

Appalachian Search and Rescue Conference  
Center for Emergency Medicine of Western Pennsylvania

# Wilderness EMT Lesson Plan

## Part XIII: Wilderness Trauma

Draft Version 1.7 August 12, 1994

Comments to:

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## **Verbose Outlines**

We develop our WEMT Lesson Plans in a **verbose outline format** (what you see here). Why? Because the material is new to enough reviewers that the usual terse ("telegraphic") lesson plan format might be incomprehensible or misleading.

Our Task Groups use these "verbose" outlines. Each part of the WEMT curriculum (about twenty in all) has a Task Group of five to twenty selected consultants. A Coordinator guides the Task Group in revising the section.

Each Task Group provides references to support its statements and for further reading. They also provide glossary entries for any new terms they introduce. (New, that is, to a reader with basic EMT and SAR training.)

Background material that should appear in the Textbook (see below), but instructors need not present in class, will appear *in a small, italic font*.

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## **Splitting the Outlines**

When the outline satisfies the Task Group, it goes to our **Editorial Board**. This Board includes officers of the Appalachian Search and Rescue Conference and Center for Emergency Medicine of Western Pennsylvania, our two sponsors. It also includes experts in emergency medicine, search and rescue, and education. The Editorial Board reviews the verbose outline, and requests any necessary revisions. Once it is acceptable to the Board, we reformat the outline, into two distinct new versions.

We rewrite the material in the standard lesson plan format, which becomes a terse "telegraphic" outline. This version will be briefly reviewed by the Project Coordinator and then released to the public. It is the result of extensive review and testing, and will be used in all our classes. But, we still publish it as a draft, because we expect many good suggestions from the public. We distribute these drafts as widely as possible. After each year of public review, the Task Groups reviews comments, and submits revisions to the Editorial Board. Once all outlines have withstood a year of public scrutiny, we will prepare a single comprehensive curriculum with a Course Guide. We will continue to review and revise the curriculum regularly.

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## **On to a Textbook**

As explained above, once the Editorial Board approves the verbose outline, we split it into two versions. Besides the terse teaching outline, it will also become the basis for a textbook chapter. The

Project Coordinator is the textbook Editor-in-Chief, and works closely with the Task Groups to consolidate and revise the verbose outlines into a comprehensive textbook. All who have contributed to the curriculum will be acknowledged as contributors. The textbook will be commercially published when completed. Until the textbook is available, we will distribute the verbose outlines or drafts of the textbook at classes.

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## **Notes: Wilderness Trauma**

Specific surgical problems are discussed in the section on *Wilderness Surgical Problems*. The principles of general medical and nursing care are covered in the section on *Principles of General Medicine*. What is left for *Wilderness Trauma*?

In this section, we cover the epidemiology, etiology, and mechanism of injury for wilderness trauma (or at least the little that we know). We also discuss the general approach to the **wilderness** trauma patient, which is very different from urban and rural trauma. This section provides a review of background information on the physiology of trauma and shock. Even though the section on *Patient Assessment* reviews the physical exam in detail, here we re-emphasize specifics that are important in the trauma patient. (A large optional section reviews the physiology of shock and trauma.) We review fluid resuscitation and fluid balance over the period of an extended wilderness evacuation.

Perhaps most importantly, we present complications of trauma that are likely to occur while the Wilderness EMT is caring for the patient. Complications such as renal failure, crush injuries, myoglobinuria, hyperkalemia, ileus, glycogen depletion, compartment syndrome, ARDS, and fluid overload. Some of these complications can be treated by a Wilderness EMT, others can be recognized and evacuation plans changed, or perhaps a surgeon can be brought into a cave to perform a fasciotomy during a lengthy evacuation.

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**XVI. Wilderness Trauma**

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**A. Educational Objectives**

1. Describe the differences between urban and wilderness trauma care philosophy due to the effects of time and distance.
2. Describe the concepts of "the Golden Hour" and "the Golden Day" as they apply to trauma care.
3. Outline epidemiologic differences between urban and wilderness trauma, and identify medical problems that are commonly found in search and rescue patients.
4. Describe how to evaluate a wilderness patient for shock and for state of hydration.
5. Define "fluid challenge" and explain its importance in assessment and treatment of shock.
6. Define each of the following terms and explain its importance for choosing fluids for wilderness patients:
  - a. crystalloid;
  - b. colloid; and
  - c. blood.
7. Outline the advantages and disadvantages of each of the following resuscitation fluids for wilderness patients:
  - a. D5W;
  - b. NS or LR;
  - c. D5NS or D5LR;
  - d. hypertonic saline/dextran; and
  - e. blood.
8. Define:
  - a. blood type; and
  - b. transfusion reaction.

9. Outline management of a patient who is suffering a transfusion reaction.
10. Describe the proper use of pressors in wilderness rescue.
11. Outline a standard differential diagnosis of decreased urine output in a patient with a Foley urinary catheter.
12. Define acute renal failure.
13. Define each of the following complications of crush injury or burns, and describe management in the wilderness:
  - a. hyperkalemia; and
  - b. myoglobinuria.
14. Define glycogen depletion, outline the reasons for assuming that all wilderness patients are glycogen depleted, and outline wilderness management for patients with glycogen depletion.
15. Define compartment syndrome, identify where it most commonly occurs, outline its diagnosis and natural history, and identify the procedure needed for definitive treatment.
16. Define ARDS and describe its management in the wilderness.
17. Identify three physical exam findings found in fluid overload.
18. Outline appropriate decision-making for a patient with both multiple system trauma and severe hypothermia.
19. Demonstrate the ability to conduct an appropriate initial physical exam on a wilderness multiple-trauma patient.
20. Identify problems associated with long use of the MAST garment, and identify a specific use for it in vertical rescue.

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**B. The Trauma Perspective**

1. What are the three main reasons that people fail to come out of the woods? They're lost. They're sick. Or, they're

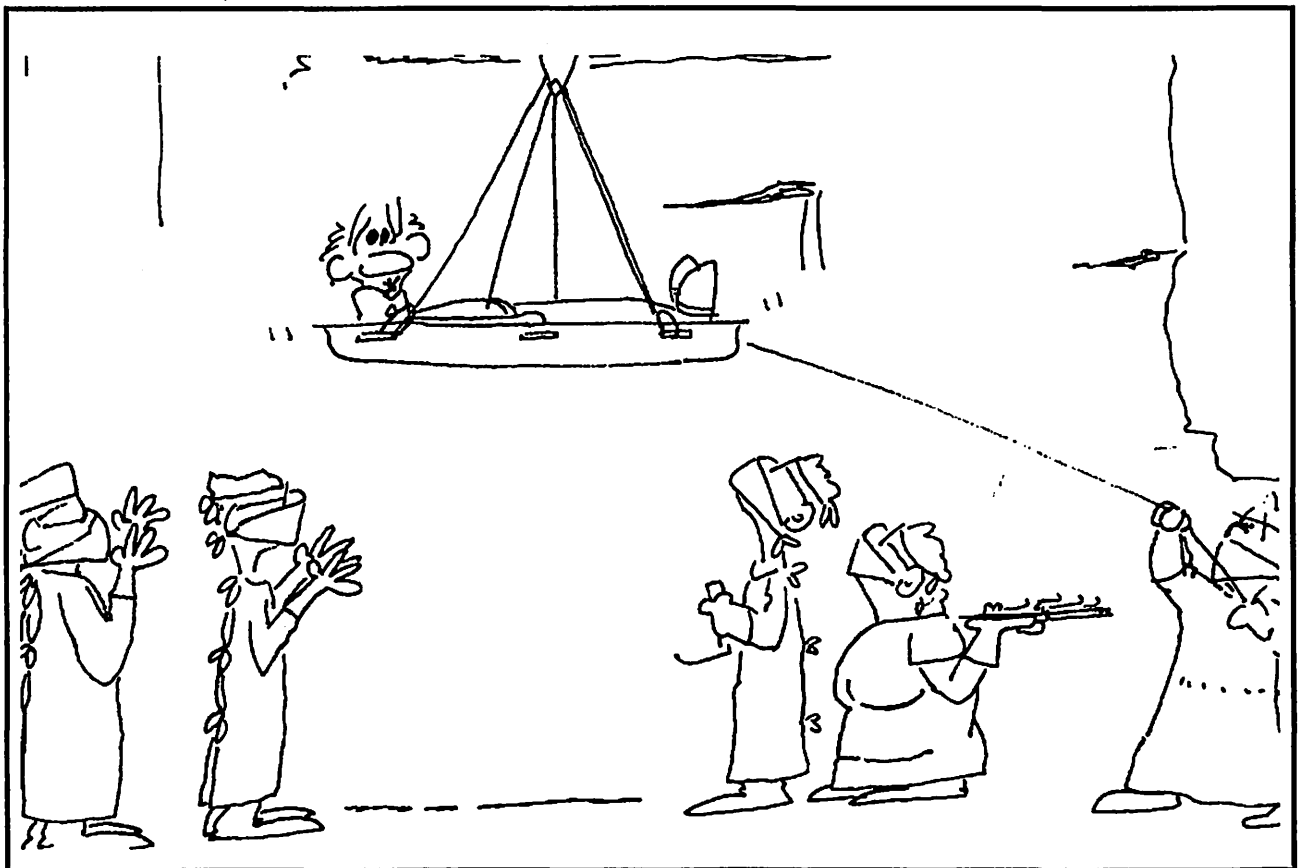
**Urban vs. Wilderness Trauma**

**Life in the Big City:**

- response time: 10 minutes
- time to hospital: 10 minutes
- all treatment en route; no treatment at scene ("scoop and run")
- address: Locust St. at River Road

**Down in the Boondocks:**

- response time: 24 hours
- time to hospital: 5 hours
- stabilization at scene
- address: down river, left at locust tree, third rock on right



hurt. The most likely is that they're hurt. "Street" EMTs divide trauma patients into one of two classes: immediately life threatening, or not life threatening. At least as far as trauma is concerned, the patients that wilderness SAR teams find alive always fall into the second category: not immediately life threatening. Survivable wilderness injuries are more often disabling than life-threatening.

2. The Advanced Trauma Life Support course states: "Death from trauma has a trimodal distribution. The **first peak of death** is within seconds to minutes of injury. Deaths occurring during this period are usually due to lacerations of the brain, brain stem, high spinal cord, heart, aorta, or other large vessels. Only a few of the patients can be salvaged and then only in large urban areas where rapid emergency transport is available. The **second death peak** occurs within minutes to a few hours after injury. The **primary focus of the Advanced Trauma Life Support Course is on this first hour of trauma management, when rapid assessment and resuscitation can be carried out to reduce this second peak of trauma deaths.** Deaths occurring during this period are usually due to subdural and epidural hematomas, hemopneumothorax, ruptured spleen, lacerations of the liver, pelvic fractures, or multiple injuries associated with significant blood loss. The fundamental principles of trauma care learned in this course can best be applied to these patients. The **third death peak** occurs several days or weeks after the initial injury, and is almost always due to sepsis and organ failure. Therefore, the first person to assess the patient can affect the final outcome."<sup>1</sup> This third death peak is the focus of wilderness trauma care.
3. Patients who initially compensate for their injuries **decompensate** very rapidly as time passes. This has given rise

to the concept of "The Golden Hour."<sup>2</sup> For every half hour a patient is in shock (the decompensated state), mortality doubles. Based on this, we can reasonably assume that mortality is higher in the wilderness setting. Despite retrospective data that is far from comprehensive, we can make several general projections about wilderness trauma patients.

- a. If the injury is immediately life-threatening, the patient will not be alive when found.
- b. If the patient is alive when found, he will most likely survive the evacuation. But:
- c. If the patient's condition is tenuous when found, you will most likely be looking at a "domino effect" of events rather than a single one. For example, a patient who sustains a major injury can have significant blood loss and rapid onset of shock, as opposed to someone who sustains a similarly disabling injury, doesn't lose as much blood, but lays on the ground for 2 days, thus becoming cold and dehydrated as well as injured. (This is one of the most consistent findings in retrospective data.)<sup>3,4</sup>
4. In the special case of aircraft crashes, if you don't find patients within the first 24 hours, you're not likely to find them alive. This is called the "Golden Day." (The Air Force Rescue Coordination Center at Scott AFB, IL, maintains a constantly-updated database on air crashes, which shows the high mortality in the first 24 hours. Contact the Aerospace Rescue and Recovery Service at Scott AFB for the latest statistics.)
5. The focus of urban trauma care is to save those who are in danger of dying in a matter of minutes. "Scoop and run" is the appropriate treatment for urban trauma patients. All measures are geared to rapid treatment and rapid transport to the operating theater. (For

example, extrication efforts emphasize speed rather than precise immobilization, and IVs are started en route if at all.) In the wilderness, though, those with such severe injuries are already dead. This is simply a function of time. It takes too long for someone to walk out for help, and too long for a team to respond. This leaves less-severely injured patients as the focus of this section.

- a. Less-severely-injured patients may still be in danger from injuries we can't fix. Evacuating the patient urgently is important for wilderness trauma care. But, the time scale is different. Delaying the evacuation fifteen minutes to perform a careful trauma assessment, will probably not make the patient's injuries any worse. However, if your exam detects hypothermia, dehydration, or myoglobinuria, you may save the patient's life.
6. Wilderness patients differ from urban trauma patients in ways other than being outside the "golden hour."
  - a. **They always have multiple problems.** (For example, cold exposure and dehydration are common.) EMT's on the street don't usually have to worry about these problems, because, for

short transport times, they can ignore them.

