PHYSIOLOGIC RESPONSES WHEN WORKING IN HOT ENVIRONMENTS

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Heat Exchange and Environmental Heat Stress

Heat stress refers to environmental and host conditions that tend to increase body temperature. Heat strain refers to physiological and or psychological consequences of heat stress.

Body heat exchange occurs by convection, radiation, conduction, and evaporation. Metabolic heat is released from active skeletal muscles and transferred from the body core to skin. Heat exchange from skin to the environment is influenced by air temperature; air humidity; wind speed; solar, sky, and ground radiation; and clothing.

Convection is heat transfer by moving a gas or liquid over the body, whether induced by thermal currents, body motion or natural movement of air or water. Heat loss by convection to air occurs when air temperature, in contact with skin, is below body temperature; conversely, heat gain by convection from air occurs when air temperature approaches or exceeds that of the body.

Heat loss by radiation occurs when surrounding objects have lower surface temperatures than the body, and heat gain by radiation (solar, sky, large objects and ground) occurs when surrounding objects have higher surface temperatures than body surface temperature. Accordingly, temperature combinations of the sky, ground and surrounding objects may exist which result in body heat gain due to radiation, even though the air temperature is below that of the body. Radiative heat exchange is independent of air motion.

Evaporative heat loss accounts for all body cooling when ambient temperatures are equal or above skin temperatures. Eccrine sweat glands secrete fluid onto the skin surface permitting evaporative cooling when liquid is converted to water vapor. When more body heat must be dissipated, these fluid losses increase due to heavier sweating, which may for short periods of time exceed 2 liters per hour. The rate of sweat evaporation depends upon air movement and the water vapor pressure gradient between the skin and the environment, so in still or moist air the sweat tends to collect on the skin. If sweat is not evaporated, the skin surface becomes soaked which suppresses sweat secretion. For this reason, it is important to allow air circulation to the skin, especially torso areas, to maximize evaporative cooling. Sweat that drips off the body or clothing provides no cooling benefit.

High air temperature, high humidity, thermal radiation, and low air movement are causes of environmental heat stress. Air temperature is measured from a shaded dry bulb thermometer. The contribution of humidity is determined from a wet bulb temperature, which is measured by covering a thermometer bulb with a wet wick. Aspirated wet bulb temperature is obtained when an air current is blown over the wick. Natural (unventilated) wet bulb temperature is obtained when natural airflow around the bulb is not augmented or restricted. Natural wet bulb temperature is higher than aspirated wet bulb temperature, particularly in still air; convergence begins at low air movements, with

the two becoming nearly the same at an air velocity of approximately 7 miles per hour (mph) (3.1 meters per second).

Physiologic Relationships

Body temperature is normally regulated with a narrow range through two parallel processes: behavioral temperature regulation and physiological temperature regulation. Behavioral thermoregulation includes seeking shade, slowing down or discontinuing exercise or work, or removing clothing/equipment. In military situations, behavioral thermoregulatory drives are often overridden by motivation to successfully complete the mission. Physiological temperature regulation operates through heat loss responses (sweating and increased skin blood flow), which are proportional to the elevated core temperature and modified by skin temperature (warm skin enhances heat loss responses). Body heat loss by conduction, convection and radiation is mediated by altering skin blood flow. Body heat loss by evaporation is primarily by secreting sweat.

If the body stores heat, skin and or core temperature will increase. In response, the body initiates heat loss responses (sweating and increased skin blood flow). Unless the heat stress exceeds the thermoregulatory system's capacity to dissipate heat, the heat loss responses will increase until they restore heat balance, so core temperature stops increasing. If climate or clothing limits heat loss below the rate of heat production, then increases in sweating and skin blood flow will not restore heat balance but will only increase physiological strain.

Heat stress increases skin blood flow that elevates skin temperature. Skin temperature generally increases with ambient temperature but remains below core temperature. When sweating does not occur, increasing skin blood flow will elevate skin temperature, and decreasing skin blood flow will lower skin temperature nearer to ambient temperature. Thus, heat loss by conduction, convection and radiation is controlled by varying skin blood flow, and thereby skin temperature. When sweating occurs, the tendency of skin blood flow to warm the skin is approximately balanced by the tendency of sweating to cool the skin. Therefore after sweating has begun, a high skin blood flow primarily acts to deliver heat to the skin where it is dissipated by sweat evaporation.

Skin temperature is higher in warmer environments, while core temperature is relatively unaffected over a wide range of ambient temperatures. Thus at hotter ambient temperatures, the core-to-skin thermal gradient becomes narrower, and skin blood flow increases to achieve heat transfer sufficient for thermal balance. If evaporative cooling is not present, the core temperature will increase and widen the core-to-skin temperature gradient to help achieve sufficient heat transfer. Likewise, if increased evaporative cooling lowers skin temperature, the core-to-skin thermal gradient becomes wider, and skin blood flow requirements are proportionately less to achieve the same heat transfer.

