Study of physiological responses to acute carbon monoxide exposure with a human patient simulator

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Cesari, Whitney A., Dominique M. Caruso, Enela L. Zyka, Stuart T. Schroff, Charles H. Evans, Jr., and Jon-Philippe K. Hyatt. Study of physiological responses to acute carbon monoxide exposure with a human patient simulator. Adv Physiol Educ 30: 242–247, 2006; doi:10.1152/advan.00063.2006.—Human patient simulators are widely used to train health professionals and students in a clinical setting, but they also can be used to enhance physiology education in a laboratory setting. Our course incorporates the human patient simulator for experiential learning in which undergraduate university juniors and seniors are instructed to design, conduct, and present (orally and in written form) their project testing physiological adaptation to an extreme environment. This article is a student report on the physiological response to acute carbon monoxide exposure in a simulated healthy adult male and a coal miner and represents how 1) human patient simulators can be used in a nonclinical way for experiential hypothesis testing; 2) students can transition from traditional textbook learning to practical application of their knowledge; and 3) student-initiated group investigation drives critical thought. While the course instructors remain available for consultation throughout the project, the relatively unstructured framework of the assignment drives the students to create an experiment independently, troubleshoot problems, and interpret the results. The only stipulation of the project is that the students must generate an experiment that is physiologically realistic and that requires them to search out and incorporate appropriate data from primary scientific literature. In this context, the human patient simulator is a viable educational tool for teaching integrative physiology in a laboratory environment by bridging textual information with experiential investigation.

Simulation; physiological adaptation; mining; chronic obstructive pulmonary disease; coal workers’ pneumoconiosis

THE HUMAN PATIENT SIMULATOR (HPS) is a sophisticated integration of software and hardware built around an interactive human mannequin and is traditionally used for clinical training of doctors, nurses, or other health professionals (J. P. K. Hyatt and C. H. Evans, Jr., unpublished observations). The HPS, however, can be utilized as an experiential tool for undergraduate education, specifically as the laboratory component of a course exploring physiological adaptation to extreme environments (2). The HPS breathes, has pulses, and can respond to intravenous drugs, cardiopulmonary resuscitative measures, defibrillation, intubation, ventilation, catheterization, and many other medical procedures (12, 14). These attributes of the HPS can be manipulated to simulate the bodily response to extreme environments. For example, to simulate high altitude in the mountains, students can decrease the oxygen concentration of the HPS’s computerized arterial system. Other examples of simulated environments include open cockpit ballooning, occupational mining, free diving, exercise, extreme heat and cold, and dehydration. The purpose of the laboratory is for students to investigate a hypothesis on human physiology in a simulated extreme environment. The HPS replaces actual environments that may be physically challenging to obtain; it also eliminates the need for the appropriate equipment to make such physiological measurements (2). In many instances, the students will compare the response of a simulated healthy with a disease-compromised individual to an extreme environment where such individuals in real life might well venture. The preprogrammed simulated “patients” combined with the realistic physiological and pharmacological reactions of a human being written into the simulator’s software allow for an effective laboratory experience. More traditional educational approaches of the HPS in medical patient-oriented education have been described elsewhere (11, 12).

The course instructors (S. T. Schroff, C. H. Evans, Jr., and J.-P. K. Hyatt) have successfully employed the METI HPS during the past several years in the “Physiological Adaptation to Spaceflight and Other Extreme Environments” course at Georgetown University to allow students to test experiments of their own design to study physiological responses to stressful environments. This assignment challenges students in a nontraditional laboratory environment by eliminating experiments with predictable outcomes or “cookbook” methodology. Groups of two to four students are asked to generate a testable hypothesis using the HPS and their chosen environment. Once the projects have been approved by the instructors, the students acquire data from the primary literature reporting on the response of the human body to a particular environment. These data are then used to program the HPS, which simulates the environment. The students use these starting parameters to test control and experimental groups, acquire data, and then interpret the results. Importantly, the students observe how changes in one physiological system affect another; the integrative nature of the human body is better understood by the students when the outcomes to their experiments are unknown. To make the data meaningful, the students must have an understanding of how the physiology is responding to their manipulations.