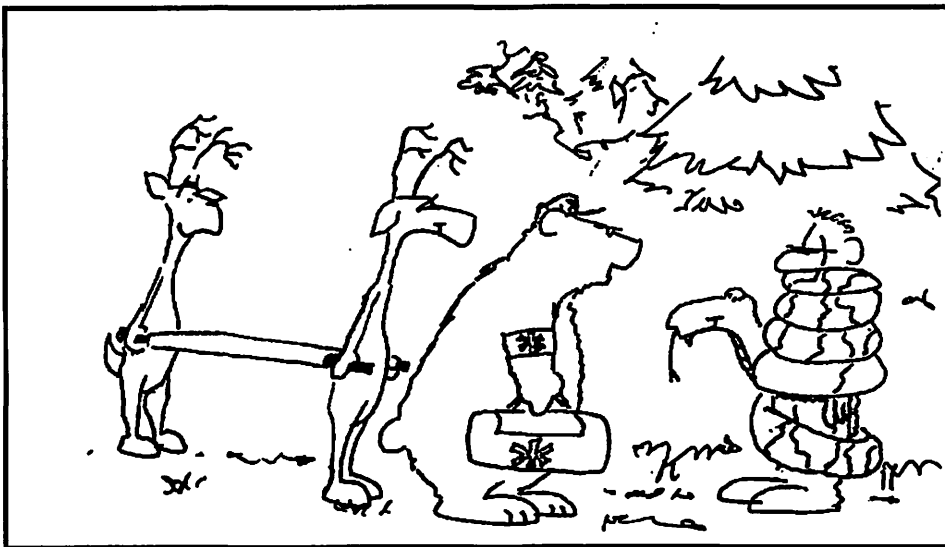
- b. **They require long evacuations.** Things happen during long evacuations. Patients develop the trauma complications that urban EMTs never see: compartment syndrome, deep venous thrombosis, acute renal failure, or adult respiratory distress syndrome. All trauma patients are in danger from such problems, but on the street, EMTs can outrun them.

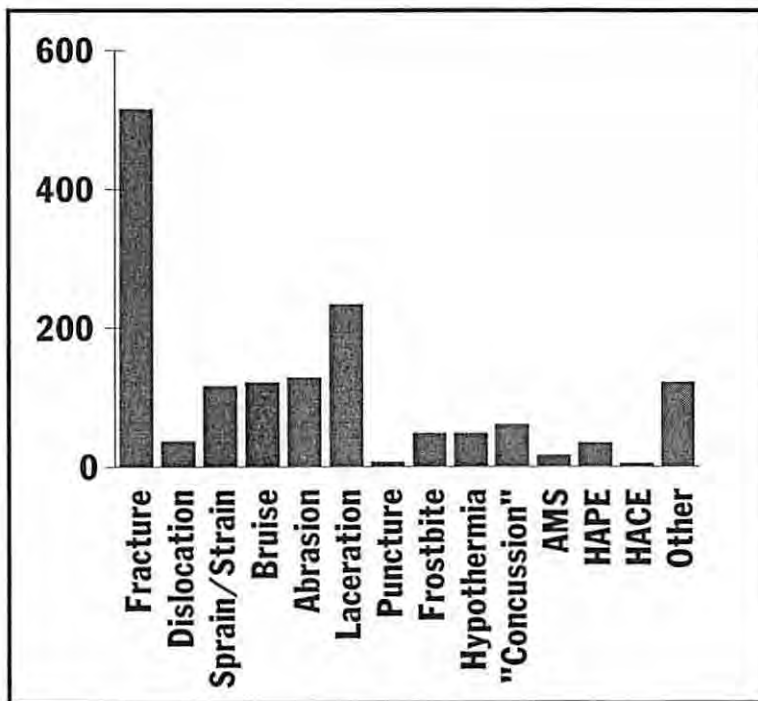
- c. **They all have normal human metabolism.** For long evacuations, you must ensure nutrition and hydration, and provide for elimination.

- d. For the most part, "street" EMTs can legitimately dump these long-term problems on the Emergency Department or Trauma Unit staff. Wilderness EMTs, on the other hand, must recognize and deal with these problems; they simply have no choice if they want to keep their patients alive and (relatively) well.

7. In summary, when we try to compare urban vs. wilderness trauma care, we can see that the similarities are few. The ideas of the "golden hour" and "golden day" make you appreciate the importance of efficient evacuation techniques; even in

the wilderness, rapid transport is still the key to trauma care. To the trauma patient, good evacuation skills are probably more important than good field medical treatment. An hour or two of delay due to an inexperienced litter





**Figure 1:**  
Mountaineering  
Accidents,  
1984-1990<sup>21</sup>

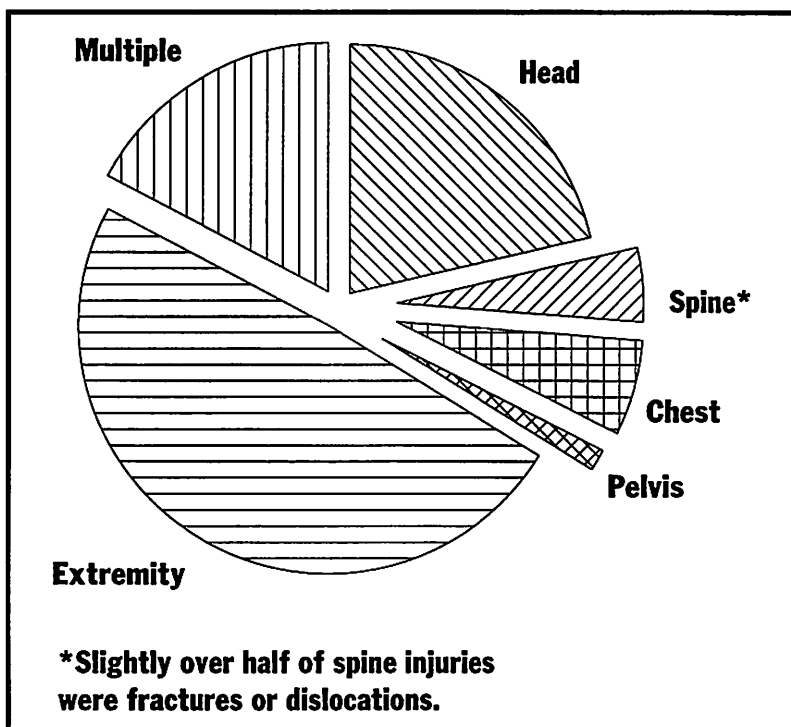
and belay team may mean the difference between life and death. Effective search techniques are just as important. All the medical expertise in the world will be futile in the face of a sloppy, poorly coordinated search. You can't apply your Wilderness EMT skills if you can't find the patient.

### C. Epidemiology and Etiology of Wilderness Trauma

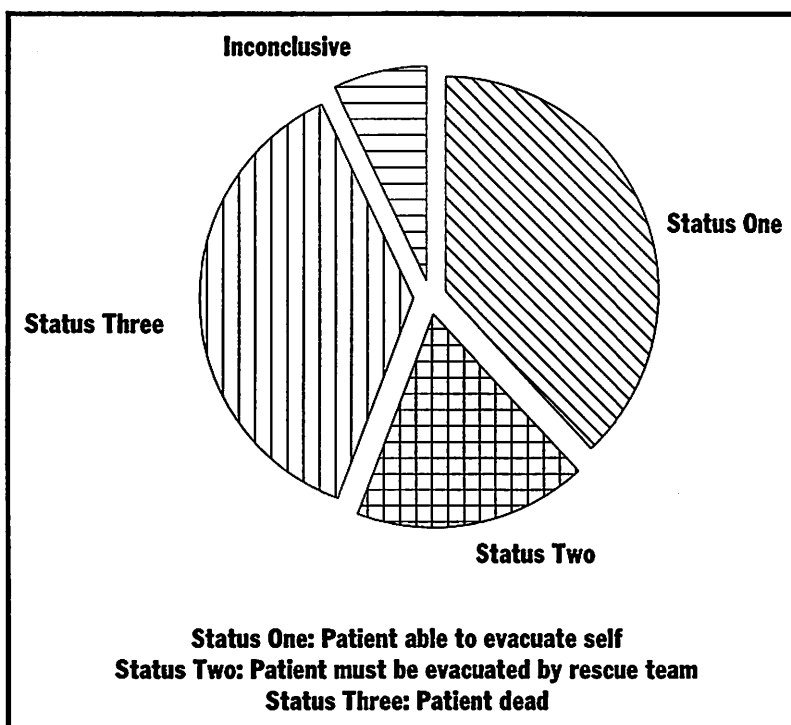
1. "We have met the enemy, and it is us"
2. Most of the "hard" data available on the subject of wilderness trauma has come from the mountaineering and caving journals. Unfortunately, very little has been published about injuries that occur on the trail. The statistical data that is available, though far from comprehensive, tells us that trauma is the most common emergency that presents in the wilderness.
  - a. Not surprisingly, the vast majority of wilderness trauma is the result of a fall. In addition, due to significant time fac-

tors inherent in the wilderness incident, patients frequently develop secondary complications such as hypothermia, infection, and dehydration, which are immediate concerns in wilderness trauma management. As we shall see, establishing "how the patient fell" is the cornerstone upon which our management is built.<sup>5,6</sup>

- b. The periodical *Accidents in North American Mountaineering* offers the most compelling information regarding how accidents occur in the wilderness. A look at the critiques of mountaineering accidents between 1960 and 1981 shows a many have a primary cause of human error. This includes climbing without a helmet, misuse of equipment, inexperience, and surpassing one's abilities.<sup>7,8,9,10,11,12,13,14,15,16,17,18,19,20</sup> The type of injuries reported in *Accidents in North American Mountaineering* have been tabulated from 1984-1990, and are presented in Figure 1.<sup>21</sup>
- c. Schussman cites climber error in 39% of accidents in the Grand Tetons from

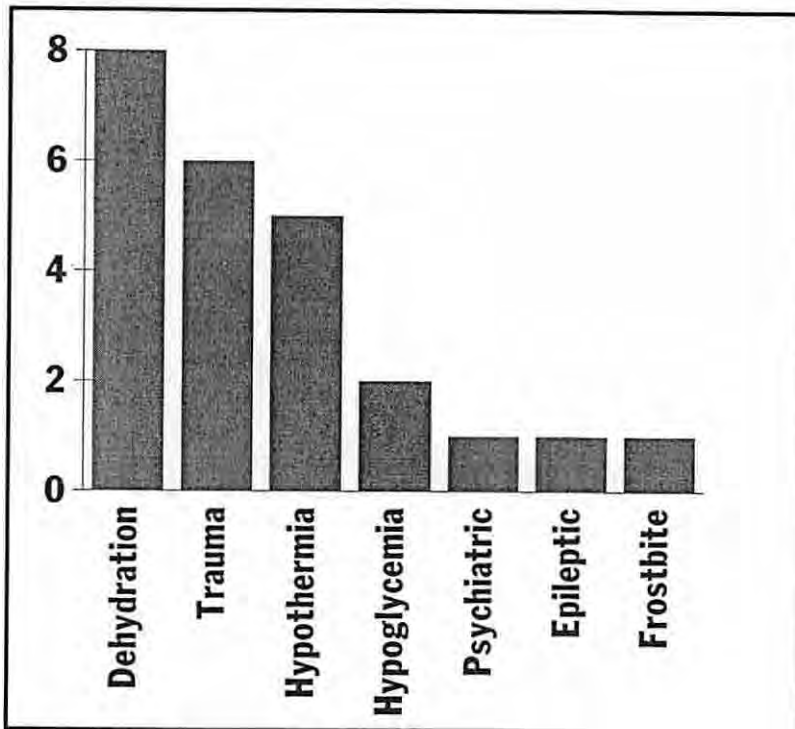


**Figure 3:**  
Climbing  
Accidents,  
Grand  
Tetons<sup>22</sup>



**Figure 2:**  
Search  
Subject  
Status,  
1975-1987,  
ASRC





**Figure 4:**  
**Status Two**  
**Injury/Illness,**  
**1975-1987,**  
**ASRC**

1981-86. Where the information was available from a review board, 50% of the patients whose climbing experience was known had no experience, or climbing experience of less than one year.<sup>22</sup> Weather conditions, as well as other environment factors, definitely influence the frequency and severity of wilderness injuries. Bear in mind, however, that the available data clearly cites human error as the *primary* etiology (cause) of wilderness trauma. Environment factors are usually secondary. In the wilderness we are our own worst enemies. Therefore personal and team safety is the first commandment of wilderness travel (and wilderness rescue!) The type of injuries in Schussman's study are summarized in Figure 2.

- d. One study of climbing injuries in the Sierra Nevada implicated acute mountain sickness and hypothermia in about half of the accidents. The presumption is that these contributed to errors of judgment resulting in acci-

dents.<sup>23</sup> Acute mountain sickness, hypothermia, exhaustion, and fatigue may all cloud the judgment.

