated Beverages - a beverage which has carbon dioxide (CO_2) injected into the fluid for effervescence and/or a more acidic taste (McCellan, 1955). Typically, a volume of CO_2 three to five times that of the liquid is used to ensure 100 percent saturation of the beverage.

CHAPTER II

REVIEW OF LITERATURE

Water is required for the majority of bodily functions. This chapter will begin with a review of the literature regarding carbonated water. The first section includes an introduction on carbonated beverage ingestion and absorption, followed by a description of pulse oximetry, animal studies, and then will conclude with a discussion of hypoxemia.

Water

Water is necessary for many bodily functions and therefore is often recognized as one of the most important nutrients. Water, being present in the intracellular and extracellular fluid, makes it a medium which maintains the acid-base balance necessary for the proper concentrations of electrolytes and ionization of complex chemical reactions (Asterita, 1986). Water per se, has no calories, but may contain trace elements, such as the magnesium and calcium found in "hard" water and the iron typically found in well water. The average healthy person ingests 5.5 pints of water daily, with approximately 70 percent from the actual drinking of water and the other 30 percent coming from the ingestion of food which contains water. Sixty percent of daily water elimination is through urination, 25 percent through perspiration, 12 percent as exhaled water vapor through the lungs, and three percent leaves the body in the feces (Asterita, 1986).

Carbon Dioxide

The dioxide of carbon (CO₂) naturally occurs as a gas. Carbon dioxide is commonly added to beverages to give them effervescence and/or a more acidic taste. Little research has been done to determine what happens to the gas after ingestion, whether it alters digestion, if any CO₂ enters into the blood, and whether or not the chronic consumption of carbonated beverages affect oxygen transport.

Carbon dioxide may enter the body, and eventually the blood stream, three ways: a) respiration, b) through the skin, and c) ingestion. The ambient air inhaled during respiration allows a CO₂ concentration of 0.03 percent to enter into the body. McClellan et al. (1945) analyzed the physiological effects of CO₂ water baths on alveolar tension, skin temperature, and respiratory metabolism. The results showed a five to ten percent increase in alveolar CO₂ tension, which led to the hypothesis that the extra CO₂ was obtained by absorption through the skin, and eliminated through the lungs.

Estimating the levels of CO₂ ingested with the consumption of a carbonated beverage is difficult because, ultimately, the amount is a function of the temperature and the pressure of the beverage (McClellan, 1955). Bottling companies typically use a volume of gas three to five times that of the liquid to make sure the beverage is 100 percent saturated. It has been estimated that there is 3-5 liters of CO₂ in one liter of liquid. Upon opening the beverage, the equilibrium is upset and the CO₂ starts to escape from the liquid (Deamer & Selinger, 1988). Considering that carbonated beverage consumption rarely exceeds 0.5 liter in one sitting, it can be estimated that 0.5 to 1.5 liters of CO₂ are probably ingested.

Sharf (1943) analyzed the factors which determine the retention of CO₂ in carbonated beverages and found that the solubility of CO₂ is decreased by: a) dissolved sucrose, b) large fractional volume of gas space above the beverage, c) the presence of nucleation sites (impurities) which allows for bubble formation, and d) higher temperatures, while, on the other hand, the solubility of CO₂ is unaffected by small amounts of flavoring, salt and coloring substances.

Carbon Dioxide Absorption

Little research has been done on the movement of CO₂ through the digestive tract. The movement of fluid into the esophagus, then the stomach and the duodenum, will depend on the gastric activity at the time of ingestion. McIver et al. (1926, p.93) stated the following in the introduction to their study:

If the exchange of gas between the lumen of the gastrointestinal tract and the blood circulating in its wall is due to the

physical process of diffusion, it must conform to the following criteria. First: an equilibrium will be reached when the partial pressure of gas in the lumen is equal to the mean tension of the gas dissolved in the circulating blood. This condition of equilibrium should be the same, regardless of the direction from which it is approached. Second: the exchange of gas which leads to the development of this equilibrium will follow the course determined by the law that the rate at which gas passes through a permeable membrane at any time will be proportional to the difference in pressure of this gas on the two sides of the membrane. Third: the actual value of the rate at which the gas passes across the membrane is, for any given pressure difference, determined strictly by the properties of the gas and by the area and thickness of the membrane.

The actual experiment included the use of anesthetized cats, with both their pylorus and cardia ligated to hold 60 c.c. of an injected gas. The gas samples included: oxygen (O_2), nitrogen (N_2), hydrogen (H_2), methane (CH_4), hydrogen sulfide (H_2S), carbon dioxide (CO_2), and air. The results showed that the movement of CO_2 from the lumen to the venous blood and vice-versa followed the gas laws of diffusion. The rate of diffusion, where the gases pass through the gastric membrane into the blood, was the fastest with CO_2 , which reached equilibrium quickly (80-90 minutes) in the stomach. The model whereby the pylorus and cardia are ligated interferes with the motility of the gastro-intestinal tract. In vivo, gases may not be as highly absorbed because of the more rapid passage into the duodenum (McClellan, 1955) or loss through belching.

The movement of fluid from the stomach and into the duodenum also depends upon the gastric activity at the time of ingestion (Poggrund and Steggerda, 1948). The passage of fluid into the duodenum is rapid when the gastric chamber is empty. If the fluid is carbonated, the dissolved CO_2 is released into a gaseous form as the temperature of the fluid increases. Some of the freed CO_2 may trigger the regurgitation mechanism and be belched, be absorbed into the bloodstream through the duodenal membrane, and/or be passed into the duodenum and eventually escape as flatus. Analysis of normal flatus produced an average value of 7.5 percent CO_2 (Poggrund and Steggerda, 1948).

The absorption rate of any gas in the intestine is dependant upon the partial pressure gradient existing between the duodenum and the venous blood. Pogrud and Steggerda (1948) introduced 500 c.c. gas volumes (nitrogen, oxygen, and carbon dioxide) ten centimeters into the rectum and took 30 c.c. samples, every 15 minutes, to later determine gas concentrations. The average venous blood CO₂ tension was found to be 52 mmHg, which the investigators believed, limited the amount of CO₂ that was absorbed into the venous blood from the duodenum. Equilibrium between the venous blood and the duodenum wall was usually established within 90 minutes.

In summary, CO₂ is readily passed through both the gastric and intestinal membrane and quickly equilibrates with venous blood. Experimental model limitations have not allowed for any objective evidence regarding the "true" relations between ingested CO₂ and its presence in the digestive tract, but researchers believe that CO₂ is controlled by the physical laws of diffusion (McClellan, 1955).

The Effect of Carbonated Beverages on Respiration

There are no studies which indicate that the consumption of carbonated beverages increases the volume of CO₂ eliminated through the lungs. Bottje & Harrison, (1984) found that carbonated water infused into the crop of heat-stressed cockerels produced lower respiration rates as compared to cockerels infused with tap water. The authors believe this phenomena is a result of the slightly lower PCO₂ found in the arterial blood of the cockerels infused with carbonated water ($p < 0.1$). However, McClellan (1955) stated that he suspected little or no alteration in the amount of CO₂ eliminated by the lungs, even if all the CO₂ from the carbonated beverage was transferred into the bloodstream, simply because of the lability of CO₂ in body fluids.

Oxygen Transport

Dissolved oxygen

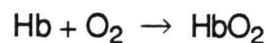
The pressure gradient between the alveolar air and the pulmonary capillary blood determines the amount of O₂ diffusing across the pulmonary

membranes. This diffusing capacity increases during exercise and is higher in endurance-trained individuals (McArdle et al., 1991).

Ventilation establishes an alveolar pressure ($P_{A}O_2$) of about 105 mmHg, which is necessary to get the O_2 into the physical solution of the blood. The low solubility of O_2 in body water at 37°C allows for only 0.3 ml $O_2 \cdot dl^{-1}$ blood to be dissolved.