Maintaining a high skin blood flow strains the cardiovascular system during physical work in the heat. High skin blood flow is associated with pooling of blood in compliant skin and subcutaneous vascular beds. This pooling reduces cardiac filling and stroke

volume, thus requiring a higher heart rate to maintain cardiac output. For these conditions, the primary cardiovascular challenge is to have sufficient cardiac output to simultaneously support high skin blood flow for heat dissipation and high muscle blood flow for metabolism. To help compensate for reduced cardiac filling, sympathetic activity is increased to elevate myocardial contractility and to divert blood flow from the visceral to skin and muscle. The reduction in visceral blood flow, if excessive, can contribute to the development of heat injury.
Since hot weather elicits high sweating rates, individuals will dehydrate if they do not replace their water losses. Dehydration reduces evaporative and convective heat loss, increases body temperature(~0.36° F or 0.2° C per percent of body weight loss), increases cardiovascular strain (heart rate ~5 beats per minute per percent of body weight loss), may reduce the core temperature that can be tolerated, and increases the risk of heat casualties. Dehydration reduces physical work capabilities in the heat. Over consumption of water (hyperhydration) provides no physiological advantage (compared to normal hydration) and acts to increase urine water loss. If individuals substantially over-consume fluids over an extended (typically >4 hours) period while not replacing their salt losses, they can develop hyponatremia (low blood sodium).

Mental Performance

Heat stress can reduce mental performance, which is probably mediated by thermal discomfort (from high skin temperature, high skin wettedness, and cardiovascular strain). However, a very incomplete database exists relating mental performance degradation relative to graded levels of heat stress and strain. Mental performance degrades the most in boring, monotonous and repetitive tasks. In addition, tasks that require attention to detail, concentration, and short-term memory and are not self-paced may degrade from heat stress. Heat stress slows reaction time and decision times. Routine tasks are done more slowly. Errors of omission are more common. Vigilant task performance will degrade slightly after 30 minutes and markedly after 2 to 3 hours.

Adaptations to Heat Stress

Biological adaptations to repeated heat stress include heat acclimatization and acquired thermal tolerance. The magnitude of both adaptations depends on the intensity, duration, frequency, and number of heat exposures. These adaptations are complementary as heat acclimatization reduces physiological strain, and acquired thermal tolerance improves tissues resistance injury for a given heat strain.

Heat acclimatization is induced when repeated heat exposures are sufficiently stressful to elevate core and skin temperatures and provoke perfuse sweating. During initial heat exposures, physiologic strain will decrease each subsequent day of heat acclimatization.

Fluid replacement

Heat stress increases the sweating rate and therefore body water needs. If fluid is not fully replaced, the dehydration will occur. The myth that individuals can be taught to adjust to decreased water intake has been proven wrong many times.

Thirst does not adequately motivate personnel to promptly consume sufficient fluids to replace sweat losses in hot environments. If thirst alone is used to guide fluid replacement, adequate hydration lags behind fluid needs for several hours. In addition to water, sodium, chloride and other electrolytes (potassium, calcium, and magnesium) are lost in sweat. A method to replace salt loss is drinking water at a rate of ¼ teaspoon of salt to each quart of water.

Injury Spectrum

Minor heat illnesses include heat cramps and heat exhaustion. Major heat injuries include EHI, exertional rhabdmyolysis, and heat stroke. The diagnostic categories of heat exhaustion, EHI, and heat stroke have overlapping features and should be thought of as different regions on a continuum rather than discrete disorders, each with its own distinct pathogenesis. All are related to elevation of body core temperature and the metabolic and circulatory processes (including changes in fluid and electrolyte balance) that are brought about by heat strain from exercise, environment and the body's thermoregulatory response.

Risk Factors

Factors that adversely influence thermoregulation can increase the risk of being a heat casualty. These include –

1. Dehydration
2. Salt depletion
3. Lack of heat acclimatization
4. Poor physical fitness
5. Excessive body weight
6. Skin disorders
7. Medications (prescription and over-the-counter)
8. Alcohol use
9. Inflammation and fever
10. Gastroenteritis
11. Chronic disease (for example, diabetes mellitus, cardiovascular disease, and congestive heart).