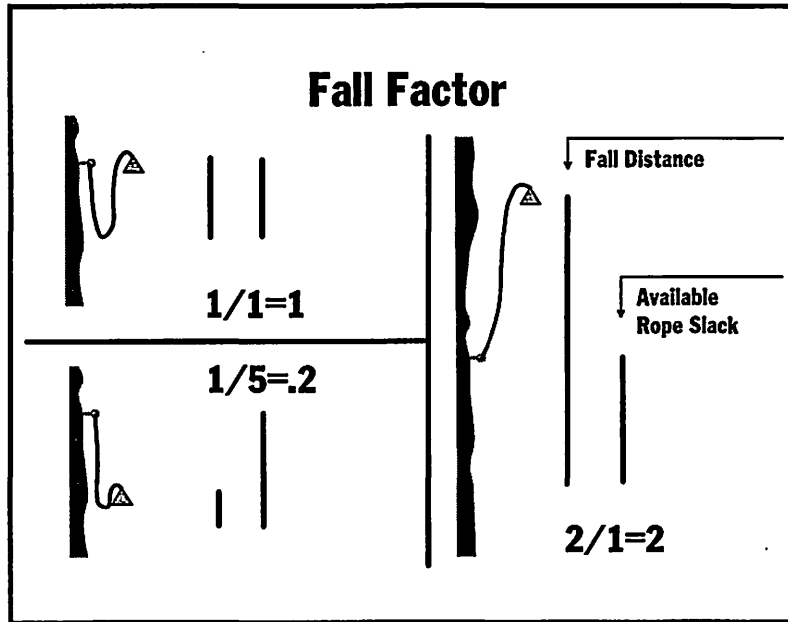
- e. One study, from the U.S. Outward Bound program, shows injury patterns roughly similar to the above studies. Of interest, the author compares the safety of outdoor recreation with that of other sports and finds injury rates similar.<sup>24</sup>

- f. Caving accidents show a similar pattern.<sup>25,26,27,28,29,30,31,32,33,34,35,36,37,38,</sup>

<sup>39</sup> The 1990 edition of *American Caving Accidents* notes the propensity of cavers who "really should know better" to be injured, stranded, or lost. The editor notes a parallel with another dangerous activity: blasting. He quotes from the Blaster's Handbook:

*Personnel designated to handle explosive materials should have intelligence and common sense and be trained in the use of explosives.*

*A small minority, however, will never have the proper attitude to become safe and efficient blasters, no matter how much*



**Figure 5: Fall Factor Calculation**

they are trained or how well they learn the technical aspects of blasting.

Men who, through ignorance, carelessness, or bravado, follow unsafe practice constitute the greatest problem to blasting safety. If a man is careless, reckless, or unwilling to mend his ways, he should be removed from all contact with explosives.

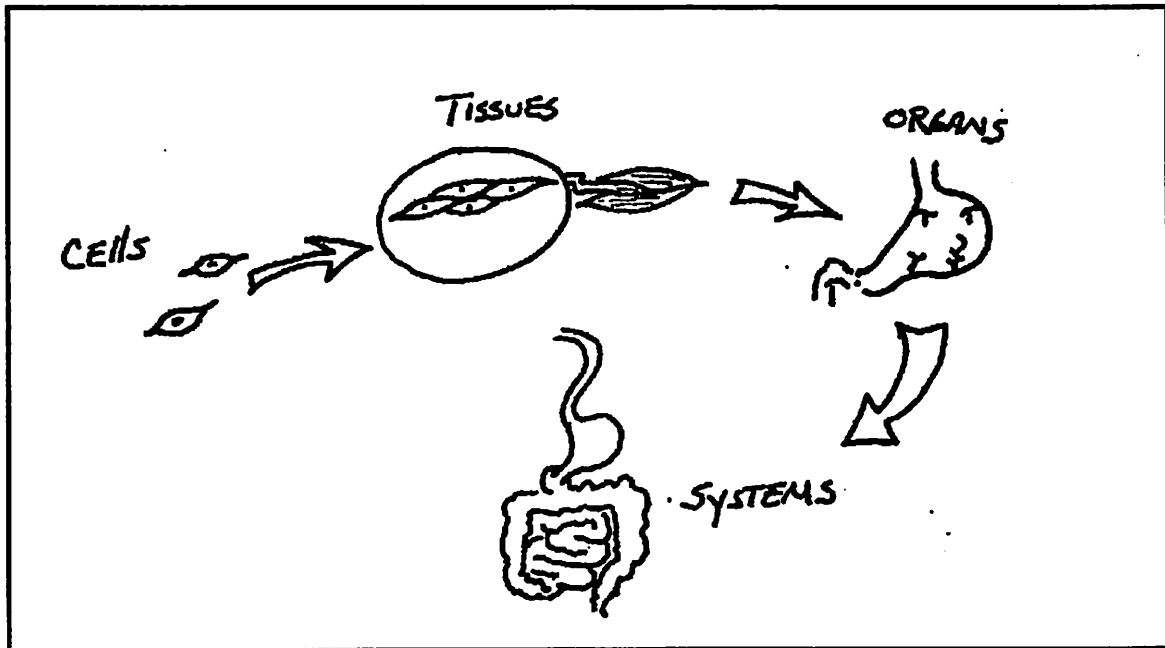
Very few blasting accidents are caused by lack of knowledge or experience. They are more frequently the result of a mental attitude that does not put safety before every other consideration.<sup>40</sup>

- g. Compared with these climbing injury statistics, information gathered by the Appalachian Search and Rescue Conference regarding their search and rescue subjects represents a more general "wilderness search and rescue" patient population. Because of a large number of search subjects not involved in major trauma, dehydration and hypothermia assume larger dimensions. Diabetic hypoglycemia, psychiatric problems, and epilepsy are also disproportionately represented. Trauma from light aircraft crashes, including burns, and gunshot wounds

from hunting accidents are the major sources of trauma in their data. (See Figures 2 and 3.)

#### **D. Mechanism of Injury for Wilderness Trauma**

1. The **mechanism of injury** basically answers the question: "what happened?" Knowledge of how an injury came about provides an invaluable framework from which to direct patient care. In essence, the mechanism of injury suggests the potential for other "unseen" injuries that may not be initially apparent. For example, a patient with leg pain from a five-foot fall would not usually be suspected of having a pelvic or C-spine injury, as opposed to a patient who fell 20 feet. Evaluating the mechanism of injury helps us to determine what injuries to expect, the likelihood of other injuries, and the aggressiveness to which we'll address that likelihood.
2. **Kinematics of wilderness trauma**
  - a. Nature's laws, which govern the world in which we live, act as both ally and enemy to the wilderness traveler. In a conceptual sense, when you look at injuries in the wilderness as consequences of the laws of nature, for instance falling from a cliff and breaking every bone in your



body illustrates the force of gravity. In other words, we examine the extent of an injury based upon physics of the injury— a concept called **kinematics**.

- b. For our purposes, the study of kinematics focuses on two physical quantities, **force** and **kinetic energy**. For those unfamiliar with these physical concepts, here are definitions of these terms:

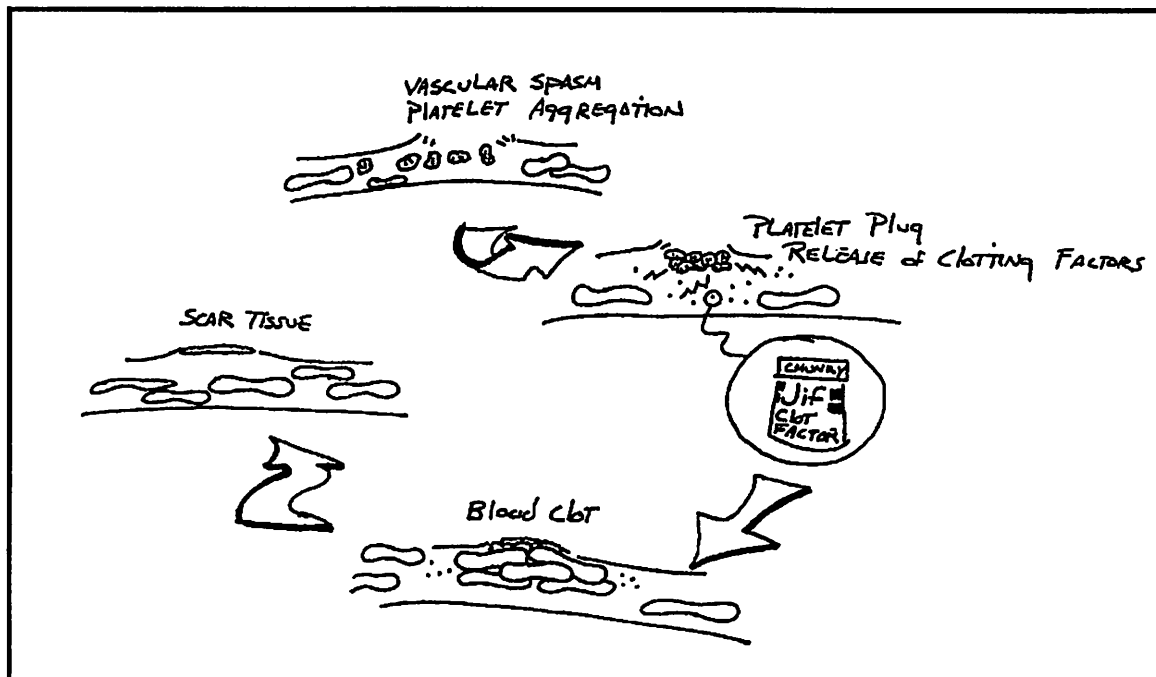
- (1) Force is essentially a push or a pull in a particular direction. (Force = mass x acceleration.)
- (2) Kinetic energy is energy associated with mass in motion. In other words, it measures the amount of energy a push or pull exerts over time. (Kinetic energy = Mass x (Speed)<sup>2</sup>).

- c. These properties operate in accordance with three rules:

- (1) **Newton's First Law:** An object in motion remains in motion until something stops it (even if that something is just friction).
- (2) **Conservation of Energy:** Energy cannot be created or destroyed; it can be stored as potential energy, or transferred from one object to another.
- (3) **Newton's Third Law:** The way this "unusual" law works is this. Imagine pushing

against a brick wall with 20 pounds of force. The 20 pounds of force that you place **on** the wall will be opposed by an equal and opposite force of 20 pounds **by** the wall. In other words, the force you exert on something gets transmitted back in the same amount.

- d. The falls and collisions that cause wilderness trauma often have many components of force and injury being transmitted through the body. We can visualize force and energy as "shock waves" like the kind you see when a stone is dropped into the water. Since the forces we are referring to have large kinetic energies as well as different directions, the resultant effect on the body is one of bending, ripping, and tearing.
- e. In the case of injuries caused by a belayed fall, the waist loops or body harness concentrate the forces on an isolated area. For instance, waist loops cause trauma to the mid-back, the lower ribs, and upper abdominal organs, including the spleen, liver, and kidneys. Harnesses spread out the force over a larger area, including many strong bones, so injuries are less likely.
- f. The C-spine and lumbar spine, commonly injured in climbing falls, have forces applied in two directions. One, the force of gravity pulling down, and two, the tension of the rope as it retards the climber's fall.



- g. Similarly, rock falls transmit their kinetic energy as they impact the body. A rock striking the head is deflected only when the opposing force of the body is large enough to change its path. Often, this occurs only after a fracture has occurred.
- h. Other common mechanisms, such as aircraft crashes and hunting accidents, share the same properties as the previous examples except with much greater magnitude.
- i. When we combine the mechanism with hypothetical kinematics we get a frame of reference from which we can reasonably predict the likelihood of certain injuries. This is the "injury potential."

### E. Physiological Response to Trauma

1. As a species, man's ability to adapt to hostile environments is virtually unmatched. Combined with our intelligent use and improvisation of clothing and shelter, our ability to live in conditions that would kill most animals is due to a complex physiologic balancing act; body fluids are conserved or excreted, changes in respiration

and excretion maintain blood pH; blood vessels constrict and dilate in response to tissue demand and temperature changes; heart rate increases with exercise or stress. In the face of stresses such as major trauma, we find that the body's ability to compensate for change is quite limited. The effects of trauma can result in a domino progression that can bring the body's vital functions crashing down like a house of cards.

2. **The injured state:** As stated, the body is continuously balancing many physiologic processes. Many processes take place within the basic building blocks of the body, the cells.
  - a. Cell+cell+cell... = Tissue
  - b. Tissue+tissue+tissue... = Organ
  - c. Organ+organ+organ... = Organ System
3. When tissue is traumatized, normal processes are interrupted at the injury site: cells are destroyed, blood supply is impaired, and cellular waste products are dumped into the surrounding tissues on a grand scale, causing further tissue disruption. The body in turn mobilizes numerous defense mechanisms to limit the amount of blood loss and tissue damage. These include:
  - a. release of extra "clotting factors";
  - b. the inflammatory response; and

c. *shunting*.

#### 4. Clotting

a. Basically, blood clotting takes place in 4 steps:

- (1) *vascular spasm*;
- (2) *platelet plugging and release of clotting factors*;
- (3) *a blood clot*; and
- (4) *formation of scar tissue*.

b. The body's first response to a break in a blood vessel is to decrease the blood flow to that immediate area. This serves two purposes: to reduce blood loss, and to allow components for clotting to accumulate.

c. Next, platelets accumulate at the site of the break, and stick together to form a **platelet plug**. Platelets are fragments of a cell found in the bone marrow. They are a standard component of blood, and are responsible for the repair of hundreds of small vessel leaks every day.

d. When the hole in the vessel is too big for a simple platelet plug, special activator substances stimulate the formation of a blood clot. The precursors of these substances are a normal part of blood.

#### 5. Inflammation

a. The inflammatory response takes place at the same time as clotting. The site of action, however, is not the same. Clotting takes place in the blood vessel, inflammation takes place in the surrounding tissues. Recall that when tissue is injured or destroyed, cellular waste products are dumped into the surrounding tissues and the circulation. Inflammation restricts the spread of these toxins, by "walling off" the injured area.

b. This is initiated when the injured cells release **histamine** into the surrounding tissue. Unlike the vasospastic response in blood clotting, histamine **increases** blood flow to the surrounding tissues, which allows large quantities of fluid and protein to pass from the blood and lymph vessels into the tissue spaces. Once this occurs, white blood cells (leukocytes) migrate into the tissue spaces, and destroy cellular waste and bacteria.