Hemoglobin

Hemoglobin is a heme-iron compound located in the erythrocyte and can quickly and reversibly bind and release O_2 without the assistance of an enzyme. These reactions can be described by the term oxygenation, and displayed as:



The hemoglobin molecule consists of a protein globin, combined with four nonprotein groups called hemes (Spence & Mason, 1987). Four polypeptide chains make-up the globin protein and each chain has an iron-containing (Fe^{+2}) heme group. Each heme group can combine with a molecule of O_2 , so the hemoglobin molecule typically transports four molecules of oxygen and is then called oxyhemoglobin. As will be discussed later, CO_2 is also transported by the protein (globin) portion of the hemoglobin molecule, in contrast to oxygen, which is carried by the iron of the heme groups. These properties allow both gases to be transported simultaneously (Solomon & Davis, 1983). Hemoglobin production requires iron and approximately 65 percent of the body's iron stores are in hemoglobin (Spence & Mason, 1987).

Aerobic training produces a small increase in the production of red blood cells and, thereby, an increase in total hemoglobin. However, at rest, the concentration of hemoglobin declines, since the increase in plasma volume seen with aerobic training is larger than the increase in red blood cells (Lamb, 1984). Shelby (1991) stated that hemoglobin concentrations in athletes are commonly 1.0 to 1.5 g/dl below the lower end of the normal range.

The quantity of hemoglobin ($grams \cdot 100 ml^{-1}$) and the oxygen binding capacity of hemoglobin ($ml O_2 \cdot g^{-1}$) determine the oxygen carrying capacity of blood ($ml \cdot 100 ml^{-1}$ blood). Men typically have 15 to 16 grams/100 ml of blood

(women 13-14 grams) and each gram of hemoglobin can bind with 1.34 ml of oxygen (1.34 ml O₂/g Hb) (Brooks & Fahey, 1984). Quantitatively, arterial O₂ transport can be described as the product of O₂ carrying capacity of hemoglobin and the arterial hemoglobin summed to the O₂ physically dissolved (Brooks and Fahey, 1984).

$$CaO_2 \text{ (arterial O}_2\text{)} = [(O_2 \text{ carrying capacity of hemoglobin)} \times \text{(arterial hemoglobin content)} + O_2 \text{ physically dissolved}]$$

The total quantity of O₂ transported from the heart to the working tissues can be determined from knowing the cardiac output and the O₂ carrying capacity of blood. A cardiac output of four to six liters·min⁻¹ (rest) will allow for 40 to 50 ml O₂ per liter of blood to be removed and the total O₂ uptake to be 0.2 to 0.3 liters·min⁻¹ (Astrand-Rodahl, 1986). An athletic male can increase their O₂ uptake from 0.25 to 5.00 liters·min⁻¹ when heavily exercising through a minimal increase in stroke volume, and by large increases in heart rate and arterial venous (a- \bar{v} O₂) difference (Astrand-Rodahl, 1986).

The quantity of oxygen extracted from the blood is reflected by the difference in partial pressure of arterial and venous blood (a- \bar{v} O₂ difference) (Berger, 1982). Endurance aerobic training is associated with larger a- \bar{v} O₂ differences, probably because the trained athlete has aerobically trained muscle tissue and a greater quantity of mitochondria to better utilize the oxygen. The larger quantity of mitochondria seen with training produces more carbon dioxide as a result of oxidative metabolism and causes more CO₂ to be present in the venous blood (Lamb, 1984).

Oxyhemoglobin dissociation curve

Oxygen is more effectively utilized during exercise by the redistribution of blood to the working skeletal muscles and by the properties of the oxyhemoglobin dissociation curve (Hlastala, 1979). The dissociation curve (Figure 1, Chapter I) describes the partial pressure of oxygen (PO₂) and its relationship to the oxygen content of blood. The sigmoidal (S) shape and the alveolar-capillary PO₂ of about 100 mmHg describes how the blood can be 98 percent saturated with O₂ while a PO₂ of three mmHg, seen in skeletal muscles

during exercise, causes hemoglobin to release its hold on nearly all of the oxygen (Berger, 1982).

In addition to the partial pressure of O_2 , other factors, such as temperature, pH, PCO_2 , and 2,3- diphosphoglycerate (2,3-DPG), increase the quantity of O_2 released to the working skeletal muscles during exercise (McArdle et al. 1991). An increase in any of these factors causes the dissociation curve to shift downwards and to the right and results in a reduction of the O_2 binding to hemoglobin by 10 to 15 percent (Brooks and Fahey, 1984). This phenomenon is called the Bohr Effect.

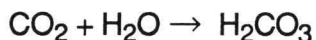
Blood temperature and the production of 2,3-DPG are both elevated during exercise and cause the dissociation curve to shift to the right. The shift of the dissociation curve may also occur as a result of lower pH levels (Glauser and Forster II, 1967). The end product of aerobic and anaerobic energy metabolism is CO_2 , which produces larger quantities of H_2CO_3 (carbonic acid), and lactic acid, which cause the dissociation curve to shift to the right (Stegemann, 1981).

The CO_2 produced by muscle tissue increases the partial pressure of carbon dioxide (PCO_2) in the blood, which also contributes to the unloading of O_2 from hemoglobin. An increase in the PCO_2 of arterial blood from 40 mmHg to 80 mmHg (representative of the temporary local changes in blood level at the tissues) would result in a 16 percent increase in the amount of O_2 released and delivered to the tissues (Berger, 1982). It is important to recognize that the factors described above (temperature, pH, PCO_2 and, 2,3-DPG) are likely to occur simultaneously with the onset of exercise.

In summary, a combination of events contribute to supplying the increased oxygen demanded by exercising muscle tissue. Oxygen transport is maximized by the increase in cardiac output and the maintenance of arterial O_2 saturation. Subsequently, more O_2 can be released to the working tissues during intense exercise because of the relationship described by the oxyhemoglobin dissociation curve and the influences of the Bohr Effect.

Carbon Dioxide Transport

Carbon dioxide is an end product of cellular metabolism. After cellular formation of CO_2 , it diffuses into the tissue fluid, across the capillary wall, and into the blood plasma where it reacts with water to form carbonic acid.

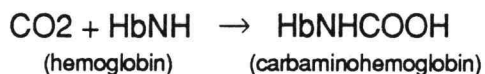


Only about five to seven percent of the CO_2 is carried in the plasma as dissolved carbon dioxide but, analogous to oxygen transport, this portion is necessary to provide sufficient movement within the circulating blood volume to establish PCO_2 (Brooks & Fahey, 1987).

A large percentage (90-95 percent) of the CO_2 diffuses from the plasma into the erythrocyte where: a) 65 percent is catalyzed by carbonic anhydrase to accelerate the reaction with water to form carbonic acid, and eventually ionize to hydrogen ions (H^+) and bicarbonate ions (HCO_3^-);



b) 20 percent of the CO_2 at the tissue level reacts directly with the amino acid molecules of blood to form carbamino compounds;



c) and five percent is dissolved in the erythrocyte and diffuses into the plasma (McArdle et al., 1991).

All of the reactions described above are reversible. At the lungs, CO_2 diffuses out of the blood into the aveoli and the concentration of CO_2 within the plasma and the erythrocyte declines (Solomon and Davis, 1983). The direction of the reaction depends upon the PCO_2 , pH, and the bicarbonate ion concentration. In the tissues, where the hydrogen ions are held by hemoglobin molecules and the PCO_2 is relatively high, there is a facilitation in the formation of bicarbonated ions. However, at the lungs, the hemoglobin molecule "gives-up" hydrogen ions, the PCO_2 is lower, and bicarbonate ions and carbonic acid metabolize into carbon dioxide and water (Spence & Mason, 1978).