Heat stroke often occurs under conditions the victim had been exposed to many times before, or while other are concurrently being exposed to the same condition without incident. This suggests that these victims were inherently more vulnerable that day and or some unique event triggered the heat injury. Evidence suggests some cases might be explained by an association between susceptibility for malignant hyperthermia and

exertional heat stroke. Many heat stroke victims are sick on the prior day. Heat stroke cases often occur during the initial hours of exercise-heat stress and do not usually occur during the hottest part of the day. These facts suggest that, on that day, the victims began the exercise-heat stress compromised. Fever and inflammatory responses from muscle injury adversely influence thermoregulation and may help mediate heat illness. Gastrointestinal problems will induce dehydration and increase risk of being a heat casualty.

Mild sunburn impairs sweating so individuals should minimize skin exposure to ultraviolet radiation.

Drugs such as glutethimide (a sleep medicine), trcyclic antidepressants, phenothiazines (tranquilizers and antipsychotic drugs), bromocriptine, and antihistamines (commonly found in allergy, cold and sleep medication) have anticholinergic actions and are associated with heat stroke. Other drugs like cocaine and amphetamines (including "Ecstasy") will increase the risk of heat stroke.

Diuretics cause loss of fluid and electrolytes and have a similar effect as dehydration. Lithium can produce a reversible nephrogenic diabetes insipidus and consequent loss of water.

Many commercial ergogenic aid/weight loss products contain Ephedrine (often under the name "Ma Hwang"), which has been implicated in several heat stroke cases. Ergogenic aids are a particularly insidious risk factor, since many people who take them do not know what they contain and often do not consider them a drug.

Heat Cramps

Heat cramps are brief, recurrent, and often are agonizing skeletal muscle cramps of the limbs and trunk. The cramp in an individual muscle is usually preceded by palpable or visible fasciculations and lasts ~ 2 to 3 minutes. Cramps tend to be re-recurrent and may be precipitated by vigorous use of affected muscles. The cramp produces a hard lump in the muscle. Heat cramps often occur in salt-depleted persons during a period of recovery (up to many hours) after a period of intense work in the heat.

Heat Exhaustion

Heat exhaustion is the most common form of heat casualty and is not associated with evidence of organ damage. It occurs when the body cannot sustain the level of cardiac output necessary to meet the combined demands of skin blood flow for thermoregualation and blood flow for the metabolic requirements of exercising skeletal muscle and vital organs. Contributing factors include dehydration-mediated hypovolemia, peripheral blood pooling in dilated and compliant skin, and possibly failure of splanchnic vasoconstriction which together limits venous return.

The signs and symptoms of heat exhaustion include generalized weakness, fatigue, ataxia, dizziness, headache, nausea, vomiting, malaise, hypotension, tachycardia, muscle cramps, hyperventilation and transient alteration in mental status. Sweating persists and may even be profuse. The diagnosis of heat exhaustion versus more severe heat illness (that is, EHI or heat stroke) is important due to the difference in treatment and prognosis. Treatment should begin immediately to prevent progression to a sever heat injury.

Heat stroke should be the working diagnosis in anyone who has a heat casualty and has alteration in mental status. Individuals who have mild symptoms but don't improve or worsen with initial management or those who have severe symptoms, need to be evacuated to the nearest medical facility for additional assessment to include laboratory evaluation.

Management is directed to correcting the two pathogenic components of the illness: excessive cardiovascular demand and water-electrolyte depletion. The load on the heart is reduced by rest and cooling. Heavy clothing should be removed from patients and they should be allowed to rest in a shaded and ventilated space, while active cooling is initiated. Water-electrolyte depletion is corrected by administering oral or parenteral fluids. Heat exhaustion victims should improve rapidly with shaded rest, cooling and rehydration; those who don't improve or get worse need to be forwarded to the next higher level of medical care.

Heat exhaustion casualties retain the ability to cool spontaneously if removed from heat stressors. However active cooling to a core temperature of 101° F (38.3° C) is strongly recommended, for two reasons. First, the resulting skin vasoconstriction rapidly reduces circulatory demand and improves venous return. Second, if casualties with the more severe condition of EHI are not cooled actively, their core temperatures may continue to rise or may fall too slowly. Since heat exhaustion and more severe heat injuries may be hard to distinguish initially, medical personnel who elect to delay active cooling to see whether the casualty can spontaneously cool will sometimes fail to provide immediate active cooling when it is required. In addition, those with heat exhaustion may progress to a more severe heat injury if not promptly cooled.

Rectal temperature should be monitored frequently (at least 15 min) to ensure that core tempertature is falling to normothermic levels. Although it is convenient to measure temperature in the mouth or the ear canal, medical personnel should not use these temperatures in the evaluation or management of heat casualties. Hyperventilation may cause oral temperatures to be ~3° C (5° F) lower then rectal temperature in heat stroke, and the temperature of the external auditory canal or tympanic membrane has been observed to be as much as ~5° C (9° F) below rectal temperature in collapsed hyperthermic runners.