6. **Shunting:** Shunting is simply a shift in blood supply from one area to another. This takes place

normally between active organs and organs at rest. (For example, more blood goes to your stomach after you eat lunch.) In trauma, however, large-scale rerouting of blood takes place in order to optimally preserve the vital organs. These include the heart, lungs, brain, and kidneys. The entire response of the body to trauma (or change) is called **compensation**. The thing to remember about compensatory mechanisms such as shunting is that they don't last forever. When the consequences of the injury prevail over the body's ability to protect itself, the body begins to decompensate or literally "break down." Herein lies the "central dogma" of trauma care: "preserve the integrity of the vital organs."

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#### F. The Shock State

1. "It ain't the fall that bothers me, it's that sharp stop at the bottom!" —Pogo, et al

2. The standard definition of shock is "inadequate tissue perfusion" caused by a variety of mechanisms. Most definitions list 5 major types.<sup>41,42</sup>

- a. *hypovolemic*;
- b. *anaphylactic*;
- c. *septic*;
- d. *neurogenic*; and
- e. *cardiogenic*.

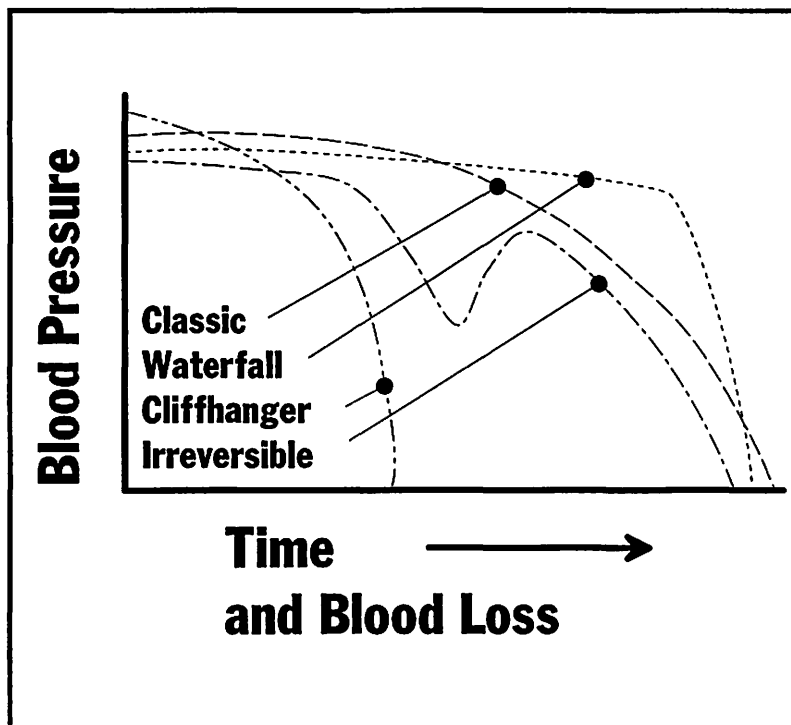
3. Keep in mind that despite the different mechanisms that lead to shock, all of them lead to the same end, which is **inadequate tissue perfusion**. This occurs for three reasons:

- a. *compromise in the pump (heart)*;
- b. *compromise in the pipes (blood vessels)*;
- c. *compromise in the fluid (the blood)*.

4. These can occur separately or together.

#### 5. Perfusion:

a. When we talk of perfusion, we are referring to the ability of red blood cells and plasma to **transport and transfer** oxygen and vital nutrients to the tissues, and to remove waste products. When tissue perfusion is impaired due to any of the compromises (heart, blood, or blood vessels), the cells' metabolic waste prod-



**Figure 6:**  
Different  
Shock  
Responses to  
Blood Loss

ucts begin to build up, which adversely affects nearby cells. The "nutrient" most in demand at this time is oxygen.

- b. When tissue perfusion cannot keep pace with oxygen demand, we call this **ischemia**. Left uncorrected, cellular death soon follows. Let us therefore expand on our "central dogma": **Preserve the integrity of the vital organs (heart, lungs, brain, and kidneys) by maximizing O<sub>2</sub> in the blood, and maximizing the delivery of oxygenated blood to the vital organs.**

### G.Shock and Blood Pressure

1. The blood pressure (BP) is one of the most valuable measures to evaluate the status of circulation and perfusion. However, you must be careful to put the BP in its proper perspective as **one** component of your evaluation for the presence of shock.
2. In some cases, such as well-conditioned athletes, BP's of 90/60 are normal, if not associated with other symptoms. In many cases, however, patients with normal to near-normal BP's after trauma will

deteriorate ("crash") quite suddenly, while others, particularly the elderly, will exhibit gradual declines. Let us examine three models that relate shock to BP.

- a. **Classic Shock:** The classic model of shock is found in our older patients where the blood vessels have lost some resiliency and are not as able to compensate by constricting down about a shrinking intravascular fluid volume. In addition, the older folks are often less able to tolerate a sustained tachycardia. As a result, when the blood volume starts to decrease, from any cause, the blood pressure starts to gradually fall. This classic model of shock, where a falling blood pressure parallels the loss of blood volume, is the model of shock that is most generally accepted among the EMS providers who are not accustomed to management of either the younger folks or trauma (which is a disease largely of the younger folks). We also call this model by a formal name on occasion: Partially Compensated Shock.
- b. **"Waterfall" Shock:** Unfortunately, not all patients exhibit the signs of hypovolemia by a gradual drop in the blood pressure and the regular and orderly development of the associ-

ated signs. In the young, well conditioned patient, blood pressure is maintained within relatively normal limits until about a quarter of the blood volume is lost. The presence of this model of shock correlates with the level of athletic conditioning of the patient. As older people become more active in vigorous athletic training, we can expect to find this model of shock more frequently.

- (1) To a limited extent, we can compensate for faults in the paths of shock and stave off a generalized system failure. We do so in a distinct pattern - the fight or flight mechanism. (This is also known as activation of the sympathetic nervous system. The sympathetic nervous system, along with its companion parasympathetic nervous system, make up the autonomic nervous system. The autonomic nervous system is responsible for maintaining the body's state of relaxation or excitement. The sympathetic nervous system, along with its companion parasympathetic nervous system, make up the autonomic nervous system. The parasympathetic nervous system is important for rest and digestion. The sympathetic nervous system is the "fight or flight" mechanism.
- (2) Faced with a declining intravascular volume, our bodies react by increasing the output of epinephrine and other hormones. In response to epinephrine, the heart beats faster in order to circulate the remaining blood volume better. These hormones also act to increase the vessel tone and make more effective use of the remaining fluid. The blood vessels in the skin and the extremities begin to contract and force the blood into the "core" of the heart, brain, and lungs. The respiratory rate increases and more oxygen is driven into the lungs and the circulation. These actions will maintain the blood pressure and perfusion of the vital organs. This lets us go merrily down the stream as the blood loss continues.
- (3) While this compensatory mechanism is an ideal way of dealing with a small amount of blood loss, it is not so good for larger volumes of fluid loss or other causes of shock. Once the patient has lost between 20 and 25% of the total intravascular volume, the mechanisms

initiated by epinephrine will no longer work, and the patient can no longer compensate for the shed blood.

- (4) If the blood vessels in the extremities stay contracted for too long, the extremities are without oxygen and nutrients. Eventually, the cells of the extremities will begin to die and the toxins produced by their death and hypoxia will enter the blood stream. The extremity blood vessels lose the ability to contract, and the resulting dilation deprives the heart, brain, and lungs of oxygen. The loss of compensation spreads with the release of the cellular toxins and the cardiovascular system collapses.... The blood pressure then precipitously falls and the patient goes over the waterfall. When this happens, the patient is close to death because the body has already used up most of the available compensatory mechanisms.
  - (5) These patients can be recognized more by the potential injuries based upon the mechanism of injury than by physical exam. A significant clue may be the presence of an unexpectedly high pulse rate coupled with a possible severe mechanism of injury. It should be emphasized that many a physician has been fooled by the compensatory mechanisms of the athletic and healthy patient.
  - (6) This model of shock is also known by its formal name: Compensated Shock. As the victim hits the waterfall, the patient has "decompensated." Older EMS folks may call this point "Cardiovascular Collapse".
- c. **"Cliffhanger" Shock:** The last model of hypovolemic shock is the "cliffhanger", which is seen in patients who are taking certain medications or who have a long term disease. These patients may be taking medications for blood pressure disorders or to control the rhythm of the heart. This model is also seen in long term diabetics, those with underlying dehydration, and those with spinal cord trauma. Because the reflexes that aid in the maintenance of the blood pressure are blunted, a small loss of blood volume will produce a disproportionate fall in the blood pressure. It is as if the patient is hanging on a cliff and any little push will send him or her

over the edge. This is the Uncompensated Shock Model. The patient is simply unable to compensate for the loss of blood and develops shock quite quickly.

### 3. Irreversible Shock

a. "Irreversible shock" is a controversial term. Some insist there is no such thing. However, it makes such an important educational point that we will present it.

b. In certain animal studies, experimenter removed blood to cause shock, then replaced it to treat the shock. If they just removed a small bit of blood, the dog became hypotensive, but recovered when the blood was replaced. When more blood was removed and then replaced, the dog at first seemed to get better, but then dropped his blood pressure and died, no matter what was done. The suspicion was that after a certain period of shock, the damage to vital organs was enough to cause death, no matter how good the resuscitation after that point.

c. Does this apply to human shock patients? Maybe, maybe not. But nobody will argue with the idea that the longer someone is shocky, even in mild shock, the more likely he or she will die later on.

4. Clearly, the most common type of shock that affects the trauma patient is hypovolemic shock, particularly from hemorrhage. Hemorrhage is the acute loss of circulating blood. Although not used clinically, the following classification of hemorrhage is widely used to teach the clinical severity of hemorrhage's effects.

a. **Class I Hemorrhage:** This is an acute loss of up to 15% of the total blood volume. In an average 70 kg male, the blood volume is approximately 5 liters. Fifteen percent of this is 750 ml. This amount of acute loss, in the absence of other fluid losses, is well-tolerated and requires no volume replacement. The body should completely replenish this blood volume in about 24 hours.

b. At this point, we should interject the parameters used to evaluate circulatory status:

- (1) pulse rate and strength;
- (2) BP;

(3) respiratory rate and depth;

(4) capillary refill (the "blanch" test);

(5) level of consciousness and anxiety level;

(6) skin color and clamminess; and

(7) urine output.

c. **Class II Hemorrhage:** this is an acute loss of 15% to 30% of blood volume, which translates to approximately 800 to 1500 ml in a 70-kg male. Patients in this range are tachycardic, capillary refill is greater than two seconds, patients may become anxious, and pulses may be weak and thready; systolic BP generally changes only slightly, but you may note a **narrowed pulse pressure**. (The pulse pressure is the difference between systolic and diastolic pressures; a narrow pulse pressure is what causes the weak and thready pulse of shock. Normal pulse pressure is about 40-50.) When the body detects this level hemorrhage, catecholamines (such as epinephrine=adrenaline) are released into the circulation. This helps to prevent a drop in the systolic pressure. But, at the same time, increases the diastolic pressure. This occurs because catecholamines clamp down the peripheral arterioles, thus increasing both resistance and pressure. The majority of these patients will eventually require blood transfusion, but can be stabilized with other replacement fluids.

d. **Class III Hemorrhage:** this is 30% to 40% acute loss (approximately 2000 ml). In the absence of other fluid losses (burns, diarrhea, etc.) this is the smallest amount of blood loss that consistently causes a drop in BP. These patients exhibit all of the previous signs, but to a greater extent, and with a significant drop in systolic blood pressure. Rapid fluid replacement and blood transfusion are immediate priorities.

e. **Class IV Hemorrhage:** is anything more than two liters of hemorrhage in a standard 70-kg male (more than 40% acute blood loss). This amount is immediately life-threatening. You may note very low BP's, very narrow pulse pressure, or absent diastolic pressure. Patients will be pale, very confused or unconscious, and have little to no urinary output. Along with the obvious transfusion requirements, surgical intervention is almost always necessary. It would be virtually



*inconceivable for a patient with a Class IV hemorrhage in the wilderness to survive.<sup>43</sup>*

- f. *Remember that these classes of hemorrhage are based on **blood loss only**. In the wilderness, patients frequently have several routes of fluid loss, which compound any blood loss. This may include dehydration from hot weather, cold diuresis from cold weather, burns, or diarrhea. Bear in mind that the four classes of hemorrhage are not meant to suggest a margin of safety. Recall the waterfall and cliff models for shock. Patients may appear to be doing well initially, only to become very ill very fast. For this reason, our approach to wilderness patients almost invariably includes aggressive fluid replacement.*