CO₂ dissociation curve

The relationship between CO₂ content and the partial pressure of CO₂ (PCO₂) can be graphically represented by the CO₂ dissociation curve (Figure 3) (Brooks & Fahey, 1984). At rest, the arterial partial pressure of CO₂ (P_aCO₂) is approximately 40 mmHg, which corresponds to an arterial CO₂ content (C_aCO₂) of 47 ml·dl⁻¹ blood. Comparatively, the mixed venous partial pressure of carbon dioxide (PCO₂) rises to 50 ml·dl⁻¹ blood. During heavy aerobic exercise, the increase of acid into the arterial blood causes the P_aCO₂ to decrease and the entry of CO₂ into the venous blood causes the the partial pressure of carbon dioxide (P_vCO₂) to increase. Figure 3 also shows the physiological range during heavy exercise where the venous-arterial partial pressure may differ by as much as 40 mmHg, (60-20 mmHg) which corresponds to an increase in the CO₂ content in the blood by 20 percent (55-35 ml· dl⁻¹) (Brooks & Fahey, 1987).

The Effect of Consuming Different Drinks on Iron Absorption

Selby (1991) reported that foods such as tea, cereal, grains, and coffee as well as drugs such as antacids, tetracycline, and H₂-blockers are detrimental to iron absorption. More closely related to this study, Hallberg et al. (1982) found that the absorption of non-heme iron from composite meals is dependant on the type of drinks ingested with the meal. The composite meal consisted of hamburgers (110 grams), string beans (60 grams), and mashed potatoes (150 grams). The meals were served after an overnight fast and were followed by the random consumption of either 250 ml of water (control) or one of the followings drinks: tea, coffee, milk, beer, Cola-Cola, wine, pure alcohol, or orange juice. Series four and five of the experiment had ten males and seven females drink light beer, which had an alcohol content of 2.25 percent, and Coca-Cola. A solution of radio-iron (10 ml 0.01 M hydrochloric acid) containing 3 mg of iron was given to the subjects to later determine the absolute absorption. The ingestion of the carbonated beverages, beer and Cola-Cola, tended to increase the absorption of iron, but the change was not statistically significant (p>0.05) when compared to water.

The Effect of Carbonated Beverages on Gastric Emptying

It is necessary to consider the effect of carbonated beverages on gastric emptying rates because it is conceivable that the amount of CO₂ that enters into the blood is dependant upon how quickly the beverage goes through the digestive system.

Hale et al. (1950) investigated whether carbonated distilled water or sodium bicarbonate had a significantly different effect from an equal quantity of distilled water or sodium chloride solution on stomach acid secretion and the rate of gastric emptying. One group of males was given the distilled and carbonated water while the other group was given sodium chloride (0.9 percent) and sodium bicarbonate (1.3 percent) solutions. After an overnight fast, one group of male subjects was given 250 c.c. of carbonated distilled water (containing 4.4 volumes of CO₂ per unit of water at 60°F) through a stomach tube and a sample was taken from the stomach every 10 minutes for one and one-half hours to determine gastric volumes and acid levels. The same procedures were performed with the other solutions. The results showed that there was no difference between the distilled water and the carbonated distilled water on gastric emptying or the output of HCL.

Lolli et al. (1952) did a follow-up study which included more gastric volume measurements, longer time periods over which the observations were made, and a meal given with the consumption of the beverages. One group received a test meal of farina and barium sulfate followed either by 100 c.c. or 200 c.c. of water or carbonated water and had gastric emptying determined by fluoroscopy. The second group was presented with 100 c.c. of a liquid test meal (undiluted commercial beef bouillon) through a Levine stomach tube and five minutes later, given either 100 c.c. or 200 c.c. of water or carbonated water. For the control tests, 100 c.c. of water was given, and for the experimental tests, 100 c.c. of carbonated water was given. The average gastric emptying time was longer after the introduction of water verses carbonated water (105 vs 65 min). Both treatment groups showed that gastric emptying was hastened to the same extent by the ingestion of 100 c.c. and 200 c.c. of carbonated water. The level of significance and other statistical information was not presented.

More recently, Beard (1990) studied the effect of carbonated solutions on gastric emptying during prolonged cycling. Eight males cycled for two hours at

70 percent of their $\dot{V}O_{2\max}$ while consuming approximately 150 ml of one of four test solutions every 15 minutes. The test solutions were: a) carbonated, 10 percent carbohydrate, b) noncarbonated, 10 percent carbohydrate, c) carbonated, non-carbohydrate, and d) noncarbonated, non-carbohydrate. The comparison between the carbonated solution with no carbohydrate concentration and the noncarbonated solution with no carbohydrate showed no difference ($p>0.05$) in the rate of gastric emptying.

Zachwieja et al. (1991) also analyzed the effect of carbonated water and flavored carbonated water on gastric emptying during cycle ergometry. Fifteen subjects rode at 55 percent of their $\dot{V}O_{2\max}$ for 20 minutes while ingesting 5.5 ml/kg body weight of either water, carbonated water, carbonated flavored water, or noncarbonated flavored water. Compared to water, both forms of carbonated drinks showed no significant effect ($p>0.05$) on gastric emptying or any of the other variables analyzed (heart rate, rating of perceived exertion, or rating of gastrointestinal comfort).

Pulse Oximetry

As stated earlier, the oxygen carrying capacity of blood is dependant upon the product of arterial oxygen saturation (SaO_2) and the concentration of hemoglobin (14-18 g/dl of blood for males and 13-16 g/dl of blood for females). Hemoglobin can loosely combine with four atoms of O_2 by opening its chemical structure so that two units slightly separate. To release the O_2 , the two units quickly come together and virtually force the O_2 out of the molecular structure in a single motion (Solomon & Davis, 1983). Pulse oximetry permits the non-invasive measurement and continuous monitoring of arterial oxygen saturation without the discomfort and risks of arterial puncture (Schnapp & Cohen, 1990).

Mechanism

Pulse oximeters work on the principles of Beer's Law, which states that light absorption can determine the concentration of an unknown solute dissolved in a solvent (Schnapp & Cohen, 1990). The probe is commonly placed on the index finger and is made up of a light source and a sensor. The quantity of light absorbed by the skin, tissue, and venous blood remains

constant. The contraction of the left ventricle, on the other hand, produces a small increase in arterial blood volume in the finger.

Light sources of two wavelengths are used (660 nm and 940 nm) because of their known absorption characteristics of oxyhemoglobin and reduced hemoglobin (Omaha manual, 1990). Comparing the ratio of pulsatile and baseline absorption of the two wavelengths can determine the ratio of oxyhemoglobin to reduced hemoglobin and then oxygen saturation (Schnapp & Cohen, 1990).

$$R = \frac{(\text{pulsatile absorbance}) \div (\text{baseline absorbance})}{(\text{pulsatile absorbance}) \div (\text{baseline absorbance})}$$

Accuracy

Yelderman & New (1983) compared the hemoglobin concentration values obtained from pulse oximetry to those from CO - oximeters. CO - oximeters typically have four or more wavelengths to determine not only hemoglobin concentrations but also the "branches of hemoglobin," such as carboxyhemoglobin and methemoglobin (Schnapp & Cohen, 1990). The comparison between the two measurements showed a correlation coefficient of 0.98; slope, 1.03, and $p < 0.0001$.

Breuer et al. (1990) compared the measurements of SaO₂ obtained from pulse oximetry, photometric measurements, and calculation procedures. The calculation of SaO₂ was determined using: a) photometric measurements, included the ultra-sound hemolysis of the injected blood sample at 505 and 600 nm wavelengths, b) the algorithms suggested by Kelman (1966), Severinghaus (1979), and Siggaard-Andersen (1980), and c) the OXI 3 pulse oximeter (Radiometer, Denmark), based on spectrophotometric analysis of two emitted wavelengths (660 - 940 nm). Linear regression analysis provided a significant correlation coefficient of $r = 0.47$ ($p < 0.001$) between pulse oximetry and photometric determinants of SaO₂. Superior validity and reliability was found with the pulse oximeter when compared to the calculated algorithms. The authors concluded that pulse oximetry can replace the invasive determinants of SaO₂, especially in situations where constant monitoring is required.