This article describes an investigation designed and conducted by students (W. A. Cesari, D. M. Caruso, and E. L. Zyka) to assess the physiological response to an acute carbon monoxide (CO) occupational exposure. The students’ basis for the experimental study was the January 2006 explosion in a coal mine in Sago, WV, that resulted in a release of CO gas as

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Simulated Response to Acute CO Exposure

During the 20th century, >125,000 mining-related deaths were recorded in the United States (3). Explosives used in mining and construction in the United States are well known sources of CO poisoning (7), as is methane gas, a natural and dangerous contaminant in mining environments. Mines can contain dangerous levels of different gases, some of which are combustible (methane), so adequate ventilation of mines is vital to provide a safe environment (13). The National Institute of Safety and Health (NIOSH) recommends an exposure limit of 35 ppm for CO, measured as a time-weighted average for up to a 10-h workday during a 40-h work week; the ceiling concentration, not to be exceeded during any part of the workday, is 200 ppm (3).

Unlike other gases present in mines, CO directly causes toxicity by combining with hemoglobin (Hb) and preventing the binding of oxygen (13). Hb’s affinity for CO is 200 times greater than its affinity for oxygen (3). Consequently, a small amount of CO dramatically reduces Hb’s ability to transport oxygen. When inspired air contains CO levels as low as 0.02%, headache and nausea may ensue; if the CO concentration is increased to 0.1%, unconsciousness and often death follow (3).

Mining is associated with several occupational health risks, with one risk being prolonged exposure to respirable coal mine dust over a working lifetime (3). Such exposures are associated with the development of occupational respiratory diseases, including simple coal workers’ pneumoconiosis (CWP) and chronic obstructive pulmonary disease (COPD). CWP is a lung disease caused by inhaling coal mine dust. Computed tomography scans have shown that micronodules form in the lung, leading to ventilation deficiencies such as increased residual volume. COPD is a chronic airflow obstruction that causes the collapse of small airways and decreases the rate of airflow through the lungs upon exhalation (3) and presents very similarly to CWP. Increased residual volume and decreased tidal volume (TV) are signs of COPD.

Because the health status of a coal miner can be greatly impaired due to chronic CO exposure and ventilation impairments, the questions and hypothesis in the present study of simulated acute high concentration CO exposure were constructed accordingly. The simulation was based upon the following questions: What is the physiological response to acute CO exposure in an otherwise healthy person? Does a coal miner’s everyday exposure to baseline levels of CO alter his ability to adapt to extreme levels of CO? The hypothesis, therefore, is: If both a healthy man and an experienced coal miner are exposed to acute CO poisoning, then hypoxia and eventual death will result in both groups. The simulated healthy individual should survive longer in CO conditions because coal miners have a previously compromised oxygen-carrying capacity that will less effectively respond to the respiratory stress induced by CO.

The hypothesis reflects observations concerning the response to respiratory stress in patients with COPD. Often, COPD patients present symptoms that are rarely seen in healthy individuals, with breathlessness being the most common (4). Other symptoms include nausea, lightheadedness, dizziness, headache, and chest or neck pain (4). COPD patients appear to experience more problems upon exposure to the simplest forms of respiratory stress such as exercise. Therefore, it is expected that a HPS coal miner with simulated CWP/COPD would experience even greater difficulties than a simulated healthy male during acute CO exposure, the latter of which would cause more severe stress than CWP/COPD alone.

Methods

Human simulation. This investigation was designed and conducted in fulfillment of the course “Physiological Adaptation to Spaceflight and Other Extreme Environments” in the Human Science Curriculum of the School of Nursing and Health Studies at Georgetown University in Washington, DC. The human simulation data were obtained using the METI (version 6) HPS (Medical Education Technologies, Sarasota, FL) located in the Human Simulation Center within the School of Nursing and Health Studies.