The patient should be reassessed frequently including repeated core temperatures and mental status checks. Intravenous lines may be placed if the patient is unable to take adequate oral fluid replacements or if medical evacuation to a higher level of care is indicated (as needed). Patients that aren't responding to initial treatment will require

laboratory tests to rule out a more severe heat illness. These tests should include complete blood count (hemoglobin, hematocrit, white blood count, and platelet count), chemistry hepatic transaminases (AST/SLT), creatine, phophokinase, lactate dehydrogenase (LDH), uric acid, and myoglobin. Urine should be collected for urine analyses and urine myoglobin. More severe cases would include evaluation of calcium, magnesium, phosphorus, protein, albumin, and lactic acid. Cases that are unexpected for the degree of heat stress exposure or seem to respond poorly to appropriate treatment monitoring of blood pressure, urine output and mental statues is needed. Cooling procedures should be stopped once the individual's core temperature has reached 101° F (38.3° C) to prevent over-shoot hypothermia.

Heat exhaustion victims may experience rapid clinical recovery. However, they must avoid re-exposure to heat stress for at least 24 hours. These patients do not require further medical evaluation or reporting. Patients who do not fully recover within one hour or require more then two liters of rehydration should be evaluated in an emergency department, usually to include laboratory tests, reporting, follow-up, and profiling.

A single episode of heat exhaustion does not imply any predisposition to heat injury; however, victims of more serious heat injuries can subsequently have decreased heat tolerance. Repeated heat exhaustion episodes require a thorough medical evaluation.

Exertional Heat Injury

Exertional heat injury represents a continuum intermediate in severity between heat exhaustion and heat stroke. There is no consensus on diagnostic criteria for distinguishing EHI from heat exhaustion or heat stroke; therefore, close monitoring of vital signs and serum chemistries is essential since during the first few hours, clinical symptoms may not reflect profound underlying metabolic abnormalities.

EHI patients show evidence of organ (for example, liver or renal) or tissue (for example, muscle) injury or dysfunction but do not display sufficient neurological abnormalities to meet the usual criteria of heat stroke. Other manifestations may include neurological abnormalities to meet the usual criteria of heat stroke. Other manifestations may include neurological symptoms, high core temperature and metabolic acidosis. Patients demonstrating combativeness, delirium, obtundation, or coma most likely have heat stroke rather than EHI.

Organ dysfunction or tissue damage may not be manifest in early EHI, so during the first hours of illness it may not be possible to distinguish EHI from heat exhaustion by symptom complex alone. Therefore, all suspected EHI patients should be thoroughly evaluated for organ damage/dysfunction before release, with re-evaluation necessary on the following day.

Suspected EHI patients should be immediately and actively cooled to a core temperature of 101° F (38.3° C). Most EHI victims are sweating profusely and will cool spontaneously after removal from the stressful circumstances; however, in some their

core temperatures may continue to rise. Delaying active cooling to see whether a casualty can cool spontaneously, is not advised. It is important to prevent progression of the heat injury process. Persistent elevation of body temperature suggests the probability of more severe pathology. Occasionally, cooling leads to inappropriate shivering while core temperature is still elevated.

Heat Stroke

Heat stroke is characterized by elevated body temperature (>40° C or 104° F, core temperature could be lower if measured after body cooling has occurred) and central nervous system dysfunction that results in delirium, convulsions, or coma. Heat stroke is a catastrophic medical emergency resulting from a failure of the thermoregulatory mechanisms. It can cause extreme elevation of the body (for example, 108° F (42° C)), producing multi-organ dysfunction. However, many cases of exertional heat stroke with severe encephalopathy but modest hyperthermia have been reported. Major neurological disturbances typically are characteristic of a non-focal encephalopathy, while some patients may show transient or persistent abnormalities of cerebellar function. The onset of heat stroke may be heralded by up to an hour of prodromata, including headache, dizziness, drowsiness, restlessness, ataxia, confusion and irrational or aggressive behavior. Heat stroke may be complicated by liver damage, electrolyte abnormalities and, especially in the exertional form, by rhabdomyolysis, disseminated intravascular coagulation (DIC) or renal failure.

Exertional and classical are two types of heat stroke that occur under different settings and produce different clinical pictures. Exertional heat stroke occurs in physically active persons who are producing substantial metabolic heat loads and it can occur in both hot and temperate conditions. Classical heat stroke occurs in individuals with impaired thermoregulation due to illness or medication, exposed passively to heat, and often dehydrated. A major difference is that exertional heat stroke is often complicated by acute rhabdomyolysis, with renal failure as a common consequence.