To measure the accuracy of pulse oximetry during incremental exercise, Powers et al. (1989) used the Ohmeda model 3700 oximeter and had ten healthy males perform a $\dot{V}O_2$ max test on a cycle ergometer with the workload increasing 30 Watts every minute until volitional fatigue while monitoring HbO₂. The results were compared to the direct measurement of %HbO₂ using a four-wavelength spectrophotometer CO - oximeter (Instrumentation Laboratories 282) and showed that pulse oximetry provided valid estimates between values of 85 and 97 percent SaO₂ during incremental exercise.

Animal Studies

Researchers have been interested in the effect of carbonated beverages on animals which are placed under heat stress. Thermal polypea occurs when the animal increases the rate of ventilation to aid themselves in evaporative cooling. This coping mechanism reduces the arterial partial pressure of carbon dioxide (PCO₂) and produces an acid-base disturbance called respiratory alkalosis (Raup, 1990). Researchers Bottje & Harrison (1984) and Raup & Bottje (1990), hypothesize that the ingestion of carbonated beverages may provide a source of CO₂ and assist in the correction of this acid-base imbalance.

Bottje & Harrison (1984) fitted twenty cockerels with a plastic cannulae in the carotid artery and crop through which a solution of tap water, carbonated water, two percent sodium bicarbonate, or three and half percent calcium chloride ($.41 \text{ ml} \cdot \text{min}^{-1} \cdot \text{kg}$ body weight) was delivered. Blood pH, PCO₂, and respiration rate was monitored every 15 minutes for 90 minutes in thermoneutral conditions (25°C), and then the temperature was elevated to 37°C for another 90 minute period. A 0.7 ml sample of blood was withdrawn through the carotid cannula into a 1.0 c.c. heparinized glass syringe to later determine the arterial blood PCO₂ and pH. The carbonated water did not alter any of the variables during the thermoneutral condition. However, carbonated water kept the blood pH lower than the tap water in the heat stress condition (7.522 vs. 7.564). The results led the authors to believe that carbonated water produced a favorable alteration of the acid-base balance during acute heat stress when compared to tap water.

In a similar study, Raup & Bottje (1990) investigated the effect of carbonated water on arterial pH, PCO_2 , and plasma lactate in heat-stressed broilers. The experimental methods were similar to those of Bottje & Harrison (1984), except the heat was increased gradually from 25°C to 37°C over a three hour period and then maintained for another two hours. The hematocrit decreased in both groups of birds but was higher ($p < 0.05$) in the carbonated water vs. the tap water group after two hours of heat stress. The difference in the hematocrit did not alter oxygen saturation of hemoglobin or the partial pressure of oxygen. After four to five hours of heat-stress, the PCO_2 was higher and arterial pH and plasma lactate levels were lower in the birds given carbonated water. The authors concluded that carbonated water is capable of altering the blood pH and CO_2 in heat-stressed domestic fowl so as to maintain the critical acid-base balance of the blood.

Hypoxemia

Recent research on exercise induced hypoxemia (EIH) by Powers et al. (1988) has investigated the possibility that inadequate oxygen saturation could be a limiting factor for elite athletes during the performance of a test of maximal oxygen consumption ($\dot{V}O_{2max}$). Six to eight subjects were divided into either: a) untrained (physically active but not participating in regular exercise program), b) moderately trained (participated in aerobic classes three to seven times per week), or c) elite, highly-trained (high level runners or cyclist with $\dot{V}O_{2max}$ greater than 68 ml·kg⁻¹·min⁻¹). The subjects performed an incremental cycle ergometer test to volitional fatigue while arterial oxygen saturation (SaO_2) was monitored via ear oximetry using the Ohmeda 3700 pulse oximeter. Exercised induced hypoxemia was defined as a SaO_2 value of less than 91 percent, and was not seen in the untrained or moderately-trained subjects. However, in 52 percent of the highly-trained athletes, EIH was seen (13 of 24 subjects), with the remaining 11 subjects having a mean SaO_2 level of 92.5 percent. The researchers hypothesized that EIH is a result of the decline in PO_2 during heavy exercise and/or the very short red blood cell transit times in the pulmonary capillary bed as a result of the exceptionally high cardiac outputs.

To investigate if EIH is a result of oxygen desaturation during maximal exercise, Powers et al. (1989) had a group of twenty subjects divided into two

groups: a) trained (performed aerobic exercise three to five times per week), and b) highly trained (SaO₂ values during maximal exercise below 92 percent).

Subjects performed two $\dot{V}O_{2\max}$ tests on a cycle ergometer while breathing air with 21 percent O₂ (normoxia for sea level) or 26 percent O₂ (hyperoxia). Both groups showed higher values of SaO₂ (trained 94.1 vs 96.1%; highly trained 90.6 vs 95.9%) during the hyperoxic condition, and this allowed the highly trained group to achieve higher $\dot{V}O_{2\max}$ values (74.7 vs 70.1 ml·kg·min). The results suggest that an increase in pulmonary gas exchange, as seen during hyperoxic conditions, may allow highly trained athletes who demonstrate EIH to achieve higher $\dot{V}O_{2\max}$ values.

Summary

Carbon dioxide (CO₂) is added to beverages to give them effervescence and/or a more acidic taste. The quantity of CO₂ in a beverage is usually three to four times the volume of the liquid but, in any case, is hard to quantify because, ultimately, the volume of CO₂ is a function of the temperature and pressure of the surrounding air. Carbon dioxide transferred in a fluid enters the gastric and intestinal tract, where it quickly equilibrates as described by the physical laws of diffusion. Respiration and gastric emptying appear not to be affected by the ingestion of carbonated water. Animal studies have shown that the ingestion of carbonated water can lower the pH level of arterial blood and increase the PCO₂.

It is evident from the review of literature that little work has been done on humans with respect to ingesting carbonated beverages. This fact is amazing, considering the percentage of the population which consumes carbonated beverages every day. The effect of chronic consumption of carbonated beverages on athletic parameters such as $\dot{V}O_{2\max}$ has not been investigated.

CHAPTER III

METHODS

The purpose of this study was to investigate the effect of carbonated beverages on oxygen saturation of arterial blood (SaO_2), serum hemoglobin levels (Hb), and maximal oxygen consumption ($\dot{V}\text{O}_{2\text{max}}$). All testing procedures were performed in the Human Performance Laboratory at Oregon State University, Corvallis, Oregon.

This chapter will be divided into three sections. The first section describes subject selection, experimental design, and carbonated beverage distribution. The second section details the instrumentation and procedures that were implemented for data collection. The final section presents the statistical analysis that was applied.

Subjects

Nine male competitive cyclists and triathletes between the ages of 19 and 24, volunteered to participate in this study. Each subject had a history of competition for at least two years and was actively training at the time.

Prior to the study, each subject completed a Medical Questionnaire (Appendix A) to ensure that they were individuals who were at minimal risk for heart disease. In addition, all subjects read and signed an informed consent document (Appendix B) acknowledging their understanding of the nature and potential risks of the study and their willingness to participate in the project. Subjects were asked to maintain their consistent pattern of dietary intake and level of training throughout the course of the study. The experiment was approved by the Oregon State University Institutional Review Board.

Experimental Design

Each subject underwent a three week period of carbonated beverage ingestion or a three week period where no carbonated beverages were consumed, followed by a $\dot{V}\text{O}_{2\text{max}}$ test. In the second three week period, the treatment of the groups crossed-over, where the subjects who were consuming

carbonated beverages abstained from all carbonated beverages, and those who were abstained began their consumption of carbonated beverages. A second maximal exercise test was performed following the second treatment period.

Carbonated Beverage Administration

At the start of their carbonated beverage treatment period, the subjects received a three week supply of Calistoga carbonated water (Calistoga Mineral Water Company, Calistoga, California 94515). Nothing had been added to the beverage except carbonation. A 28 ounce serving size contains 141 mg of sodium but no calories, protein, carbohydrates or fat. The carbonated beverage supply was divided into 21, 28 ounce pressure-sealed bottles. The subjects were to drink 28 ounces per day (1 bottle) after their afternoon workout. The beverages were to be refrigerated and consumed in one sitting. The consumption of other carbonated beverages (ie. soda pop, beer, etc.) was permitted with the understanding that a record be kept which monitored the type and quantity consumed per day (Appendix C). The subjects were asked to limit their drinking of other carbonated beverages such that they did not to exceed a total of 64 ounces per day.