The goal of this study was to examine the response to acute hypoxia resulting from CO exposure. To test this, we simulated two subjects and examined the length of time before physiological failure was observed. Failure was defined as the emergence of cardiac arrhythmias, e.g., ventricular fibrillation, ultimately leading to simulated death. This simulation thus may provide more insight on the rapidity of CO toxicity and which physiological systems are compromised due to exposure to this gas.

The first subject studied was emulated using the METI HPS “Standard Man” profile representing a healthy male adult (Table 1). The second subject (a coal miner) was also derived from the METI HPS profile of the “Standard Man,” where minor changes in the programming were made to emulate a coal miner as described below.

To simulate an environment with variable levels of CO, the NIOSH recommendation of exposure to 35 ppm CO was used as the baseline, and a maximum exposure of 1,300 ppm CO was chosen since it was the value measured in the mine after the West Virginia explosion. It is important to note that no measurements were taken in between the baseline and maximum levels of CO in the West Virginia mine and that we are assuming the preexplosion CO level in the mine was 35 ppm. Therefore, intermediate levels of CO were also assumed to have risen in a linear fashion in the mine and during the present simulation. Importantly, this simulation only focused on the physiological affects of CO exposure: no other physiological stressors in the mine (i.e., high-pressure environment, darkness, physical labor, and long shift hours) were taken into account. As such, the prescribed simulation conditions may limit conclusions as they pertain to the effects of mining environments per se on physiological systems.

Simulating CO exposure. Ideally, direct CO delivery to the HPS through a respirator would have been preferred. Because CO is not included in the HPS ventilatory delivery of anesthetic gases, the HPS’s computer programs were altered by the investigators to simulate rising CO levels from baseline (35 ppm) to maximum (1,300 ppm) levels. Simulated blood characteristics were altered by changing the HPS’s hematocrit and blood volume values. Hematocrit identifies the potential for oxygen delivery within the body, and by incrementally lowering this parameter during the experiment, that potential is decreased. However, the HPS’s lowest programmed limit for hematocrit is 5%. Therefore, to approach 0%, which simulates 100% blood CO saturation, plasma volume was also changed as described below.

To alter the hematocrit to reflect changes in CO in the inspired air (CO_in) as the mine becomes more concentrated with CO, the relationship between inspired air and blood CO was established using Eq. 1, which was based on data collected from an experiment by Woehlick et al. (15). In that study, patients were given a breathalyzer test.
Table 1. Healthy male profile

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Shown is the simulated healthy male preprogrammed profile in the METI human patient simulator used to examine physiological responses to acute carbon monoxide exposure. This profile was also used for the simulated coal miner, with alterations to functional residual capacity and tidal volume to simulate chronic obstructive pulmonary disease (COPD) and coal workers’ pneumoconiosis, as described in METHODS. SpO2, spot oxygen saturation.

measuring CO; simultaneously, tests were administered to measure the CO content in the blood. The relationship between breath CO (CObreath) and blood CO (COHb) can be calculated from the following:

$$\text{COHb} = 2.633 + 0.1531 \times \text{CObreath} \quad (1)$$

Using this mathematical relationship, the starting (35 ppm) and ending (1,300 ppm) CO values translate into carboxyHb levels of 8.05% and 201.6%, respectively. Because no more than 100% of the blood’s Hb can be bound to CO, the maximum calculated value (201.6%) was capped at 100% in the present HPS investigation (Fig. 1A).