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## H. Trauma Assessment

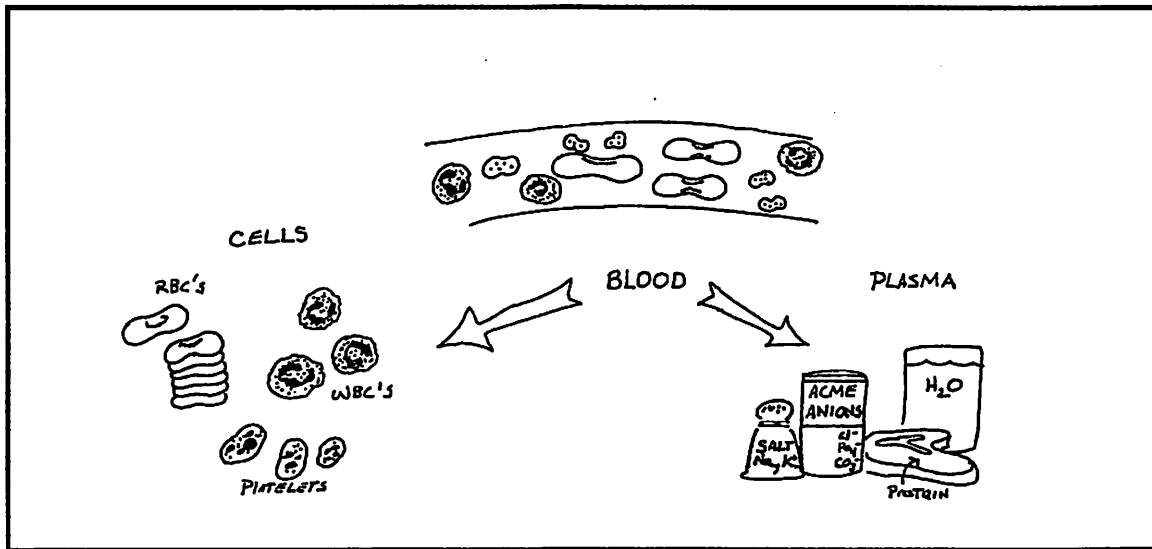
1. Your assessment of a wilderness trauma patient should be organized into three categories:
  - a. the primary survey;
  - b. the shock assessment; and
  - c. the secondary survey or H&P. (Some include shock assessment as part of the primary survey; we don't care how you organize your assessment as long as you do it in an appropriate order.)
2. During your primary survey, don't just check for the presence of a pulse; estimate its strength, as a measure of circulatory status. Similarly, estimate the adequacy and quality of respirations. Does the patient have the deep respirations that show the acidosis of shock, paradoxical respirations indicating a flail chest, or the periodic respirations that indicate head injury (or high altitude).
3. Having ensured adequacy of the "ABC's" for the moment, you must now answer the question: is the patient shocky? If the primary survey has not already suggested this, the questions to answer are these:

- a. Are any of the primary survey findings abnormal? (pulse or respiration quality)
  - b. Is the patient alert? Is the patient anxious? (beyond what the situation warrants.) Always bear in mind that an altered level of consciousness or unreasonable anxiety are often the earliest clues of shock.
  - c. Is the patient tachycardic? (beyond what the situation and the patient's pain warrants)
  - d. Does the patient's skin suggest shock? (cool and clammy; poor color)
  - e. Does the mechanism of injury suggest shock?
4. If the answer to any of these questions is "yes," then you should consider the patient to be in shock until proven otherwise. In assessing for shock, we don't need to prove that it's there; we need to prove that it's **not** there.
  5. Take the time to check the patient properly and identify all problems. Problems that EMT's on the street can ignore ("let the docs in the hospital worry about that...") may grow into severe problems during a long evac.

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## I. Trauma Management

1. Management-wise, the wilderness patient is a "two-edged sword." The patient has survived long enough to be managed, but due to the routinely long evacuation time, these patients have high likelihood of deteriorating in transit. The medical team therefore must be diligent in monitoring the patient's vital signs and neurological status. Let us again restate the central dogma: **Preserve the integrity of the vital organs (heart, lungs, brain, and kidneys)** by maximizing O<sub>2</sub> in the blood, and maximizing the delivery of oxygenated blood to the vital organs.



2. **The fluid challenge:** A basic concept in trauma resuscitation is the **fluid challenge** or **fluid bolus**. The idea is to give a certain standardized volume of fluid, then to stop **and reassess the patient**. The patient's response to this bolus gives you a good assessment of his or her volume status. A patient who responds to a single bolus, and who has no known continuing blood loss, can be put on maintenance fluids and watched carefully for new signs of shock. Additional fluid boluses can be given if needed. The importance of this concept is that it gives you a definite endpoint to stop and reassess the patient. The standard size of fluid boluses are generally 20cc/kg for pediatric patients. For adults, a one liter bag is somewhere between 10 and 20 cc/kg; standard boluses are one or two liters. Smaller boluses may be used, as well; some trauma surgeons use boluses of 250 or 500 cc.<sup>44</sup>

- a. Patients who respond to one or a few fluid boluses have usually lost less than 20% of their volume. They can be put on maintenance fluids of about 150 cc/hr.
- b. Many patients, however, tend to deteriorate once the infusion rate is cut

back to maintenance. They have some source of ongoing fluid loss (e.g., pelvic fractures or burns) and you should give additional fluid boluses as needed. You should look for any possible way to hasten the evacuation (e.g., calling for a helicopter.) These patients need blood as soon as possible. **If they're not improving, get moving!**

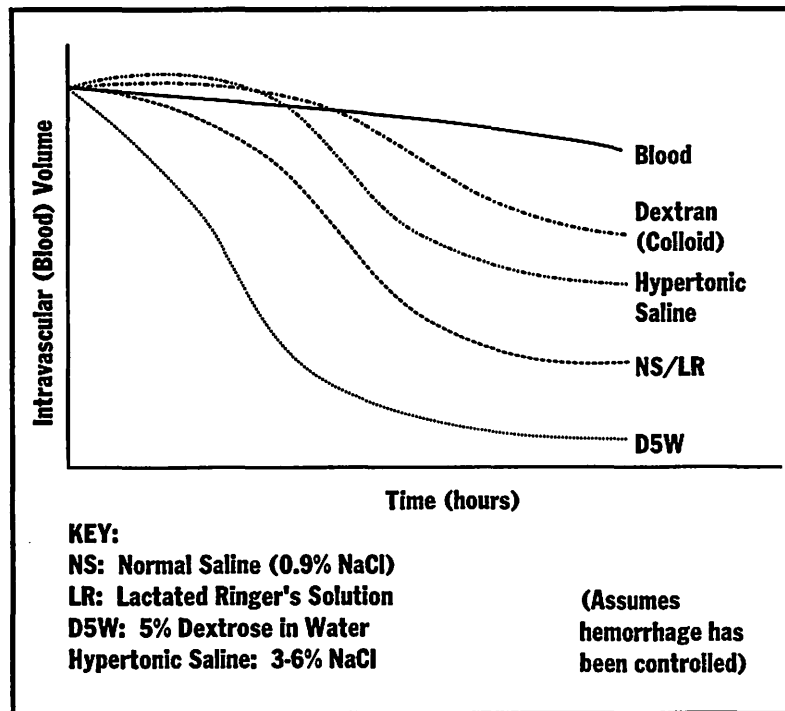
### J. Controlled vs. Uncontrolled Hemorrhage

1. Current research in trauma management is focusing on the differences between controlled and uncontrolled hemorrhage. Evidence is starting to suggest that fluid resuscitation in the field may actually make outcomes **worse** for urban trauma patients. Even though giving fluid to a patient with uncontrolled hemorrhage may increase the blood pressure, there is also an increase in blood loss. This is simply replacing thick blood with thin crystalloid.
2. Due to long transport times, those in the wilderness who have uncontrolled hemorrhage will die regardless of therapy. A reasonable assumption therefore

is that wilderness trauma patients all have controlled hemorrhage. Therefore studies of uncontrolled hemorrhage do not apply to wilderness patients.

### K. Fluids

1. If you are an EMT-Basic, and you can't start IV's, you may wonder why we insist on teaching you all about IV fluids. There are several good reasons. You may keep caring for the patient after the EMT-P is gone or incapacitated. Or, you may need to choose the fluids to carry into the site for an EMT-P who is already there (this is especially true in cave rescue).
2. Let's review vascular physiology (which is new for EMT-Basic's). Please recall the previous diagram. Remember that the cells of the body are essentially one big sponge: the cells absorb fluids and give them up again. The big questions to answer about giving fluids are: Where do you want the fluid to go? and, How long do you want it to stay there? The circulatory fluid is made up of blood and plasma.
  - a. blood = blood cells + plasma
  - b. plasma = water + salts + protein
  - c. As a general rule, the more stuff you put into water, the longer it will stay in the intravascular space. This stuff can be salt, sugar, or protein. The bigger the particles dissolved in the fluid, the longer the fluid stays in the intravascular space.
  - d. Plasma tends to leak out of blood vessels into the tissue; "oncotic pressure" (osmotic pressure from plasma proteins) tends to keep water in the vessel.
  - e. If you put water into a vein (e.g., D5W, where glucose is quickly "eaten up") it is quickly distributed among all the body water (a matter of minutes), in and out of the cells, so little stays in the blood vessels.
  - f. If you put salt water into vein, it distributes first through the blood, and in about an hour, into the tissue water but **not** into the cells. (NS or LR takes an hour or less to distribute this way.) The osmotic pressure of the salt helps keep the fluid in the blood vessels.
  - g. If you put plasma or albumin in a vein, it tends to stay in the blood. The oncotic pressure of the plasma or albumin is, in a sense, even stronger than the osmotic pressure of salts.
  - h. All bets are off if capillaries are leaky, leaking protein (e.g., in burns, or in ARDS) or even leaking blood cells (from trauma).
3. When you determine the need for IV fluids, the fluids you can use fall into three groups: crystalloid, colloid, and blood.
4. **Crystalloids:** Crystalloids are so named because they are solutions of salt crystals dissolved in water. Crystalloids are routinely considered "first-line" resuscitation fluids, and include lactated Ringer's solution ("LR") and 0.9% NaCl ("normal saline," "NS"). NS is called "normal saline" because it is roughly "isotonic" with blood. This means it has roughly the same amount of salt as the blood does, even though the salts may not be exactly the same. (NS is just table salt in water.) Recently, **hypertonic saline** (7.5% or 9% NaCl), usually mixed with the colloid dextran, has been introduced for resuscitation. In the case of LR and NS, both are about equivalent in their effect on shock and hypotension. Essentially, you have to give three liters of LR or NS to get roughly the same effect as giving a liter of whole blood.\* Unlike blood, which contains cells and colloids, NS and LR only contain salts and water. These tend to diffuse from the blood vessels out into the tissues, because they dilute the existing



**Figure 7:  
Volume  
Effects of  
Intravenous  
Fluids**

cells and colloids in the blood.<sup>45</sup> Hypertonic saline is currently the object of considerable study for prehospital use. The basic attraction of this fluid is the very rapid response achieved with volumes as small as 200 cc in patients with severe but (now) controlled blood loss. Although still inconclusive, the basic mechanism appears to be a drawing-in of extracellular fluid (fluid outside the cells) into the vascular space. Once the fluid is in the vascular space, the Dextran keeps the fluid from leaking back out. Investigations strongly suggest an increased survival in patients receiving 7.5% or 9% saline/6% dextran in controlled hemorrhage. In uncontrolled hemorrhage, however, it was noted that the hypertonic saline made the hemorrhage worse. The benefits of a 200 cc bottle of hypertonic saline/dextran over five liters of NS should be immediately

obvious to any WEMT's who has ever carried a medical pack.<sup>46,47</sup> Dextran also has advantages for treating patients with hypothermia and frostbite; see the section on *Cold-Related Disorders*.

- a. Hypertonic saline (very concentrated: 7.5% or 9% compared with 0.9% for Normal Saline) is being studied as a resuscitation fluid. It has a great theoretical advantage for wilderness rescue: since wilderness rescuers can only carry a few liters of fluid because of its weight, carrying hypertonic saline could make the rescue team's few liters more effective in resuscitation. One animal study<sup>48</sup> showed that hypertonic saline was more effective than normal saline in resuscitating rats, **provided** that the hemorrhage was controlled: rats whose arterial bleeding was uncontrolled did worse with hypertonic saline than normal saline,

\* For historical reasons that need not concern us, a "unit" of whole blood contains 450 cc of blood, or roughly half a liter of blood.

but when the arterial wound was compressed by a MAST pressure suit, the hypertonic saline was better than normal saline. While more research needs to be done, especially on the long-term (i.e., wilderness) consequences of hypertonic saline resuscitation, some wilderness EMS medical directors may reasonably include hypertonic saline as part of the WEMT's medical kit.

5. Although quite safe for the most part, all crystalloids share major disadvantages.

- a. **They don't carry O<sub>2</sub>, they have no clotting factors, and the more crystalloid you give over a short period of time, the more you dilute the blood that is still circulating.** Particularly in massive fluid resuscitations, it's plausible that a patient can have more than enough "volume" when replaced with crystalloid, but may eventually reach a point where that volume doesn't have enough "blood" character, such as O<sub>2</sub> carrying or clotting ability, to maintain adequate perfusion.
- b. This compromise in clotting ability is called coagulopathy. It can also result from massive clotting associated with major trauma; clotting factors are simply used up. Paradoxically, this problem with clotting can actually result in disseminated intravascular coagulation (DIC). In DIC, clots form in blood vessels all over the body, causing harm in many different organ systems. There is no way to manage this in the wilderness except to give blood plasma.

#### 6. Colloids

- a. Colloids, unlike crystalloids such as D5W, NS, and LR, are not true solutions. Colloids are so-called because they are colloidal suspensions, rather than true solutions, because the colloid particles are too large to truly dissolve. Since the colloidal particles are suspended in the water well-enough

that they might take years to settle out, you might wonder why we bother making the distinction. There is, indeed, an important reason for making the distinction.