Non - Treatment Period

Each group participated in a three week period where they are asked to refrain from consuming any carbonated beverages (as listed above). This asked for total abstinence.

INSTRUMENTATION AND PROCEDURES

Two physiological testing periods were conducted over a six week period. The testing periods included two determinations of $\dot{V}O_{2\max}$ and an evaluation of body composition (for subject description only). Each subject performed a $\dot{V}O_{2\max}$ test after the first three-week treatment period (carbonated beverage consumption or abstention), and again at the conclusion of the second three-

week treatment period (cross-over treatment). Body composition was determined before the second $\text{VO}_{2\text{max}}$ test.

Instrumentation

Gas Analysis: Standard open circuit spirometry was used to measure O_2 consumption, with O_2 and CO_2 concentrations being determined by an Applied Electrochemistry S-3A Oxygen Analyzer (Sunnyvale, California) and a Beckman LB - 2 Carbon Dioxide Gas Analyzer (Cardiopulmonary Instruments Department, 2500 Harbor Boulevard, Fullerton, California), respectively. A Parkinson - Cowen Dry Gas Meter (Barchard Engineering LTD.) was used to measure the inspiratory volumes. The gas analysis instrumentation was interfaced with an Apple II Plus microcomputer (10260 Bandley Drive, Cupertino, California 95014) using the REP - 200 C Software (1985) by Rayfield Equipment LTD (Chicago, IL). Before each $\text{VO}_{2\text{max}}$ test, the oxygen and carbon dioxide analyzers were calibrated with a known gas mixture. Subjects wore a headset which supported a two-way breathing valve and a mouthpiece that allowed for the inhalation of ambient air and exhalation of air into the gas analysis system.

Heart Rate Monitoring: During the $\text{VO}_{2\text{max}}$ tests, each subject was continuously monitored via the Mason-Likar 12-lead exercise electro - cardiograph (ECG) lead system (Pollock et al., 1984). The ECG monitoring was performed with a Quinton Model 630A electrocardiograph (Quinton Instruments Company, 2121 Terry Avenue, Seattle, Washington 98121) with continuous oscilloscope display with a recording made during the last ten seconds of every workload. Heart rate was monitored through the V_5 lead.

Hemoglobin Concentration: Prior to the test of aerobic capacity, a 5 c.c. sample was removed from an antecubital vein by a certified phlebotomist using standard venipuncture techniques. A Sigma hemoglobin diagnostic kit # 525-A (Sigma Chemical Company, St. Louis, MO) was used to determine total blood hemoglobin concentration which was based on the cyanmethemoglobin method.

Oxygen Saturation: An Ohmeda Biox 3740 Pulse Oximeter (1315 West Century Drive, Louisville, CO 80027) was used to monitor oxygen saturation throughout the maximal oxygen uptake test. The Flex II Probe was placed on the distal aspect of the left index finger and secured with tape.

Maximal Testing Procedures

Maximal oxygen consumption was measured during a progressively loaded, continuous maximal test performed on a cycle ergometer. Each subject was briefed on the methods involved in the testing and allowed a short (2-5 minutes) warm-up. The exercise test began at a workload of 80 Watts and at a cadence of 80 rpm. The workload was increased 30 Watts/min until the subject was unable to maintain the 80 rpm pedaling cadence. The peak $\dot{V}O_2$ was considered $\dot{V}O_{2max}$ when any two of the following three criteria were met: 1) a plateau in oxygen consumption with an increase in power output, 2) a respiratory exchange ratio of 1.10 or higher, or 3) achievement of plus or minus 10 beats/minute of age predicted maximal heart rate (Powers et al., 1988). Trained cyclists have been shown to obtain similar results on bicycle and treadmill ergometry with respect to $\dot{V}O_{2max}$ (Hermansen & Saltin, 1969). Gas analysis was performed at 30 second intervals throughout the test. The arterial oxygen saturation (as determined by the pulse oximeter) as well as heart rate were recorded at the end of every minute.

Body Composition Assessment

Body composition was measured by hydrostatic weighing to determine body density. At least five to six underwater trials were performed, with the average of the three highest values within 100 grams of each other being used as the underwater weight (Bonge & Donnelly, 1989). Vital capacity was determined using a Collins spirometer (220 Wood Road, Braintree, Massachusetts 02184). The subjects were given three trials with the highest of the three taken as the actual value (Craig & Ware, 1967). Residual lung volumes were estimated as being 24 percent of the vital capacity (Wilmore, 1969). Body density was computed using the equation of Goldman & Buskirk (1961) and percent body fat calculated using the equation of Siri (1961).

STATISTICAL PROCEDURES

The statistical analysis employed a Student's paired t - statistic. An alpha level of 0.05 was accepted as the criterion for statistical significance. The physiological data were loaded onto a Lotus software package and then transferred to STATGRAPHICS 3.0 software (Statistical Graphics Corporation, Rockville, Maryland, 1987).

CHAPTER IV

RESULTS AND DISCUSSION

The intent of this investigation was to examine the differences in arterial oxygen saturation (SaO_2) during the performance of a maximal oxygen consumption test, serum hemoglobin (Hb) concentrations, and maximal oxygen consumption ($\dot{V}\text{O}_{2\text{max}}$) after a three week period of carbonated beverage consumption as compared to after three weeks of consuming no carbonated beverages. The organization of the chapter is: a) results, and b) discussion.

Results

The results of the study are presented in the following sections: a) description of carbonated beverage consumption, b) description of subjects, c) SaO_2 during the performance of two $\dot{V}\text{O}_{2\text{max}}$ tests, d) Hb concentration changes over the two treatment periods, and e) $\dot{V}\text{O}_{2\text{max}}$ over the two treatment periods. Raw data for these variables are presented in Appendix D.

Carbonated Beverage Consumption

During the three week carbonated beverage treatment period, the subjects were told they could consume other carbonated beverages besides the 28 ounces provided. Only four of the nine subjects chose to consume excess carbonated beverages and recorded the type and quantity of beverage consumed on a report form which was given to them at the start of the treatment.

The five which consumed only the carbonated water provided drank a total of 588 ounces over the three week treatment period. The average excess carbonated beverages consumed by the other four subjects over three weeks was 4.30 ounces per day. Thus the average carbonated beverage consumption of the subjects in this study during the three week treatment period was 29.91 ounces per day.

Description of Subjects

Nine 19-24 year old ($M = 21.67$ years $SD \pm 1.87$ years) male triathletes and cyclists participated in the study. The subjects averaged 76.51 kg ($SD \pm 5.42$), and 11.4 ($SD \pm 1.6$) percent body fat. From the medical questionnaire, the subjects reported running an average of 19.88 miles per week, cycling an average of 84.4 miles per week, and swimming an average of 2.27 miles per week.

The data from the two tests of maximal oxygen consumption ($\dot{V}O_{2max}$), including values for SaO_2 and Hb, are presented in Table 1. The Student's paired t - statistic was used to compare SaO_2 , Hb, and $\dot{V}O_{2max}$ after three weeks of carbonated beverage consumption (C) and after three weeks of no carbonated beverage consumption (NC).

Arterial Oxygen Saturation (SaO_2) and Hemoglobin (Hb)

No significant differences in SaO_2 or Hb were found between the two treatment periods (Table 2). The data for SaO_2 and Hb are graphically presented in Figures 7 and 8 respectively.

Maximal Oxygen Consumption ($\dot{V}O_{2max}$)

The results of the student's t - statistic showed that there was no significant difference in $\dot{V}O_{2max}$ values as a result of consuming carbonated beverages (Table 2). The data are graphically represented in Figure 9.

Table 1

SaO₂, Hb, and, $\dot{V}O_{2\max}$ at the End of the Carbonated Beverage Treatment and the Non-Carbonated Beverage Period.