Next, the duration of the simulation was established. The simulated CO increase from 8.05% to 100% was set to occur over a 3-min period. This time course was chosen based on COair measurements obtained following a controlled kerosene stove explosion (9). Determining COair at any time during the 3-min simulation (Fig. 1B) can then be calculated from the following:

$$\text{CO} = 1.07 \times \text{time (min)} + 8.05 \quad (2)$$

Finally, the spot oxygen saturation (SpO2), denoting the percentage of Hb bound to oxygen, was also explicitly changed. Preliminary HPS investigations altering only hematocrit did not affect the HPS’s physiological responses; there was a change in ischemic index, but other physiological parameters (heart rate and respiration rate) remained unaffected. Altering both the hematocrit and SpO2, however, demonstrated that acute CO toxicity could be emulated. Simulating changes in COair as it was reflected in the blood was accomplished by changing SpO2 and was calculated based on the values obtained from Eq. 1. To simulate starting (35 ppm) and ending (1,300 ppm) COair, 8.05% and 100% COHb was entered into Eq. 3 (8) as follows:

$$\text{SpO2} = 99.3 - (\text{COHb} \times 0.1) \quad (3)$$

This resulted in initial and final SpO2 values of 98.5% (35 ppm CO) and 89.3% (1,300 ppm CO), respectively. Thus, to adequately simulate CO exposure, the HPS was programmed to decrease initial and final hematocrit and SpO2 values to these levels over a 3-min period.

Acute CO toxicity in a simulated healthy adult male. The starting preprogrammed hematocrit (42.3%) was first lowered to 39% by replacing 525 ml of blood with plasma to simulate a COair of 35 ppm at time 0. During the 3-min simulation, blood was incrementally replaced by plasma until the simulator’s lowest possible hematocrit (5%) was achieved. The replacement of blood with plasma was programmed to occur evenly from time 0 until 5% hematocrit was reached. Although the METI program does not permit a hematocrit of <5% to be attained by selectively changing the hematocrit value in the program, a lower HPS hematocrit was achieved by diluting the blood with plasma; thus, a 15,000-ml volume of plasma was added when the hematocrit reached 5% and resulted in a hematocrit near 0%. Clearly, blood volume cannot physiologically increase to 15,000 ml, but these changes were necessary for the purpose of the simulations. At this point in the experiment, preliminary trials were performed to examine the simulator’s response to this large plasma volume increase. There were no physiological changes indicative of pulmonary

![Fig. 1. Derivation of acute carbon monoxide (CO) exposure used in the present human patient simulation (HPS). To simulate an environment with varying CO levels, the relationship between CO concentration in the blood (COHb) and breath (COair) (A) and COair as a function of time (B) were used. A: relationship between COair and COHb derived from Eq. 1 (see text). The relationship was derived only from initial (35 ppm CO) and final (1,300 ppm CO) environmental levels (see text), which accounts for the displayed perfect linearity associated with CO levels in the air and blood. B: simulated COair can be ascertained at any time point during the present simulation as derived from Eq. 2 (see text).](http://advan.physiology.org/)
edema or heart failure in either the control (healthy male) or experimental (coal miner) conditions. Thus, the final adjustment to the patient profiles was to alter the $S_{\text{R}}$$_{O_2}$. The $S_{\text{R}}$$_{O_2}$ also was programmed to decrease from 98.5% (initial) to 89.3% (final) over 3 min. Considering that the hematocrit and $S_{\text{R}}$$_{O_2}$ were altered, the $S_{\text{R}}$$_{O_2}$ values must be interpreted differently: $S_{\text{R}}$$_{O_2}$ values between 89% and 98% by themselves would appear to be high in the presence of a greatly diminished hematocrit. However, this represents the saturation of incrementally fewer red blood cells from time 0 to 3 min. For instance, at 3 min, a $S_{\text{R}}$$_{O_2}$ equal to 89% indicates that only 89% of the very few remaining red blood cells have oxygen bound to Hb. Thus, lowering hematocrit and $S_{\text{R}}$$_{O_2}$ values and increasing plasma volume were performed to achieve a hematocrit of ~0%, which simulates ~100% CO$_{Hb}$ saturation.