- b. The salts in crystalloids will stay within the blood vascular system for an hour or so. After this, the salts equilibrate across the capillary walls into the interstitial water, taking water along with them. After this, only about 1/3 of the salt and water you infused is in the vascular system; the other 2/3 is in the interstitial space.
- c. Colloidal suspensions, on the other hand, contain such large molecules that they will hold their water in the vascular space for an indefinite time. The colloidal particles simply can't get through the capillary walls. (The only exception is when the capillary walls are damaged, as in burns or ARDS.)
- d. Common colloids are plasma, albumin, and dextran.
  - (1) Plasma is what is left of blood after you take out all the cells. It contains a wide variety of proteins, including albumin, clotting factors, and antibodies. Since it is a blood product, it has the same danger of transmitting infectious disease as does blood itself (see below). Plasma must be refrigerated or frozen for preservation, and should be given within a few hours of thawing.
  - (2) Albumin is the major blood protein. Giving albumin has the same effect as giving plasma, as far as expanding the blood volume. However, it does not replace clotting factors. Unlike plasma, which contains certain fragile proteins, albumin may be heat-treated to remove the danger of infectious disease. Albumin need not be refrigerated, but has a limited shelf life.
  - (3) Dextran and related plasma substitutes are suspensions of artificial

starch molecules. Dextran is cheaper than albumin, and has a longer shelf life. Even if your wilderness EMS unit doesn't use hypertonic saline as discussed above, a few bags of dextran might make a reasonable addition to a field medical kit. Especially if combined with some crystalloid, they will give more effective fluid resuscitation than if you used an equivalent volume of all crystalloid.

### 7. Blood and Blood Products

- a. Blood is unquestionably the fluid of choice for moderate to severe hemorrhage. Essentially, blood is the same as tissue, and in fact, some authors view blood as the equivalent of an organ.
- b. However, the same properties that make blood so desirable make it potentially dangerous. Although it has specialized structure and function, human tissue cells are not universally interchangeable. If not a reasonable match, the body interprets the new cells as "foreign" and begins a very aggressive process of defense to eliminate the "new" cells called rejection. This is an event which can be immediately life threatening and is characterized by sudden fever, restlessness, anxiety, flushing, tachypnea, tachycardia, hypotension, and coma. This is called a hemolytic reaction. We therefore restrict the use of blood transfusion to what is referred to as a universal donor: type O negative.
- c. Each individual has a unique set of markers on the surface of his or her red blood cells: the **blood type**. If you don't have a particular marker, then you will develop an allergic reaction if

you are given blood with one of these markers on it.

- d. The most well-known markers are A, B, and Rh. Someone who is AB+ has all three markers. This AB+ person can receive blood from anyone: a universal recipient. Someone who is O- has none.\* O negative blood is "universal donor" blood; it can be given to anyone without a worry about a severe allergic reaction.
- e. *Actually, things aren't quite that simple. The AB and Rh antigens are the major antigens that you can find on your blood cells, but there are many other "minor" antigens, too. In the hospital, the blood bank will test the donor's blood against that of the recipient, looking for reactions caused by these minor antigens. You may have heard someone in a hospital talking about sending blood for "type and crossmatch"; cross-matching is actually testing the two bloods together for reactions.*
- f. Many infectious diseases can be transmitted by blood: hepatitis, AIDS, and syphilis, for examples. Blood banks test blood for Hepatitis A, Hepatitis B, Hepatitis C, AIDS, syphilis, and other diseases. They also look for elevated levels of liver enzymes that could indicate other forms of hepatitis. None of these tests are 100% reliable, so blood transfusion always carries at least a small risk of infectious disease. With testing for AIDS and Hepatitis C, though, blood transfusions are much safer than a few years ago.
- g. In the hospital, many people who receive blood get "packed cells," that is, mostly red blood cells without much plasma. For trauma resuscitation and for possible wilderness use, though whole blood is the product of choice, since plasma contains important pro-

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\* The nomenclature is confusing; people are either Rh positive (have the antigen) or Rh negative (don't have the antigen). Someone who has blood type AB has both A and B antigens, but someone who has blood type "O" has neither A nor B antigens.

teins for those in shock after blood loss. For prehospital use, the standard is to carry O- "universal donor" blood. There is some risk of a transfusion reaction from one of the minor antigens, but blood is so important in resuscitation that the risk is worth the benefit.\*

- h. As a general rule, you give three liters of crystalloid followed by one unit (500 cc) of whole blood. Once you establish IV access, you transfuse blood under constant monitoring for reaction. You should warm all IV fluids and insulate the bags and tubing. If the patient shows signs of a transfusion reaction, **stop the transfusion**. Give an antihistamine if the patient develops hives or other symptoms of a generalized allergic reaction.
- i. In certain situations, you may even be able to send out blood for typing, as for example, in a long cave rescue. (In some cave rescues, you can get to the patient easily but it may still take many hours or days to bring the patient out.)
- 8. "Pressors" are drugs that have some effects similar to adrenaline (epinephrine).
  - a. These drugs are used to increase blood pressure in an attempt to restore perfusion. Examples include dopamine, dobutamine, and norepinephrine. They act by increasing the heart rate and heart contractility, and by causing vasoconstriction.
  - b. In volume-depleted or hypothermic patients, however, pressors may make the patient worse, even though they

raise the blood pressure. They may divert blood away from organs such as the GI system, resulting in death of these organs. Use of these drugs in the trauma patient is a last-ditch effort and generally useless. The only exception is that small doses of dopamine may actually direct blood to the kidneys and protect them from damage from lack of blood flow. However, the practical difficulties of providing a carefully-regulated rate of dopamine infusion make this impractical in almost all wilderness rescues.

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## L. Trauma Complications

### 1. Ischemic Brain\*\*

- a. Acute ischemia from low blood pressure may interfere with thinking (as may hypothermia, fatigue, or exhaustion); increasing ischemia may result in fainting.
- b. Prolonged ischemia may cause irreversible damage (the "4 to 6 minute" rule of basic CPR: the brain can only tolerate a few minutes without perfusion before irreversible damage). It's hard, however, to determine whether, after a period of ischemia, coma will last for hours, days, or forever. There is no way to make this determination in the field. If you can deliver such a patient to the ED comatose but with a stable BP, good oxygenation, and a good urine output, there is a chance that the patient will recover, perhaps even completely.<sup>49</sup>

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\* Universal donor blood is safe to give in the prehospital setting, as shown by a prospective study of 50 patients, performed by Allegheny General Hospital of Pittsburgh, which was presented at an aeromedical meeting in October 1986.

\*\* Other problems of long evacuations, including DVT's, PE's, infections, nutrition, waste management, comfort, analgesia, pulmonary care, and psychological aspects, are covered in the sections on *Principles of General Medicine* and *Wilderness Medical Problems*.

- c. **Provide adequate perfusion and oxygenation to the brain. If you don't do this, nothing else matters.**

## 2. Kidneys

- a. The general rule for all wilderness patients is "keep the urine output at least 30 to 50 cc/hr." (Or, for children, 1-2 cc/kg/hr.) Why? Because urine output is a sensitive indicator of perfusion, much better than blood pressure or pulse. However, there are other causes of a decreased urine output. Since you must place a Foley catheter

- b. Your differential diagnosis of decreased urine output should include the following:

- (1) Obstruction of catheter may come from a kink in the tubing or something pressing on it (e.g., tubing under the patient in the Stokes litter). If the patient has blood in the urine, a clot may form in the tubing. If you have some clean water or saline (sterile is preferred but not absolutely necessary), you can use a catheter-tip syringe to irrigate the Foley catheter and perhaps remove the clot.
- (2) Anatomic obstruction of the ureters (or less often the urethra) may come from trauma. Though rare, a large hematoma in the area of the kidneys could cause this.
- (3) Acute renal failure may cause decreased urine output. (Some other kinds of acute renal failure cause a decrease in the excretion of waste product, but don't interfere with the excretion of fluid.) In a trauma patient, hypotension or shock may cause acute renal failure. This may persist even after perfusion is restored to the kidneys. The diagnosis of renal failure in the field will be difficult. Decreased urine output despite other signs and symptoms of adequate perfusion suggests renal failure. Decreased urine output with

signs of fluid overload strongly suggests renal failure.

- (4) Decreased perfusion to kidneys, whether from intravascular volume depletion or cardiac failure, can cause decreased urine output.

## c. Crevice Entrapment

- (1) Entrapment with crush injury is described in the next section. Entrapment with crush injury is a common scenario in urban disasters. However, it is possible for a patient to be entrapped with minimal or no crushing. Such simple entrapment is most prevalent in cave rescue. An example is caver stuck in crawlway, or who has slid down into a "keyhole" crevice. Despite the dangers associated with crush injury, simple entrapment is still deadly. Several North American cavers have died from simple entrapment in the past decade. Some died of hypothermia, but even with autopsy results it's not clear why the others died.
- (2) Men and women tend to become entrapped differently, because of differences in bony anatomy. Women generally have larger hips than men; this is an advantage in child-bearing. It is also an advantage for entrapment in a crevice. Men, instead of getting wedged in by their hips as do women, instead get their chest wedged in. This means that men tend to have more trouble breathing than do women; this can lead both to poor oxygenation and to panic that leads to increased O<sub>2</sub> demand. The urgency of rescue efforts for a simple entrapment should always be high, but even higher for men than for women.
- (3) Simple entrapment is often complicated by other factors:
  - (a) lack of access: you may not even be able to take vital signs, or to start IVs, or even to give oral fluids;





**Figure 8:  
Caver  
Trapped  
in  
Keyhole  
Passage**

- (b) hypothermia -- from conduction due to large contact area with rock;
  - (c) dehydration -- from lack of access to fluids;
  - (d) blood clots in the legs (deep venous thrombosis) or lungs (pulmonary embolism) from immobilization; and
  - (e) psychological problems: panic, and claustrophobia
- (4) Medical treatment for simple entrapment depends on the exact situation, but the following principles generally apply:
- (a) Provide warmth.
  - (b) Provide food and water, and IV fluids if possible.
  - (c) To prevent deep venous thrombosis and pulmonary embolism:
    - i) encourage the patient to move his or her legs on a regular basis to prevent clots;
    - ii) consider giving a half tablet of aspirin, because it will act as a mild blood thinner and may help prevent deep venous thrombosis; and
    - iii) consider dextran or subcutaneous heparin, as long as the patient has had no traumatic injury that might start bleeding again. Both dextran and SQ heparin help prevent deep venous thrombosis.
  - (d) Consider sedation. A dose of haloperidol, while it is known to predispose slightly to hypothermia, might be appropriate as long as you are providing adequate amounts of heat to the patient.
- d. Crush Injuries: Crush Syndrome, Myoglobinuria, and Hyperkalemia<sup>50,51,52</sup>**
- (1) Crush injuries and entrapment in collapsed buildings were common in the bombing of London during World War II ("the blitz"). Many people were found alive but trapped. Indeed, the people seemed to be doing quite well, having survived days of entrapment. However, as the rubble was taken off their crushed limbs, many suddenly died. Of those who survived, many died a few days later of kidney failure. We now know

why this happens and can help prevent it.

**(2) Crush Syndrome**

- (a) When a person is trapped under a rock in a cave, or in a building collapse, rescue too often ends with sudden death. Though the person has survived days of entrapment, the sudden release from entrapment allows "evil humors" (various poisonous waste products, including potassium and lactic acid) to escape into the blood. The entrapped limb may also act like a sponge, soaking up precious intravascular fluid, causing sudden shock. This "crush syndrome" is well-recognized, and can be prevented. However, in disasters, many rescuers are not medically trained, and don't know to prepare the patient properly for release, so deaths still occur.
- (b) The stress of release from entrapment may also contribute to renal failure (kidney failure). Renal failure can occur even if the patient is still producing urine. Renal failure causes death over the course of several days, as waste products build up in the blood. The effects of renal failure may be staved off by using artificial kidneys (dialysis), and this may be enough to allow the kidneys to recover on their own. Dialysis can only be performed with specialized equipment in medical facilities.
- (c) You can help prevent hyperkalemia, acidosis, shock, and renal failure by preventing dehydration. If a patient is already dehydrated from long entrapment, **rehydration** prior to release is essential. Intravenous fluids are ideal but you won't always have them. If the entrapped person does not have an ileus, oral fluids may be acceptable. (See the section

on *Burns and Lightning* for more about ileus.)

- (3) **Myoglobinuria:** With crush injuries to muscle and other soft tissues, and with severe burns, large amounts of myoglobin are released into the circulation. This is also called "rhabdomyolysis," meaning breakdown (lysis) of skeletal muscle (rhabdomyo). Myoglobin is an O<sub>2</sub>-carrying molecule found in muscles. It is similar to the hemoglobin molecule found in red blood cells. Myoglobin and hemoglobin are toxic to the kidney. (Hemoglobin in red blood cells and myoglobin in muscle cells are necessary, but hemoglobin and myoglobin are toxic when free in the blood. After lightning strikes, hemoglobin may be released from thousands of damaged red blood cells. The "loose" hemoglobin can cause problems similar to myoglobin.) When dehydration causes concentration of urine in the kidney, the high levels of myoglobin or hemoglobin in the urine are known as myoglobinuria and hemoglobinuria. Myoglobin is dark brown, and with myoglobinuria, the urine looks very much like tea. Hemoglobin is a bit more red, but still dark.
- (4) If you are caring for or transporting a patient, and based on the mechanism of injury you suspect myoglobinuria or hemoglobinuria, check the patient's urine. If it is brown or tea-colored, start treating for possible myoglobinuria. While the exact treatment is up to your Wilderness Command Physician, here is a common protocol:
  - (a) Increase intravenous fluids as necessary to maintain a urine output of 100 cc/hr (4 cc/kg/hr in children) unless there are definite signs of fluid overload. Note that mannitol and furosemide (see below) may increase urine output considerably.