Subject	$\dot{V}O_{2\max}$ C (l·min ⁻¹)	$\dot{V}O_{2\max}$ NC	SaO ₂ C	SaO ₂ NC (percent)	Hb C	HbNC (g/dl)
1	4.60	4.91	94	94	12.94	12.95
2	5.13	5.06	95	95	13.93	14.78
3	4.32	4.44	94	93	13.19	14.30
4	4.22	4.42	95	93	14.00	14.61
5	4.82	4.49	92	92	14.11	14.09
6	4.41	4.21	97	93	14.58	14.15
7	4.72	4.87	94	93	13.33	13.66
8	5.04	5.02	92	93	13.63	14.61
9	<u>4.47</u>	<u>4.44</u>	<u>93</u>	<u>93</u>	<u>13.66</u>	<u>14.02</u>
<u>M</u>	4.63	4.65	94.0	93.2	13.71	14.13
<u>SD</u>	0.32	0.31	1.58	0.8	0.51	0.19

C = after three weeks of carbonated beverage consumption

NC = after three weeks of consuming no carbonated beverages

Table 2**Computed t - Statistic Results for SaO₂, Hb, and $\dot{V}O_{2max}$ (C,NC)**

	t	p
SaO ₂	1.305	0.210
Hb	1.668	0.115
$\dot{V}O_{2max}$	0.923	0.923

C = after three weeks of carbonated beverage consumption

NC = after three weeks of consuming no carbonated beverages

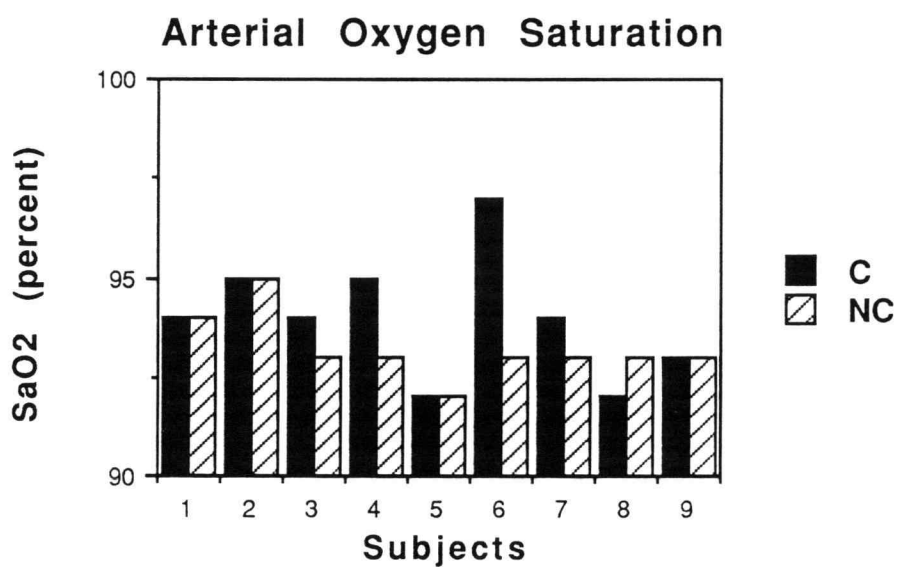


Figure 7. Arterial oxygen saturation levels at $\dot{V}O_{2\max}$ after three weeks of carbonated beverage consumption (C) and after three weeks of consuming no carbonated beverages (NC). There was no significant difference between the two treatment periods ($p>0.05$).

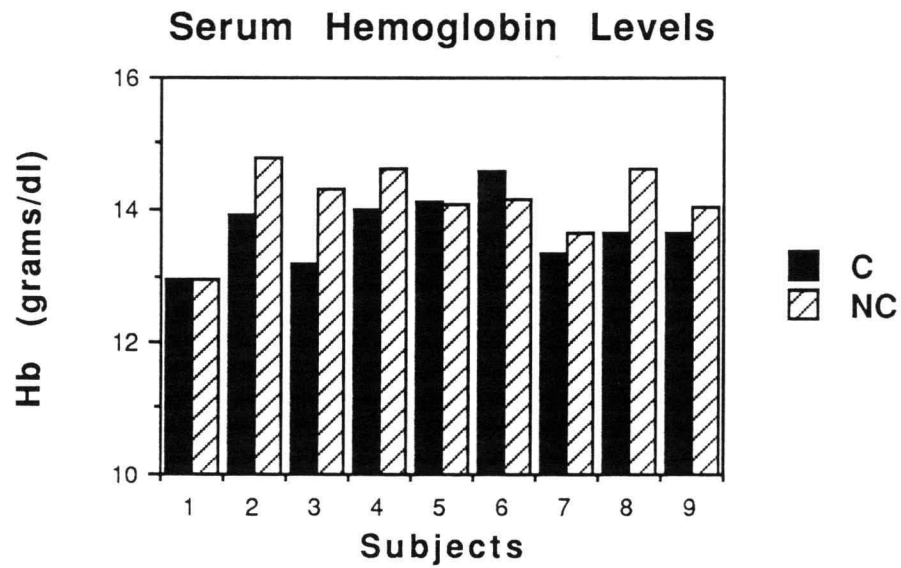


Figure 8. Serum hemoglobin levels after three weeks of carbonated beverage consumption (C) and after three weeks of consuming no carbonated beverages (NC). There was no significant difference between the two treatment periods ($p>0.05$).

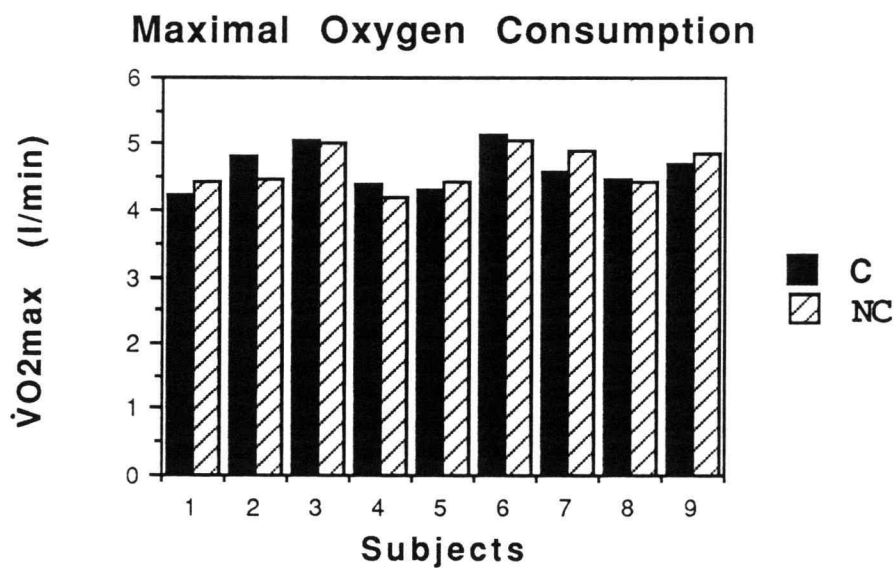


Figure 9. Maximal oxygen consumption after three weeks of carbonated beverage consumption (C) and after three weeks of consuming no carbonated beverages (NC). There was no significant difference between the two treatment periods ($p>0.05$).

Discussion

The discussion of the results will include: a) SaO₂, b) Hb, c) VO_{2max}, and d) summary. The discussion will analyze how the variables were affected by the three week period of carbonated beverage consumption as compared to three weeks of no carbonated beverage consumption.

In order to have an effect on any of the variables of this study, it is necessary that the excess CO₂ ingested from the carbonated beverages enters into the bloodstream. The absorption of CO₂ from the gastro-intestinal tract into the blood is defined by the laws of diffusion (McClellan, 1955). The average venous blood CO₂ tension of 52 mmHg probably limits the amount of CO₂ that enters into the blood. The remaining CO₂ in the digestive pathway is either belched or passed as flatus. Pogrund & Steggerda (1948) stated that if any CO₂ were to be absorbed into the blood, that equilibrium would be established in 90 minutes. Considering these factors, it appears unlikely that the chronic consumption of carbonated beverages would have any lingering effect on oxygen transport or aerobic performance.