The traditional diagnostic criteria of heat stroke, coma or convulsions, hot dry skin, and core temperature above 106°F (41.3°C) reflect experience primarily with the classical form. Rigid adherence to these criteria will lead to under diagnosis of exertional heat stroke, since coma, convulsions, and cessation of sweating may be late events in exertional heat stroke. Moreover, patients may receive medical attention after they have had a chance to cool somewhat and regain consciousness, especially if they still are sweating.

The principle foci of heat stroke injury are brain, liver, kidney, muscle and hemostasis. Since the seriousness of injury is mostly, but not entirely, predicted by the magnitude of body temperature elevation and duration, other pathogenic factors probably have a role in the evolution of heat stroke, including tissue ischemia, hypokalemia, exercise-induced lactic acidosis and systemic inflammatory response, perhaps triggered by products of muscle injury and or by endotoxin leaking from the gut.

The clinical outcome of patients with heat stroke is primarily a function of the magnitude and duration of body temperature elevation. The most important therapeutic measure is rapid reduction of body core temperature. Rapid cooling can reduce heat stroke mortality from 50 to 5 percent. Cooling should begin in the field, and victims must be quickly removed to a cool shady place, their clothes removed, and their skin kept moist. Active cooling should be started immediately and continued during evacuation. Cooling and evaluation should proceed simultaneously. While cooling in underway, rectal temperature should be closely monitored. Active cooling should be discontinued when the rectal temperature reaches 101°F (38.3° C) to avoid hypothermia.

Any effective means of cooling is acceptable. Immersion in cool or iced water with skin massage is the most rapid method. Ice sheets and ice packs are effective. No pharmacologic agents that will “theoretically” accelerate cooling should be used. If attempts to cool the patient are
successful, the body core temperature may rise again once cooling is stopped. Occasionally, cooling leads to inappropriate shivering while the temperature is elevated; however, the active cooling will remove much more heat than being produced.

Exertional Heat Injury – Case Study

On October 17, 2002, Dale R. Spring, miner, age 49, was fatally injured and Theodore C. Milligan, mine rescue team trainer, age 38, was critically injured when they collapsed while evaluating conditions in an inactive underground gold mine. Milligan passed away on October 23, 2002.

The victims were part of a mine rescue team that had been directed to explore a gold mine that had been inactive for more than two years. Spring had a total of 26 years mining experience, 6 years and 12 weeks with this company. Milligan had a total of 10 years mining experience, 2 years with this company.

On the day of the accident, a three-man team entered the mine and advanced 800 feet before the effects of high heat, high humidity, and foggy conditions forced their return to the surface. Underestimating the hazards presented by this environment, the second team entered the mine and advanced about 2,000 feet before deciding to return to the surface.

The accident resulted from a failure to accurately assess the risks from environmental exposure to excessive heat and humidity.

GENERAL INFORMATION

The Storm Exploration Decline (Storm Decline), an inactive underground gold exploration mine, was located near Elko, Eureka County, Nevada.

The Storm Decline had been developed from the lowest level of the open pit in March 1999. The decline was developed to explore ore bodies to identify potential gold-bearing ore reserves. Conventional mining methods utilizing drilling, blasting, and trackless haulage were used to advance the decline. The mine was closed during April 2000 and access to it was controlled by locked gates.

Two weeks prior to the accident, the engineering department contacted the mine rescue team training coordinator to ask if the next scheduled underground mine rescue training could be conducted at the Storm Decline to map underground conditions, and to evaluate the mine for the possibility of reactivating it.

DESCRIPTION OF THE ACCIDENT

On the day of the accident, Dale Spring and Theodore Milligan (victims) and seven other mine rescue team members reported for work at the Mine rescue station between 6:20 and 7 a.m., their normal starting time. A supervisor who had worked at this mine when it was developed was selected to accompany the team during this activity. Team members

reviewed the map of the Storm Decline and Milligan explained to the team that their assignment would involve mapping conditions of the Storm Decline for possible reactivation of the mine.

Each team member worked with a partner to bench test their self-contained breathing apparatus. Spring and Milligan assisted other team members in testing their apparatus. None of the other team members could recall if Milligan or Spring tested their own apparatus during this time. The team loaded their apparatus into the back of a pickup truck and drove 6 miles to the Storm Decline, arriving at the portal at about 9 a.m.

Milligan again briefed the team at the portal on the conditions they could encounter in the decline. The team was told to anticipate low levels of oxygen and high heat, and not to exceed their limits. They were instructed to check the ground conditions, utilities, and atmospheric conditions, monitor mine gases, take pictures of the exploration, and verify the conditions accurately on the mine map. The nine team members were divided into three three-man teams. The teams were to carry the following equipment: a pager phone, 500-foot rolls of blasting line for communications, a TMX 412 multi-gas monitor, a digital camera, and scaling bar.