Thus the patient may require large amounts of intravenous fluids to "keep up:" if the urine output is more than 100 cc/hr., measure the urine output each hour and increase the IV rate as needed to match this.

- (b) If plenty of intravenous fluids are available, or if the patient has no ileus, has no contraindications to oral fluids, and can take large amounts of oral fluids, give the following medications. Do **not** give these medications unless you will be able to keep the patient hydrated despite a large urine output. They will cause diuresis (increased urine output) and thus decrease the concentration of myoglobin in the urine and in the kidney. If you will be treating the patient with oral fluids only, you may give the dose of furosemide orally. Increase intravenous fluids as necessary to match the patient's urine output, or to maintain the specified minimum urine output. Note also that, once you give these medication, you cannot use the urine output to gauge the patient's fluid status; the diuretics will keep the urine output high even if the patient gets dehydrated. You must **match** the urine output with appropriate amounts of intravenous fluids to keep the patient from dehydrating.

- i) Add 12.5 grams of mannitol (half of a 25 gram prefilled syringe) and 44-50 mEq of sodium bicarbonate to every liter of IV fluid until the urine clears. <sup>\*53,54,55</sup>

- ii) Give a single dose of 20 mg of furosemide intravenously (or orally; see above). This dose needs to be increased if the patient has been on a diuretic before and has developed resistance to diuretics.

- (5) **Crush Hyperkalemia:** Cell fluid is unlike blood and other body fluids, in that cells contain lots of potassium. The blood potassium is normally regulated at a very low level.\*\* Higher levels quickly cause arrhythmias, cardiovascular collapse, and death.

- (a) When many cells are damaged, they will release their potassium into surrounding tissue fluid. If the area is perfused, this potassium will diffuse into the blood. Release of potassium from damaged cells generally takes about 24 hours or a bit longer. If you are facing a prolonged cave evacuation, but where rescuers can get to and from patient quickly, you may want to draw a tube of blood and send it to the hospital for electrolytes. In the more common situation where the patient has massive crush injuries, you do not know the patient's potassium level. If so, at about 18 hours after the time of injury, change to IV solutions with no potassium (i.e., NS instead of LR.

- (b) Release of potassium may be delayed if a limb is trapped under a heavy weight and is ischemic. The potassium may not reach the circulation until the rock or rubble is lifted from the limb, at which there may be a massive surge of potas-

*Amputation -  
TK*

\* The specific recommendations on urine output and mannitol/bicarbonate come from the American Burn Association's Advanced Burn Life Support program. The mannitol will cause diuresis, and the bicarbonate will make the urine alkaline, promoting excretion of the myoglobin or hemoglobin.

\*\* The normal blood potassium is 3-5 mEq/100cc compared with 140 mEq/100cc of sodium.

sium into the blood, causing cardiac arrest. If you can, hydrate with NS (not LR) before and after removing the rock or rubble. (A healthy adult can tolerate 2 liters of saline given over a few minutes without any signs of fluid overload. Patients who are dehydrated can probably tolerate more.)

### 3. Gut Ischemia

- a. Fear, pain, or ischemia may cause an ileus (lack of normal peristaltic movements moving food and fecal material along the GI tract. See the section on *Burns and Lightning* for more on ileus). You should not give fluids to a patient with an ileus.
- b. However, if you are facing a long evacuation, you can hear normal bowel sounds, and the patient says he or she is passing gas, you may want to provide PO fluids and nutrition. (Remember that IV dextrose only provides a minimal amount of food energy. You are also limited in the IV fluids that you can carry, but you can prepare oral fluids by disinfecting local water.) Fluid needs are more important than food needs; nutrition is important, but hydration is **vital**.
- c. If severe and prolonged, bowel ischemia may lead to the necrosis of parts of the bowel. This is usually fatal.

4. **Coagulopathy** (abnormal blood clotting): recognize if coagulopathy occurs, but there is no treatment in the field. Signs of coagulopathy would be IV "stick" sites starting to bleed again, spontaneous nosebleeds, and bleeding under the skin. Coagulopathy is probably rare in wilderness trauma patients. The major cause is urban trauma is giving massive amounts of anticoagulated bank blood.

5. **Resuscitation Hypothermia:** Resuscitation hypothermia is a common problem in urban trauma. It is caused by giving

large amounts of relatively cool IV fluids and blood. The patient's depressed metabolism also plays apart. As a WEMT, you will probably never evacuate a patient who requires such massive infusions. These patients will be dead at the scene. However, you will probably see hypothermia from environmental conditions in almost all your patients.

6. **Glycogen Depletion:** Glycogen depletion is common in wilderness patients.

- a. The patient's glycogen may be depleted from pre-existing exhaustion or hypothermia. Poor coordination and poor judgment from exhaustion and hypothermia may be the immediate cause of an accident that results in a wilderness rescue.
- b. The stress of trauma increases glycogen use, and may make depletion very rapid, even if there was no depletion prior to the accident.
- c. If a trauma patient can reasonably take fluids, and the evacuation is prolonged, oral food supplementation is the best way to replete glycogen reserves. (See the section on *Principles of General Medicine* for more about oral fluids for wilderness patients.) If the evacuation is short, or if the patient can't take PO fluids, give food through the IV in the form of dextrose. Use only fluids with D5 (D5NS, D5LR) and add two amps of D50 to every other bag of IV fluids (which brings it up to 10% dextrose).

### 7. Compartment Syndrome

- a. Compartment syndrome is caused by blunt trauma to a muscle compartment. Muscle compartments are groups of muscles bounded by walls of tough fibrous tissue. The most common compartment to develop compartment syndrome is in the anterior compartment of the lower leg.
- b. The natural history of compartment syndrome as follows.

- (1) Swelling from trauma causes pressure in the compartment to build up.
  - (2) The pressure finally gets to be more than the pressure inside the veins; the veins collapse, and blood can no longer leave the compartment. This sets up a vicious cycle where increasing pressure holds the veins more tightly closed, causing increased pressure, which holds the veins even more tightly closed, and so forth.
  - (3) When pressure in the compartment exceeds the pressure in the capillaries, muscle perfusion stops; this will cause severe muscle pain and tenderness over the compartment.
  - (4) The increasing pressure damages sensory nerves traveling through the compartment, so you will find numbness over the compartment and distal to it (e.g., in the web space of big toe for the calf compartment).
  - (5) As pressure climbs, it next damages motor nerves (e.g., the patient can't move the big toe).
  - (6) Finally, arteries traversing the compartment collapse from the pressure. (E.g., the patient may lose the dorsalis pedis pulse.)
- c. Diagnose compartment syndrome by looking for the following:
- (1) severe pain, swelling, and tenderness;
  - (2) a progression of findings: the patient loses sensation distally, then loses motor strength distally, and finally, loses the distal pulse.
- d. Definitive diagnosis can be made by placing a needle in compartment and directly measure the pressure (this is not practical in field because the equipment is seldom available.)
- e. Definitive treatment of compartment syndrome is simple: fasciotomy. A fasciotomy is a surgical operation involves cutting open the skin and the fascia to relieve the pressure. Fascia

are the gristly membranes that enclose muscular compartments in the legs and arms. However, the operation is difficult, and best performed by experienced surgeons. (Fasciotomy is more difficult than the similar operation of escharotomy, which is discussed in the section on *Burns and Lightning*.) If you think a patient has compartment syndrome, and you face a long evacuation, try to have a surgeon brought in to operate on the leg. Other first aid treatments have been suggested for compartment syndrome: elevation and cold. There is little or no evidence that either is effective, and at least some reason to suspect that each might actually be harmful.

#### **8. Adult Respiratory Distress Syndrome**

- a. Adult Respiratory Distress Syndrome is commonly known by the abbreviation ARDS, and was often called shock lung.
- b. ARDS is caused by capillaries in the lung leaking protein and fluid into the alveoli. Shock seems to cause some sort of damage to the cells lining the capillaries so they become leaky. ARDS usually occurs roughly 24 hours after trauma and shock.
- c. Diagnosis of ARDS is simple but not easy. First, if a patient develops classic signs of pulmonary edema about 24 hours after shock or injury, you must suspect ARDS. (Signs of pulmonary edema include râles in the bottom of the lung fields, shortness of breath, and cough sometimes productive of frothy sputum). If you've been giving the patient fluids, you must also suspect fluid overload. With fluid overload, you will generally be able to find some pedal or presacral edema, or abnormally distended neck veins (see below). If you are at high altitude, you must also suspect High Altitude Pulmonary Edema (HAPE). (See the sec-

tion on *Altitude Illness* for more on HAPE.)

d. Management of ARDS, like its diagnosis, is simple but not easy.

- (1) Give high-flow oxygen. Hypoxia is the major problem in ARDS.
- (2) Have the patient use pursed-lip breathing (see the section on respiratory illnesses in *Wilderness Medical Problems* for more about this).
- (3) If supplemental O<sub>2</sub> and pursed-lip breathing are not enough to keep the patient in good respiratory status, use positive pressure ventilation (i.e., a bag-mask). Converting the normal negative pressure in the lungs into positive pressure will help keep fluid-filled alveoli stretched open so that gas exchange can occur more readily.<sup>56</sup>
- (4) Keep the patient dry but not shocky. By this, we mean that you should be very careful not to give the patient any more fluid than needed to eliminate signs of shock. Monitoring the urine output every hour is an excellent aid to your management of the patient's fluid status.

#### 9. Fluid overload

- a. When you're giving fluid to a patient with dehydration, blood loss, or burns, you might accidentally give more than the patient can handle. Most people can excrete large amounts of fluid via the kidneys, so fluid overload usually occurs only when the patient has underlying problems (e.g., congestive heart failure) or when you are giving massive amounts of fluids very fast.
- b. In a wilderness rescue, amount of fluid we can take to the patient is usually limited. And, the likelihood of wilderness patients having congestive heart failure or similar problems is small. Therefore, fluid overload is less of a problem in the wilderness than in the Emergency Department. However,

patients who have are dehydrated, or who have suffered crush injury or burns, are at risk for developing acute renal failure. In some kinds of renal failure, the kidneys may not be able to excrete fluids normally. Such patients are at risk of fluid overload even with minimal IV fluids.

c. Fluid overload is a simple clinical diagnosis. By this we mean that laboratory tests and X-rays are not as important as your physical exam. This means that you should be able to diagnose fluid overload reliably in the wilderness. There is no one sign that is guaranteed to signify fluid overload, but the following four physical signs all strongly suggest it.

- (1) You may hear **râles** in the lower parts of lungs with the patient sitting or standing. As the fluid overload gets worse, the râles spread to include all lung fields. Râles are also heard in pneumonia and ARDS.
- (2) You may see **distended neck veins** because the veins are over-full with fluid.
  - (a) Normally, when you sit or stand up, your neck veins collapse because the pressure in the central veins is low. (A normal central venous pressure is about 5-10 cm above the heart. If you wish, you can use a ruler to measure the central venous pressure by measuring the vertical distance from the top of the column of blood in the neck veins to the heart.) When you lie down, though, the neck veins fill up with blood. If you were fluid overloaded, your central venous pressure would be high, and your neck veins would not collapse.
  - (b) Neck veins are difficult to see in some patients. You can see them best by shining a penlight or headlamp beam obliquely across the lateral neck. This makes them cast

shadows.\* The examination of the neck veins is best learned by practice. We encourage you to practice on your friends and family. You should also pay careful attention to neck veins during your clinical training in the Emergency Department. For more information on neck veins and other details of physical diagnosis, you should refer to Barbara Bates' excellent textbook.<sup>57</sup>

- (c) To examine the neck veins of a litter patient, first examine the neck with the litter flat. Have the patient turn his or her head slightly to the opposite side, if the neck isn't immobilized. Find the neck vein. Now, have the litter team tilt the head of the litter up about 30° and recheck the neck veins. If the vein doesn't go flat, you know the central venous pressure is more than normal. To see how high it is, you can ask the litter team to tilt the litter even higher, until you can see the neck vein finally go flat. (In a patient who isn't in a litter, you can simply ask the patient to sit up.)
- (3) An **S3 gallop** is an abnormal heart sound heard in cases of fluid overload, congestive heart failure, and sometimes with a myocardial infarction. (See the section on *Patient Assessment* for more on the S3 gallop.)
- (4) If a person has fluid overload for several hours, the excess fluid finds its way into the skin. The fluid leaks out of capillaries in the lowest part of the body. If the person is walking or sitting, this will be the feet and ankles

(pedal edema). If the person is confined to bed or in a litter, this will be in the lower back (presacral edema). Most edema is "pitting edema" If you can press your finger into the skin and it leaves a dent, we call this "pitting edema." Your finger momentarily presses out the edema fluid from the skin. With your patient packaged in the litter, it's hard to see the back. It's much easier to reach your hand in, press on the back for a few seconds, and then carefully feel if you've left a dent.

- d. Treatment of fluid overload is straightforward.
  - (1) Stop IV fluids.
  - (2) If necessary, administer a diuretic such as furosemide (e.g., Lasix®).