SaO₂

The values obtained for SaO₂ were not different across the two treatment periods. The average SaO₂ values after the subjects consumed the carbonated beverages for three weeks showed a moderate upwards trend, but the differences were not statistically significant (Table 1).

Powers et al. (1988) demonstrated that highly trained aerobic athletes are capable of experiencing hypoxia during maximal exercise. The arterial oxygen saturation for some athletes was reduced from 98 percent at rest to 90 percent during maximal exercise. Powers et al. (1989) also showed an increase in VO_{2max} values in highly trained individuals when under hyperoxic conditions. These results demonstrate that a decrease in SaO₂ hinders the aerobic performance of highly trained athletes when near maximal workloads. The lability of carbon dioxide in body fluids suggested to McClellan (1955) that even if all of the carbon dioxide from a carbonated beverage transferred into the blood stream, there would be little or no alteration in the amount of CO₂ eliminated by the lungs. It could be hypothesized that consuming carbonated

beverages would put more CO₂ into the blood, and thereby lower SaO₂ by the Bohr effect. The lowering of SaO₂ would have to occur at the lungs in order to have an effect on $\dot{V}O_{2max}$. The results of this study showed that SaO₂ was not affected by the consumption of carbonated beverages.

Hb

There were no differences observed between the hemoglobin values at the end of the two treatment periods. The average value after consuming carbonated beverages for three weeks was 13.71 g/dl as compared to 14.13 g/dl after three weeks of consuming no carbonated beverages (Table 1). Hemoglobin is an important molecule in the discussion of oxygen transport. The consumption of carbonated beverages could alter the absorption of iron from a meal and thereby decrease the amount of hemoglobin available for the transport of oxygen. However, Hallberg & Rossander (1982) investigated the effects of different drinks on the absorption of non-heme iron from a composite meal and found that the carbonated beverages tested actually tended to improve the absorption of iron.

The hemoglobin molecule contains a protein (globin) portion which carries CO₂, and an iron portion, which carries the O₂. This chemical structure allows for both CO₂ and O₂ to be transported simultaneously (Solomon & Davis, 1983). If the consumption of carbonated beverages were to increase the quantity of CO₂ in the blood, it would not effect the O₂ carrying capacity because of the chemical structure whereby both gases can be transported on different portions of the hemoglobin molecule.

The subjects in this experiment displayed lower serum hemoglobin concentrations (13.98 g/dl) as compared to the average male (15-16 g/dl) (McArdle, 1985), but this is to be expected with the typical increase in plasma volume seen in elite aerobic athletes (Lamb, 1984).

$\dot{V}O_{2max}$

There were no differences observed in $\dot{V}O_{2max}$ values over the two treatment periods. The average $\dot{V}O_{2max}$ values of 4.63 l·min⁻¹ (C) vs 4.65 l·min⁻¹ (NC) were almost identical (Table 1). The test-retest reliability of maximal oxygen

consumption has been shown to be 0.95 (Noble, 1986) and the standard error as high as eight percent in aerobically trained males (Clear & Frisch, 1984).

The four factors which cause the oxyhemoglobin dissociation curve to shift are: a) blood temperature, b) the partial pressure of carbon dioxide (PCO_2), c) pH, and d) 2,3-DPG. The ingestion of carbonated beverages could cause two of the four factors to be altered. A decrease in blood pH and an increase in PCO_2 from the ingestion of carbonated beverages could cause the dissociation curve to shift to the right.

The excess CO_2 consumed with the carbonated beverage could upset the acid-base balance of the blood. The CO_2 produced by the muscle tissues, plus the CO_2 consumed with the carbonated beverage, could cause the pH to be substantially lowered and thereby allow the dissociation curve to shift to the right. However, no change was seen in VO_{2max} values, perhaps because of the shape of the dissociation curve. A shift to the right allows more oxygen to be released to the active tissues, while at the lungs, still almost complete saturation of arterial blood.

Blood pH is partially regulated by ventilation. During maximal exercise, ventilation helps establish an environment where the excess acid can be buffered and, in turn, prevent blood pH levels from dropping too dramatically. If the consumption of carbonated beverages caused more CO_2 to be present in the blood, then one would expect a decrease in maximal oxygen consumption. Ventilation would not be adequate to buffer the CO_2 produced by the tissues as well as the CO_2 from the carbonated beverages. In cockerals subjected to heat stress, Bottje & Harrison (1985) found that respiration rate was lower in the birds infused with carbonated water as compared to birds infused with tap water. Neither the quantity of ambient air inhaled or the respiration rate were variables of this study, but again, there were no changes in VO_{2max} values over the two treatment periods, suggesting that the extent of carbonated beverage consumption that occurred in this study does not impair the many factors relating to maximal aerobic capacity.

Summary

The consumption of carbonated beverages had no effect on VO_{2max} , SaO_2 , or Hb. The CO_2 ingested with a carbonated beverage was not of sufficient

quantity to establish a partial pressure of carbon dioxide in the digestive tract which would allow much CO_2 to enter into the blood stream, to effect the acid-base balance of the blood, or alter the oxygen-carrying capacity of blood. Hemoglobin, one of the principal factors of oxygen transport in the blood, was also not affected by the ingestion of carbonated beverages.

CHAPTER V

SUMMARY, CONCLUSIONS, AND RECOMMENDATIONS

Summary

The purpose of this study was to determine the differences in arterial oxygen saturation (SaO_2), serum hemoglobin (Hb) concentration, and maximal oxygen consumption ($\text{VO}_{2\text{max}}$) after three weeks of consuming 28 ounces of carbonated water per day as compared to three weeks of consuming no carbonated beverages. Nine male cyclists and triathletes served as subjects, with a mean age of 21.67 (SD \pm 1.87) years, weight of 76.51 kg (SD \pm 5.42), and body fat of 11.4 percent (SD \pm 1.6). All subjects were competitive regionally, had at least two years of racing experience, and were healthy and actively training during the course of the study.

The subjects were randomly assigned to either: a) drink 28 ounces of carbonated water per day, or b) drink no carbonated beverages for three weeks. The subjects reported to the laboratory to have a blood sample taken for later determination of Hb concentration and then performed a $\text{VO}_{2\text{max}}$ test while SaO_2 was being monitored. For the next three weeks, the subjects crossed-over their treatment regimen and then returned to the laboratory for the second determination of SaO_2 , Hb, and $\text{VO}_{2\text{max}}$.

The Student's t - statistic was used to compare SaO_2 , Hb, and $\text{VO}_{2\text{max}}$. No significant differences were found between the carbonated period (C) and the noncarbonated period (NC) in SaO_2 (94.00 vs 93.22 %, $p= 0.21$), Hb (13.71 vs 14.12 g/dl, $p= 0.11$), and $\text{VO}_{2\text{max}}$ (4.63 vs 4.65 $\text{l}\cdot\text{min}^{-1}$, $p= 0.92$). Carbonated water appears to have no effect on aerobic performance with respect to the oxygen carrying capacity of blood as seen by the absence of differences in SaO_2 , Hb, and $\text{VO}_{2\text{max}}$.

Conclusions

From the experimental design implemented, the following conclusions can be made:

1. Moderate quantities of carbonated water does not affect arterial oxygen saturation (SaO_2).
2. Hemoglobin concentrations (Hb) are not affected by the consumption of carbonated water.
3. Carbonated water does not affect maximal aerobic capacity as seen by the lack of change in maximal oxygen consumption ($\text{VO}_{2\text{max}}$).