**Acute CO toxicity in a simulated coal miner with CWP and COPD.**

The coal miner simulation was accomplished by modifying the HPS values for the healthy adult male as described above. Specifically, the healthy male programmed set points for functional residual capacity (FRC) and TV were altered to reflect signs of CWP and COPD. The HPS has a default FRC setting of 2,000 ml. The FRC was increased from 2,000 to 2,800 ml to reflect the trapping of air in the lungs as a person with CWP/CWP exhales. The 2,800-ml value corresponds to a moderate case of COPD or CWP. The HPS also has a preprogrammed default TV setting of 700 ml (1). For the simulations, the TV was decreased from 700 to 500 ml to reflect the collapse of small airways, which impedes the movement of air in and out of the lungs. The 500-ml value corresponds to a moderate case of COPD or CWP (1). Altering FRC and TV parameters caused other parameters such as respiratory rate to vary in the manner expected in patients with COPD and CWP, which occurred due to the HPS’s integrative programming. Other than these described changes, the coal miner and healthy adult male simulations were performed using the preset METI physiological values.

**Statistics.** The simulations for both the healthy adult male and coal miner were performed in triplicate. For each criterion measured ($S_{\text{R}}$$_{O_2}$, heart rate, and respiration rate), a Pearson correlation coefficient ($r$; Microsoft Excel) was conducted to determine the degree of similarity of each independent experiment performed in triplicate on each of the two simulated individuals.

**RESULTS AND DISCUSSION**

Although many simulated physiological functions were recorded during each experiment, only $S_{\text{R}}$$_{O_2}$ (Fig. 2), heart rate (Fig. 3), and respiration rate (Fig. 4) are shown. In all instances, the other physiological parameters did not exhibit a significant change. The three independent simulation experiments for the control (healthy adult male) and experimental (coal miner) conditions were strongly correlated for $S_{\text{R}}$$_{O_2}$ (control: $r = 0.89$; experimental: $r = 0.99$), heart rate (control: $r = 0.71$; experimental: $r = 0.86$), and respiration rate (control: $r = 0.87$; experimental: $r = 0.89$). These $r$ values indicate that the results attained for these parameters are replicable.

As predicted, both the simulated healthy male and coal worker expired as a result of hypoxia resulting from acute CO exposure. Contrary to the hypothesis, however, both the simulated healthy male and coal miner reached this end point simultaneously. Time to failure, as defined by the onset of cardiac arrhythmias, occurred at ~215 s after incremental increases of CO exposure were initiated at time 0. However, the coal miner exhibited failure at a lower $S_{\text{R}}$$_{O_2}$ than the simulated healthy male. The coal miner consistently decompensated at a $S_{\text{R}}$$_{O_2}$ of ~86%, whereas the healthy male decompensated at a $S_{\text{R}}$$_{O_2}$ of ~89% (Fig. 2).

**Fig. 2.** Representative spot oxygen saturation ($S_{\text{R}}$$_{O_2}$) changes during a 3-min experiment with the METI HPS simulating acute CO exposure in a healthy adult male (a) and a coal miner (b). The simulated CO increase over time shows incremental decreases in $S_{\text{R}}$$_{O_2}$, reflecting impaired blood oxygen saturation. The $S_{\text{R}}$$_{O_2}$ indicates the percent saturation of simulated red blood cells.

Although the coal miner started out with a lower $S_{\text{R}}$$_{O_2}$, the rate at which his $S_{\text{R}}$$_{O_2}$ fell was faster than that of the healthy male. Therefore, the vertical gap before the acute increase in CO exposure was narrower than the vertical gap seen in Fig. 2 after the CO exposure. At ~200 s, there was an abrupt increase (~40 beats/min) in heart rate in both the control and experimental subjects. This acute change likely represents the period when each subject exhibited ventricular tachycardia, which was the defined point of physiological failure from CO toxicity. Similarly, cardiac decompensation in the simulated healthy male and coal miner occurred at the same time (Fig. 3). Figure 3 also shows that the coal miner decompensated at a lower $S_{\text{R}}$$_{O_2}$ compared with the healthy male. Respiration rate (Fig. 4) also followed a similar pattern as $S_{\text{R}}$$_{O_2}$ and heart rate. The lung impairments in the coal miner due to simulated CWP and COPD are reflected by the higher respiration rate throughout the simulation.