After examining and testing the mine opening at about 9:55 a.m., team one, consisting of Bart Freteig, Dan Marque, and Kurt Tomton, miners, entered the mine wearing their apparatus but not under oxygen. They proceeded about 500 feet down the decline. At that point, the multi-gas monitor alarm indicated a low oxygen level reading of 19.5 percent. After reporting the oxygen level to the surface, they donned their facemasks, went under oxygen, and advanced.

As the team advanced and stopped at unspecified intervals, they cut the wire and hooked up the phone to call outside. After calling, they spliced the wire with the remaining wire on the spool and continued to advance. As one spool ran out, they spliced another one on. The teams left the phone at their farthest point of advance for the next team's use.

At 10:25 a.m., team one reached the 800-foot level and phoned the surface. They reported the following conditions to the surface: oxygen readings of 18.5 to 19.0 percent, heat readings of 103 degrees Fahrenheit (F), and fog that prevented taking photographs and limited their visibility. They reported that they were going to retreat to the surface. At 10:32 a.m., the team arrived at the surface.

After being debriefed by team one, team three, consisting of Milligan, Spring, and Brett Campbell, mechanic, decided they would continue the exploration and entered the decline at 10:45 a.m. Team two, consisting of Lenny Wilcox, miner, Gary Pitt, Part 48 Trainer, and Lee McCombs, miner, functioned as the backup team with McCombs assigned to monitor the mine phone and record the information called outside. Spring was spooling out wire for the mine pager phone. Milligan monitored gas readings, and Campbell assisted both. Team three proceeded down the decline to the 500-foot level where they donned their apparatus. At 11 a.m., after advancing to the 800-foot level,

Spring called the surface. He reported oxygen readings of 10.2 to 10.3 percent, high heat, and stated they were continuing down the decline.
At 11:17 a.m., the team stopped at about 1000 feet and Spring reported 17.3 percent oxygen and a temperature of 103 degrees F. The team continued to advance and at 11:25 a.m. they reached the 2,000-foot level. Spring called McCombs to report that they could not read the multi-gas monitor because the indicator was fogged. He reported 104 degrees F and high humidity, and said they were going to retreat back to the surface.

The team left the mine phone and started walking up the 15-percent decline with Spring in the lead. Milligan was having difficulty coping with the heat and stopped to rest every 20 to 30 feet. When Milligan told Campbell he was not sure he could make it to the surface, Campbell stayed with him as they walked up the decline. They eventually caught up with Spring, who was also exhausted from climbing the steep slope. Campbell then decided to continue alone at a slow pace to get help. Campbell tried to signal team two by shorting the phone line as he made his way up the slope.

At 11:40 a.m., as Campbell neared the surface, McCombs heard Campbell's yell for help. Wilcox, Pitt, and McCombs went down the decline about 100 feet, where they met Campbell. Campbell was completely exhausted and informed them that Spring and Milligan were down, and that their units might be out of oxygen.

At 11:45 a.m., the company's emergency response plan was activated and emergency medical personnel were requested. After team two retrieved the three oxygen cylinders from the apparatus used by team one, they entered the mine with the spare cylinders and located Spring about 700 feet from the mine opening, unconscious, lying face down with his mask on. Spring's oxygen bottle was checked and found to contain about 1,500 pounds per square inch gauge (PSIG) of oxygen. The bypass on Spring's apparatus was used during the effort to revive him. After these efforts were unsuccessful, Spring's mask was removed and his vital signs were checked. His pulse was quick and weak and no respiration activity was detected. CPR was started and continued for several minutes, with no signs of response. The team then decided to look for Milligan.

Milligan was found about 70 feet farther down the decline from Spring's location. He was lying on his side with his mask off, unconscious, gasping for breath. Milligan's apparatus was checked and it indicated the oxygen cylinder was empty. The team proceeded to give Milligan oxygen using their own masks and he became semi-conscious. They tried to carry Milligan up the 15-percent grade to the surface, but the steep slope made their attempts unsuccessful. They continued to care for Milligan until they were relieved by emergency medical personnel, who arrived at the mine at 12:52 p.m. At about 1:30 p.m., Milligan was placed on a stretcher, transported to the surface, and life-flighted to Elko General Hospital. He was transferred the next day to the LDS Hospital in Salt Lake City, Utah, where he died on October 23, 2002. The cause of death was attributed to lack of oxygen to the brain from environmental exposure.

Spring's body was transported to the surface where he was pronounced dead. The cause of death was attributed to multiple organ failure from environmental exposure.

Accident Location

The accident occurred inside the inactive Storm Decline. The mine was driven on a 15-percent downgrade for approximately 3,400 feet. Drift height was about 15 feet with an average width of 15 feet. Rockbolts were installed on 4-foot centers for ground support.