Recommendations

Considering that no studies were found which investigated the effect of carbonated beverages on oxygen transport, it was difficult to determine the most appropriate experimental design. Future research could possibly include the following:

1. Considering the body's ability to maintain an acid-base balance, analyze the effect on these variables after the acute ingestion of a carbonated beverage.
2. Instead of using maximal oxygen consumption as the determining variable of performance, investigate the effect of carbonated beverages on the ventilatory threshold.
3. Determine blood pH and lactate levels before, during, and after the test of maximal oxygen consumption.
4. Investigate the effect of carbonated beverages other than carbonated water.
5. Considering the small changes seen in the variable of this study, use the direct determinate of arterial and venous oxygen saturation.
6. Again, considering the small changes seen in the indirect measurement of arterial oxygen saturation and the variability of maximal oxygen consumption from trial to trial and from subject to subject, it is necessary to use more subjects.
7. Test the effects of carbonated beverages on performance variables such as one mile race times or time to fatigue at 75 or 90 percent of $\text{VO}_{2\text{max}}$.

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APPENDICES

APPENDIX A
MEDICAL QUESTIONNAIRE

MEDICAL QUESTIONNAIRE

Name _____ Date _____

Address _____

Age _____ Phone (w) _____ (h) _____

Weight (lbs.) _____ Height (ft.-in.) _____ Gender _____

CIRCLE THE APPROPRIATE RESPONSES :

1) Have you smoked cigarettes in the last five (5) years ?

yes no

2) Have you ever been treated for high blood pressure ?

never in a physician's office in the hospital

3) Have you ever been treated for "sugar diabetes" (diabetes mellitus) ?

never with pills with insulin injections

4) Did either of your parents have a "heart attack" or bypass surgery ?

do not know neither one both

5) Have you ever had an elevated blood cholesterol level ?

do not know no yes

6) Do you know your blood cholesterol level ?

yes _____ mg/100ml blood no

7) Are you taking any prescription medications ? If yes, please list the kind, the amount, and how long you have been on this medication.

8) Describe your exercise habits (miles per week).

bike : _____

swim : _____

run : _____

anything else ? _____

9) Please quantify how much carbonated beverages you consume per day. Under each category of beverage, indicate the volume you consume per day in ounces or quarts (32 ounces). The usual volume of cans and bottles are 12 or 16 ounces, soft drink cup sizes are usually 12, 16, 20, 32 ounces, while two (2) liters is slightly more than 2 quarts.

soft drinks

beer

carbonated water drinks
(eg. Perrier, Calistoga, Koala)

APPENDIX B
STATEMENT OF INFORMED CONSENT FOR
PARTICIPATION IN RESEARCH STUDY

CONSENT FORM

Title : Arterial oxygen saturation during maximal exercise after a period of carbonated beverage consumption.

Investigator : Max Waibler

Purpose : To determine if the consumption of 32 ounces of carbonated beverages a day affects the percent saturation of hemoglobin with oxygen during maximal exercise.

I have received an oral explanation of the current study and understand that they consist of the following :

All testing will be done at the Human Performance Lab in the Women's Building at Oregon State University. The experiment will require four lab visits over a 6 week period. Three of the lab visits will be an aerobic capacity test and the other will be a test of body composition. After the initial aerobic capacity test, I will be randomly assigned to one of two groups: a) group 1 - consume 32 ounces of carbonated water per day or b) group 2 - refrain from drinking any carbonated beverages. After three weeks, I will perform a second aerobic capacity test. Another three week period will follow where group 1 will abstain from consuming carbonated beverages and group 2 will consume 32 ounces of carbonated water per day. A final aerobic test will follow the cross-over treatment period. Each lab visit will take 30-40 minutes.

1. Test of maximal oxygen consumption. The test will be conducted on a cycle ergometer and will begin at a light workload and progressively increase in intensity every minute until I am too fatigued to continue. The expected time duration of the test is 8-12 minutes. The effort put forth at the highest intensity of the exercise is equal to a one mile cycling race. During the exercise test I will be breathing through a mouthpiece so that the amount of oxygen I am using can be determined , and I will have a pulse oximeter taped to my finger, so that the amount of oxygen bound to hemoglobin can be determined. Prior to the exercise test, a 5 ml (5cc) blood sample will be taken from a forearm vein for the determination of my blood iron content.
2. Hydrostatic weighing to determine the percent body fat. This test consists of sitting in a tank of warm water (97°F) on a chair suspended from a scale. Near the end of a maximal exhale, I will tuck my head under the water and hold my breath for 2-3 seconds as the scale is being read. I will repeat this procedure for a total of 6-8 trials so a consistent reading can be obtained. I will also perform a test to determine my residual volume (the air in my lungs at the end of a maximal exhalation), which entails breathing into a spirometer filled with pure oxygen for a period of 30-60 seconds. Two-to-three trials will be performed.

I understand that the test of maximal oxygen consumption has a chance of precipitating a cardiac event (such as abnormal heart rhythms) or even death. However, the possibility of such an occurrence is slight (less than 1 in 10,000), since I am in good physical condition with no known symptoms of heart disease, and since the test will be administered by trained personnel who will be monitoring electrocardiographic and other physiological responses to the test.

The benefits of my participation in the study include contributing to the scientific study of the effect of carbonated beverages by endurance athletes and obtaining knowledge concerning my aerobic capacity and body composition.

I understand that the results of my participation in the study will remain confidential and that I will not be identified in any way in the presentation or publication of the findings of this investigation.

I am aware that the blood sampling procedure follows standard, hygienic practices for blood withdrawal and may leave a bruise at the site of needle insertion.

Persons at increased risk for Hepatitis B or HIV (commonly called AIDS) should not donate blood or any other body fluids and therefore should not participate in this investigation. Persons at increased risk include men who have sexual contact with another man since 1977, persons who have used intravenous drugs, Haitian immigrants, and persons who have had sexual contact with either a member of one of these groups or a person who has AIDS.

I have been completely informed of and understand the nature and purpose of this research. The researchers have offered to answer any questions that I may have. I understand that my participation in this study is completely voluntary and that I may withdraw from the study at any time. Questions about the research or any aspects of my participation in it should be directed to Max Waibler (phone # 737-6789). I understand that Oregon State University does not provide a research subject with compensation or medical treatment in the event the subject is injured as a result of participation in the research project.

I have read the foregoing and agree to participate in this study.

Subject's Signature

Date

Subject's Address

Investigator's Signature

Date

APPENDIX C
REPORT FORM

APPENDIX D

RAW DATA

Subject Description

Subject	Age	Weight (kg)	% Fat
1	23	70.5	10.1
2	24	77.6	9.9
3	20	73.0	11.4
4	21	70.5	10.0
5	24	83.5	12.4
6	20	72.5	14.6
7	23	80.0	10.1
8	19	76.0	11.3
9	21	85.0	12.7

Arterial Oxygen Saturation (SaO₂)

Subject	SaO ₂ after C	SaO ₂ after NC
1	94	94
2	95	95
3	94	93
4	95	93
5	92	92
6	97	93
7	94	93
8	92	93
9	93	93

C - after three weeks of carbonated beverages consumption

NC - after three weeks of consuming no carbonated beverages

mean - SaO₂C = 94 (SD= 1.58) SaO₂NC = 93.22 (SD= 0.83)

Serum Hemoglobin Levels (Hb)

Subject	Hb after C	Hb after NC
1	12.94	12.95
2	13.925	14.78
3	13.19	14.295
4	14.00	14.61
5	14.11	14.085
6	14.58	14.15
7	13.33	13.66
8	13.63	14.61
9	13.66	14.02

C - after three weeks of carbonated beverages consumption

NC - after three weeks of consuming no carbonated beverages

mean - HbC = 13.71 (SD= 0.51)

HbNC = 14.13 (SD= 0.56)

Tests of Maximal Oxygen Consumption (VO_{2max})

Subject	VO_{2max} after C	VO_{2max} after NC
1	4.60	4.91
2	5.13	5.06
3	4.32	4.44
4	4.22	4.42
5	4.82	4.49
6	4.41	4.21
7	4.72	4.87
8	5.04	5.02
9	4.47	4.44

C - after three weeks of carbonated beverages consumption

NC - after three weeks of consuming no carbonated beverages

mean - $VO_{2max}C$ = 4.64 (SD= 0.32)

$VO_{2max}NC$ = 4.65 (SD= 0.31)