Both the shape and values of the heart rate curves for the simulated coal miner and healthy male appeared identical (Fig. 3), indicating that the coal miner most likely did not compensate with heart rate any differently than the healthy male. The shape of the respiratory rate curves for both the coal miner and healthy male appeared identical; however, the coal miner’s values were consistently higher than those of the healthy male (Fig. 4). The higher respiratory rate is not particularly significant; it can be attributed to the coal miner’s previously weakened respiratory system. The coal miner inhales and exhales more frequently than does the healthy male to compensate for the lesser volume of air that can be exchanged with each breath. Neither the heart rate (Fig. 3) nor respiratory rate (Fig. 4) data suggest that the coal miner had greater cardiovascular or respiratory compensation than the healthy male. Consequently, the coal miner may have employed the same compensatory mechanisms as the healthy male by increasing both
heart and respiratory rate but with more ease and efficiency, which may be, in part, due to the CWP/COPD in the coal miner.

The ability of the coal miner to compensate and sustain life, although briefly, at a lower SPO2 is consistent with the idea that persons who have previously been exposed to CO physiologically adapt to chronically low levels of oxygen saturation. Several questions stem from this statement, which is based on our experimental results. Are our other observed experimental data changes significant? If so, what do they convey about the nature of the ability of the coal miner to adapt versus that of a healthy male? To explore the possible reasons for the coal miner’s ability to sustain life at a lower SPO2, other simulated data factors were taken into consideration. By separately comparing the change in heart rate and then the change in respiratory rate over time between the coal miner and healthy male, it might become evident whether or not the coal miner was compensating to sustain life in some way that the healthy male was not.

COPD patients compensate for inflamed airways and reduced lung volume by hyperinflating the lungs, which generally occurs during periods of physical exertion. This is a possible explanation as to why the simulated coal miner failed at a lower SPO2 than the healthy male (10). It is possible that in the simulated CO environment, the coal miner’s hyperinflation mechanism may have been beneficial. Practically, the coal miner may more readily provide an adaptive response mechanism to CO toxicity than a healthy individual because he is already accustomed to responding to some degree of respiratory stress.

A simpler explanation for the difference in SPO2 at the time of decompensation for the healthy male and coal miner is that the HPS observed variation in our investigation is not important. As previously noted, SPO2 should not be viewed in the standard manner because the hematocrit was decreased. At the time of physiological failure (~215 s after the acute increase in CO), both the healthy male and coal miner had a hematocrit of 5.0%, which was greatly reduced by the simulated addition of 15,000 ml of plasma to most accurately simulate a hematocrit of 0%. Basically, very few effective red blood cells would have been present in both the simulated healthy male and coal miner because of the emulated carboxyHb at the time of decompensation. The SPO2 represents the numbers of these red blood cells that were bound to oxygen. The simulated coal miner and healthy male decompensated at a SPO2 of ~86% and ~89%, respectively. The ~3% difference between these values would have significance if the hematocrit had not also been lowered. The decrease in hematocrit, however, means that this 3% only represents a slight difference in the number of red blood cells bound to oxygen that were circulating. Both simulated subjects had so few red blood cells at the time of failure that a 3% difference represents a difference of only a few red blood cells. Perhaps the difference in SPO2 should not be regarded as physiologically important. Finally, it is also possible that the simulated healthy male and coal miner responded realistically to a drastically lowered (5%) hematocrit inasmuch as the warning signs to CO toxicity are so subtle that major physiological responses are unaffected.