The Storm Decline had not been worked since April 2000 and mechanical ventilation was not provided to clear the mine air prior to the rescue personnel entering. The map provided to the team was last updated in April 2000.

Equipment

The nine rescue team members were equipped with the Biopak 240S self-contained breathing apparatus manufactured by Biomarine Incorporated.

The equipment provided for the team's use at the Storm Decline site included the following: a digital camera, a scaling bar, a TMX 412 multi-gas monitor, two Femco mine pager phones, several 500-foot spools of two-strand blasting wire, and a Bacharach sling psychrometer for measurements of temperature and humidity.

The TMX multi-gas monitor, manufactured by Industrial Scientific Corporation, was equipped with sensors installed to measure oxygen, carbon monoxide, sulfur dioxide, and lower explosive limits of combustible gases. The monitor did not contain a hygiene board. The last calibration record for this monitor was October 9, 2002.

First-aid supplies, spare self-contained breathing apparatus, spare compressed oxygen cylinders and a stretcher were not provided at the Storm Decline.

Gas and Temperature Readings

The gas readings taken in the mine by the teams were phoned to the surface where they were logged. Readings logged on October 17, 2002, are as follows:

Team No. 1

Location	Oxygen	Temperature
500 feet from portal	19.5 to 20.0 percent	none reported
800 feet from portal	18.5 percent	103 degrees F.

Team No. 2

800 feet from portal	10.2 to 10.3 percent	none reported
1,000 feet from portal	17.3 percent,	103 degrees F.

2,000 feet from portal none reported 104 degrees F.

Biomarine User Instructor Manual

The Biomarine User Instruction Manual directed persons preparing the apparatus for use to obtain a frozen coolant canister charge (Gel-Pak/Gel-Tube) and install it into the coolant canister. The frozen Gel-Pak/Gel-Tube, required by MSHA/NIOSH approval, can reduce the temperature of the breathing gas entering the canister to maintain the gas temperature and dew-point below 90 degrees F (according to Biomarine manuals). The Biomarine breathing apparatus No. 10 worn by Milligan and No. 15 worn by Spring did not contain frozen Gel-Paks/Gel-Tubes as required. MSHA standards require that miners use respiratory protective equipment in accordance with training and instruction. (citation listed in report)

The Biomarine User Instruction Manual cautions that a poor face piece seal will cause a significant decrease in Biopak duration. Both of the victims had not shaved off their goatees prior to wearing the self-contained breathing apparatus on the day of the accident. MSHA standards require that miners use respiratory protective equipment in accordance with training and instruction. Mandatory standard 57.5005(b) prohibits the use of respirators (apparatus) when conditions such as a beard project under the face piece and prevent a good face seal.

The third member of team three, Brett Campbell, was clean-shaven and had inserted a frozen Gel-Pak/Gel-Tube in his apparatus as required. Members of teams one and two were also clean-shaven and had inserted frozen Gel-Paks/Gel-Tubes in their apparatus.

Testing

Tests were conducted on the four Biomarines, Biopak 240S, closed-circuit self-contained breathing apparatus (SCBA) involved in the fatalities and rescue efforts. The testing was performed jointly by the National Institute for Occupational Safety and Health (NIOSH) and MSHA's Office of Technical Support. The testing began on November 25, 2002, at NIOSH's laboratory facility at Bruceton, Pennsylvania, and concluded on December 16, 2002.

Tests conducted on apparatus No. 15 worn by Spring determined the presence of a leak in the high pressure oxygen gauge line that resulted in the gauge reading 800 PSIG less than the actual oxygen pressure available in the unit's compressed oxygen cylinder. A second leak was found in the oxygen line threaded connection near the compressed oxygen cylinder. Neither defect was found to contribute to the cause of the accident.
The log-book at the mine rescue station documented that all self-contained breathing apparatus had been inspected and tested on September 27, 2002, and found to be free of defects.

Training

Both Milligan and Spring had received training in accordance with 30 CFR, Parts 48 and 49.

ROOT CAUSE ANALYSIS

A root cause analysis was performed on the accident. The following causal factors were identified:

Causal Factor: The risk assessment process conducted by management, prior to sending the mine rescue team to conduct an exploration of the inactive Storm Decline, was inadequate. Procedures were not established to address all hazards affecting the safety of the rescue team members while performing this task.

The mine had been inactive for more than 2 years. Management knew the mine was not ventilated and the temperature in the mine was expected to be near 100 degrees F with high humidity, possible low oxygen levels, and elevated levels of carbon dioxide.
The mine rescue team coordinator was informed that he should utilize mine rescue personnel to assess the physical conditions of the Storm Decline. However, mine management did not correctly evaluate the hazards that this assignment presented. Management was aware of low oxygen levels and high temperatures prior to the rescue team members entering the mine.