An additional explanation to account for the observations in this simulation investigation is that perhaps the results of these experiments should not be discussed with clinical or adaptive explanations but rather by the act of simulation itself. The METI HPS was not originally programmed for extreme environments. Rather, the HPS was programmed for students to observe patient responses in a clinical setting. The methods by which the HPS was manipulated during the present investigation were drastic and unconventional in relation to the expected acute medical care-related manipulations: the hematocrit and SPO2 were decreased to levels that are rarely seen in a clinical setting, i.e., the hematocrit was incrementally decreased to 5% and 15,000 ml of plasma were added to obtain a ~0% hematocrit. It is possible that these settings, although necessary to simulate the extreme environment, were too extreme for the HPS to yield a realistic physiological response. The data shown in Figs. 2–4 also support this alternative conclusion. The heart rate (Fig. 3) and respiratory rate (Fig. 4) especially exhibited identical trends in two vital signs of both simulated subjects. This similarity might have occurred as a consequence of the

![Fig. 3](https://example.com/fig3.png)

**Fig. 3.** Representative changes in heart rate during a typical 3-min experiment with the METI HPS simulating acute CO exposure in a healthy adult male (□) and a coal miner (■). Changes in blood oxygen saturation triggered a commensurate rise in heart rate. The abrupt increase (~40 beats/min) in heart rate at ~215 s represents the period when each simulated subject exhibited cardiac arrhythmias.

![Fig. 4](https://example.com/fig4.png)

**Fig. 4.** Representative respiration rate changes during a typical 3-min experiment with the METI HPS simulating acute CO exposure in a healthy adult male (□) and a coal miner (■). Changes in blood oxygen saturation triggered a commensurate rise in heart rate and respiration rate.
HPS’s programming for acute clinical settings. Perhaps the HPS resorted to preprogrammed default equations to respond to the extreme parameters. Such equations in the clinical response preset programming would produce the similar responses observed in Figs. 2–4.

In conclusion, the comparative simulated responses to acute high-level CO exposure in a healthy male versus a coal miner with COPD generated data that are open to several possible interpretations. The first interpretation suggests a coal miner’s ability to employ certain compensatory physiological mechanisms with greater ease than that of the model healthy male. This adaptive response accounts for the coal miner’s ability to sustain life at a lower SpO2. The second line of reasoning proposes that the experimental data are not physiologically relative because of the nature of the simulation. The HPS was manipulated to respond to extremes for which it was not preprogrammed. Therefore, the simulated response may not be physiologically accurate. A third interpretation is that the results represent the extreme at the point of decompensation, where it is difficult to distinguish physiological and pathological responses. Future HPS investigation with intermediate simulated levels of CO exposure between 35 and 1,300 ppm are needed to determine the extent to which the preprogrammed acute medical care settings of this HPS in its present configuration are relative for the study of physiological adaptation to this particular extreme environment.

This project demonstrates undergraduate student potential when challenged with an assignment to formulate a hypothesis and carry out the appropriate methodology to best answer that research question with limited resources and time. It also shows a successful product from an assignment with few outlined restrictions by the instructors; yet, given this focused, but unstructured, framework, independent critical thought, group work and cohesiveness, and learning were accomplished that diverged from traditional didactic means. Furthermore, the presentation of each group’s findings in oral and written forms not only enhanced these skills but also provided a formal setting to the project and course. The study presented here exemplifies the complexity of some of these projects. Certainly, the results of this study may have had additional interpretations, and there are points raised in the RESULTS and DISCUSSION that could be challenged. Many of these criticisms could arise when scrutinizing the HPS alone or the actual physiological responses to high CO levels. Here, we present this student study relatively unaltered by the instructors (S. T. Schroff, C. H. Evans, Jr., and J.-P. K. Hyatt) to exemplify student performance on a difficult topic using an imperfect system (the HPS). Their hypothesis and incorporation of primary research literature was thoughtful and logical, the methodology was carefully constructed and physiologically realistic (per the instructions of the assignment), and the interpretation of the results was appropriate and arguably at the level of graduate students. Without question, this assignment received the highest marks from the instructors.

REFERENCES