The mine rescue team coordinator was left to direct this task and was not assisted by management to develop a protocol that listed a specific sequence of exploration along with procedures to be followed.

Even though all mine rescue team members were trained and experienced, management was responsible for communicating with teams in these circumstances and for safely directing their actions.

Corrective Action: A plan should be developed to establish exploration procedures. The plan should address the specific tasks each team is to complete. The procedures should include actions that must be initiated by the team to ensure their safety and limit the distance they can explore.

Causal Factor: The apparatus manufacturer's User Instruction Procedures were not followed while readying the self-contained breathing apparatus in that the frozen Gel-Paks/Gel-Tubes were not installed in the apparatus' coolant canisters worn by Milligan and Spring.

When the team wore their apparatus during underground training the established practice was to have the mine rescue team trainer and rescue team members coordinate the training exercise. It was determined through interviews that some of the miners had not installed the frozen Gel-Paks/Gel-Tubes in their apparatus during prior underground training as required.

Corrective Action: Mine rescue training procedures should be reviewed to ensure that responsibilities regarding the testing and use of self-contained breathing apparatus are clearly established. All mine rescue team members must be familiar with these procedures and management should actively participate during practice and training to ensure that self-contained breathing apparatus are properly maintained and utilized.

Causal Factor: Communication between the rescue team underground and the surface was inadequate.

The rescue team carried 500-foot spools of two-stranded blasting wire along with a Femco mine pager phone. This method of communication required the team to cut the blasting wire and connect it to the pager phone each time they wanted to communicate to the surface.

This method of communication did not provide a means for the surface to remain in constant communication with the team to ascertain their location and their condition at all times while they remained underground.

Corrective Action: Mine rescue training procedures should be reviewed to ensure that sound powered communication systems are used at all times when teams enter unknown underground atmospheres. These communication systems must be approved as stipulated by Code of Federal Regulations, Part 23, and must be capable of providing continuous voice contact between the fresh air base and the mine rescue team.

Possible Causal Factor: The apparatus manufacturer's User Instruction Procedures were not followed in that both victims had failed to shave off their goatees prior to wearing the self-contained breathing apparatus.

The Biomarine User Instruction Manual stipulated users of Biopak 240S apparatus should be clean–shaven because the presence of facial hair could interfere with the apparatus' face piece seal, resulting in a significant decrease in the duration of service time.

The Biopak 240S apparatus maintains a positive pressure inside the face piece to prevent the user from breathing gases outside the face piece. A poor face piece seal would cause the unit to send larger amounts of compressed oxygen from its cylinder in the effort to maintain the proper pressure inside the face piece.

Milligan was aware that his team had encountered oxygen levels below 11 percent in one area of the decline as they advanced. Although the investigation was not able to determine why Milligan had removed his mask, it was determined that his oxygen cylinder contained less than 3000 PSIG when he donned his apparatus. It is also likely that his goatee prevented an air tight seal around his face piece. This condition would have caused oxygen to leak continuously as Milligan wore the apparatus. It is unlikely that he would have removed his mask unless he had exhausted his oxygen supply knowing that low oxygen levels had been measured in the mine.

The established practice at the mine was to allow mine rescue team members to wear the Biopak 240S apparatus during underground practice, in known fresh air, without having to be clean-shaven. Although this practice was not uncommon, it was mine management's responsibility to ensure that all required procedures were being followed when teams enter unknown underground atmospheres.

Corrective Action: Mine rescue training procedures should be established to ensure that every mine rescue team member is clean-shaven to ensure that no facial hair is present that would interfere with the face piece seal prior to donning self-contained breathing apparatus when entering unknown atmospheres.

CONCLUSION

On October 17, 2002, two mine rescue team members collapsed, and later died, while attempting to climb a 15-percent decline to the surface for a distance of about 2,000 feet.

The victims were part of a mine rescue team that had been directed to explore a gold mine that had been inactive for over two years. Two weeks prior to the accident management requested that the next scheduled underground mine rescue training be conducted at the Storm Decline to map underground conditions to evaluate the mine for the possibility of reactivating it.

Management was aware that this mine had not been ventilated since April 2000. They also knew the temperature in the mine was expected to be near 100 degrees Fahrenheit with very high humidity.

On the day of the accident, a three-man team had entered the mine and advanced 800 feet before the effects of high heat, high humidity, and foggy conditions forced their return to the surface. The failure to recognize the hazards presented by this environment resulted in the second team being allowed to advance into the mine and travel about 2,000 feet before returning to the surface.

The accident resulted from a failure to accurately assess the risks from environmental exposure to excessive heat and humidity.