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### M. Accidental Hypothermia and Trauma

- 1. In some people's minds, the wilderness management of hypothermia and multiple trauma might conflict. For instance, some say that hypothermia might be protective for head injuries, and rewarming a hypothermic patient with a head injury might worsen the outcome of the head injury. Others worry that rewarming might make bleeding worsen. This is not true.<sup>58</sup>
- 2. Despite these worries, we can find no evidence to support the idea that trauma should change the treatment of hypothermia as described in the section on *Cold-Related Disorders*. In the field, add as much heat as possible. This is

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\* What we are describing here is examining the external jugular vein. Examining the internal jugular vein is more reliable but more difficult. The internal jugular vein does not protrude on the surface of the neck like the external jugular. Instead, you must recognize the level of the blood column by recognizing the "fluttering" of the venous pulse just behind the carotid arterial pulse.

true regardless of whether the patient has sustained trauma. (See the section on *Cold-Related Disorders* for more about hypothermia.)

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### N. The Wilderness Trauma Exam

1. This is just to supplement the general information on physical exam discussed in the section on *Patient Assessment*, and to emphasize specific important points of the trauma exam.
2. **General Assessment:** Check the skin color and general appearance of patient (OK? sick? very sick?).
3. **Head, Ears, Eyes, Nose, Throat**
  - a. Palpate the head carefully.
  - b. Check pupils and extraocular motions.
  - c. Determine if the face is stable (wiggle the upper jaw). If unstable, don't place an NG tube or endotracheal tube through nose, because it might go into the brain.
  - d. For an awake patient, ask if the dental occlusion is normal ("Do your teeth fit together right?")
  - e. Look carefully in the ears, nose, and throat for blood or CSF.
4. **Neck:** Palpate for tenderness, deformity, and for muscle spasm. (A detailed discussion of cervical spine injury is found in the section on *Wilderness Surgical Problems*.)
5. **Chest**
  - a. Assess air entry by listening.
  - b. Percuss both sides.
  - c. Check for anterior/posterior and lateral stability by pressing with both hands.
  - d. Palpate for point tenderness indicating rib fractures.
  - e. Listen to the heart sounds.

6. **Abdomen:** Note the general contour and appearance, presence or absence of bowel sounds, and palpate for guarding, tenderness, or masses.
7. **Pelvis:** Check for stability (press in and out on the pelvic wings, then press down on the pubis).
8. **Genital/Rectal**
  - a. Expose the genitals (an absolute must) and look for bleeding from the urethra.
  - b. If you suspect pelvic trauma, do a gloved rectal exam: feel for a high-riding prostate in men (a contraindication to a Foley) and check for gross blood. (Musculoskeletal exams are covered in the section on *Wilderness Surgical Problems* and neurologic exams are covered in the section on *Patient Assessment*.)

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### O. Continuing Trauma Management

1. Management of the trauma patient over an extended period in the wilderness includes fluid management, nutrition, monitoring vital signs including level of consciousness and urine output, and good general nursing care. This is all covered in the section on *Principles of General Medicine*.
2. Specific priorities for extended management of the trauma patient, include the following:
  - a. Control bleeding. (Use a temporary BP cuff tourniquet as described above if you need to better direct your direct pressure on the bleeding site).
  - b. Estimate fluid losses, and replace them cc for cc; give maintenance fluids as explained in the section on *Principles of General Medicine*.
  - c. Fluids are more important than nutrition over periods on the order of days. (See *Nutrition, Convalescence, and Re-*



cuperation in the section on *Principles of General Medicine* for making decisions on whether or not to give oral fluids.)

- d. Maximize brain perfusion pressure. If the patient is head-injured, keep the head up to keep ICP down, but also keep the feet up slightly to keep the BP up; this is hard to do in a litter, so you need to position the patient based on the patient's problems and response to tilting head-up.
- e. Give O<sub>2</sub>, assure a good airway and effective ventilation. For head injury, over-ventilation may bring the blood CO<sub>2</sub> down, constrict brain blood vessels, and reduce cerebral edema.
- f. No tourniquets. (See the section on *Wilderness Surgical Problems* for exceptions.)
- g. MAST: use the MAST garment only temporarily, i.e., about 1.5 hours, otherwise, it might cause irreversible ischemic damage to legs. (The MAST garment is not designed for use longer than an hour or so.) You may find the MAST garment helpful in patients with only mild dehydration or mild shock when they must be raised vertically out of a pit or lowered vertically down a vertical cleft. Inflate the MAST prior to raising, slowly deflate the MAST right after leveling the stretcher again, monitoring the blood pressure carefully. (Since this goes against the standard "street" teaching that EMTs never deflate the MAST in the field; you need to have on-line medical command or specific standing orders for this.)<sup>59,60,61</sup>

## Glossary

**Albumin:** The predominant protein in blood plasma.

**Alveoli:** The small terminal air sacs in the lung.

**Anaphylaxis:** A massive immediate allergic reaction characterized by swelling of the mucous membranes of the airways and shock. Histamine is a major component of anaphylactic shock.

**ARDS:** Adult Respiratory Distress Syndrome. Also known as "shock lung." Caused by capillaries in lung leaking protein (and thus fluid) into normally-air-containing areas (alveoli). ARDS sometimes occurs about 24 hours after trauma.

**Autonomic Nervous System:** As opposed to the somatic nervous system that provides sensation and movement, the autonomic nervous system is responsible for controlling the state of internal organs. Activation of the sympathetic portion of the autonomic nervous system causes an elevation in blood pressure, an increase in heart rate, and generally makes the body ready for "fight or flight." The parasympathetic nervous system is responsible for rest and digestion.

**Bolus:** A specific amount of a drug or fluid given rapidly as a single dose.

**Capillary:** The smallest blood vessels. Site of gas and nutrient exchange.

**Cardiogenic Shock:** Pump failure. Generalized poor perfusion from inadequate cardiac output, usually from a myocardial infarction.

**Catheter-tip Syringe:** A syringe that has a large tip of a size to fit nasogastric (NG) tubes, Foley urinary catheters, and chest tubes. Most syringes have a "Luer-Lock" tip that fits needles and intravenous tubing.

**Coagulopathy:** Abnormal blood clotting.

**Colloids:** Fluids that contain water and suspended particles such as proteins. These particles do not settle out because they are so small, but are not truly "dissolved." Examples include blood plasma, dextran (a type of starch suspension) and albumin (a common blood protein).

**Compartment Syndrome:** Swelling of one of the muscular compartments of a leg or less commonly an arm, leading to pressures so high that nerves are damaged and perfusion is cut off.

**Compensation:** The use of defense mechanisms to return the body to a normal state.

**Conservation of Energy:** Energy cannot be created or destroyed; it can be stored as potential energy, or transferred from one object to another.

**Crystalloid:** A fluid containing water and salts. Normal Saline and Lactated Ringer's solution are examples of crystalloids.

**D5W:** An intravenous solution of 5% dextrose (glucose) in water.

**Decompensation:** Failure of defense mechanisms to cope with external stresses.

**Dialysis:** the diffusion of solute molecules through a semipermeable membrane, passing from the side of higher concentration to that of the lower; a method sometimes used, in patients with defective renal function, to remove from the blood elements that are normally excreted in the urine.

**DIC:** Disseminated intravascular coagulation.

**Differential Diagnosis:** A list of possible causes of a particular sign, symptom, or combination of signs and symptoms.

**Disseminated Intravascular Coagulation:** DIC. Clotting throughout the body as a result of illness or injury.

**Diuretic:** A medication that causes an increased urine output.

**Epidemiology:** The statistical study of injury and illness. Used to assess diseases, medical treatment, and preventive measures.

**Epinephrine:** Also known as adrenaline. The major hormone in the blood responsible for the sympathetic "fight or flight reaction" (sweaty palms, tachycardia, nervousness).

**Etiology:** The cause of a medical or surgical problem.

**Fasciotomy:** Cutting open the fascia. Fascia are the gristly membranes that enclose muscular compartments in the legs and arms. Used to relieve pressure from compartment syndrome.

**Foley:** A Foley urinary catheter.

**Foley Catheter:** A catheter placed through the urethra into the urinary bladder. Foley catheters may be placed to measure urine output, to keep bedding or Stokes packaging dry, or to allow urine to pass in cases of urinary retention.

**Glucose:** Also known as dextrose. The simplest kind of sugar. It is the only kind the brain can use, and is the form of sugar found in the blood. Other more complex or different sugars are found in foods (e.g., lactose=milk sugar, fructose=fruit sugar, sucrose=table sugar).

**Glycogen:** "Animal starch." A readily available energy store found in muscle and liver.

**Golden Day:** Refers especially to aircraft crashes. The number of survivors markedly decreases over the first 24 hours after a crash.

**Golden Hour:** For every half hour a patient is in shock, the mortality doubles. The patient is much more likely to survive if appropriate surgical treatment is started in the first hour.

**Hematoma:** A collection of blood in soft tissues.

**Hemolytic Reaction:** An allergic reaction to incompatible transfused blood that causes lysis (rupture) of red blood cells. This causes severe illness.

**Histamine:** A substance found in certain cells in the skin and blood. When released, histamine causes inflammation (bronchial constriction, skin and mucous membrane swelling, and itching). Histamine effects are blocked by antihistamine drugs.

**Hormone:** A chemical released in one part of the body that travels through the blood and has an effect in a distant site. Epinephrine is a hormone, as is hydrocortisone.

**Hyperkalemia:** high potassium in the blood.

**Hypertonic Saline:** A salt solution more concentrated than that found in blood.

**Hypotension:** Low blood pressure.

**Hypovolemia:** Low blood volume. Normal for an adult is 5-6 liters.

**Hypoxia:** Inadequate oxygen in the blood.

**Ileus:** Lack of normal peristaltic movements moving food/fecal material along through the stomach and intestines. Caused by illness or trauma.

**Inflammation:** A set of defenses operating at the tissue level. Inflammation classically causes "rubor, tumor, calor, dolor, and functio laesa" (Latin for redness, swelling, warmth, pain, and decreased function). These mechanisms protect the area from further injury and infection, and help institute healing.

**Injury Potential:** The likelihood of particular injuries for a particular mechanism of injury.

**Intravascular:** Within the blood vessels.

**Ischemia:** When tissue perfusion cannot keep pace with oxygen demand.

**Kinematics:** The physics of trauma. Particularly, the ways that the laws of motion can help explain injury.

**Kinetic Energy:** Energy associated with mass in motion. In other words, it measures the amount of energy a push or pull exerts over time. Kinetic energy = Mass x (Speed)<sup>2</sup>.

**Leukocytes:** white blood cells.

**LR:** Lactated Ringer's. An intravenous solution of several salts similar to those found in blood.

**MAST:** Military Anti-Shock Trousers. Sometimes refers to Military Assistance to Safety and Traffic, where military helicopters are used for civilian emergency medical transport.

**Mechanism of Injury:** The physical forces and other external factors that contribute to a particular injury.

**Myoglobin:** An O<sub>2</sub>-carrying molecule found in muscles. It is similar to the hemoglobin molecule found in red blood cells. When released in large amounts after a crush injury, it is toxic to the kidney.

**Myoglobinuria:** High levels of myoglobin in the urine. May cause renal failure.

**Necrosis:** Death of tissue.

**Neurogenic Shock:** Shock caused by dilation of blood vessels. This may come from direct spinal injury (actually, to the paraspinal sympathetic nerve ganglia), or from a sudden outflow of nerve impulses after a psychological shock.

**Newton's First Law:** An object in motion remains in motion until something stops it (even if that something is just friction).

**Newton's Third Law:** The way this "unusual" law works is this. Imagine pushing against a brick wall with 20 pounds of force. The 20 pounds of force that you place on the wall will be opposed by an equal and opposite force of 20 pounds by the wall. In other words, the force you exert on

something gets transmitted back in the same amount.

**NS:** Normal Saline. An intravenous solution of 0.9% sodium chloride (table salt). It is "normal" because it is similar in salt concentration to blood.

**Oncotic Pressure:** Osmotic pressure from plasma proteins.

**Osmotic Pressure:** Pressure tending to move fluids across a semi-permeable membrane such as blood vessel walls. Caused by salt and other dissolved and suspended chemicals. Osmotic pressure makes water move from the less concentrated side across the membrane to the more concentrated side, thus diluting the more concentrated side.

**Parasympathetic Nervous System:** The parasympathetic nervous system, along with its companion sympathetic nervous system, make up the autonomic nervous system.

**Pedal Edema:** Swelling in the skin around the ankles. Generally from congestive heart failure, renal failure, liver failure, or fluid overload.

**Perfusion:** The ability of red blood cells and plasma to transport and transfer O<sub>2</sub> and vital nutrients to the tissues, and to remove waste products.

**Pitting Edema:** If you can press your finger into the skin and it leaves a dent, we call this "pitting edema." Your finger momentarily presses out the edema fluid from the skin.

**Plasma:** The fluid component of blood, excluding white and red blood cells and platelets.

**Platelets:** Small cell fragments found in large numbers in the blood. Their primary function is to plug leaks in blood vessel walls.

**Presacral Edema:** Swelling in the skin of the lower back. Generally from congestive heart failure, renal failure, liver failure, or fluid overload. Seen instead of pedal edema in those restricted to bed rest.

**Pressors:** Also known as vasopressors. Pressors are drugs that increase blood pressure. Dopamine, dobutamine, epinephrine, and norepinephrine (e.g., Levophed®).

**Protein:** The basic building block of living things. Made up of intricately folded chains of small molecules called amino acids. Though there are only about 20 amino acids, there are millions of different proteins. Muscles are made of proteins, many hormones are proteins, and enzymes are proteins.

**Pulmonary Edema:** Fluid in the lungs. The fluid oozes out of small capillaries. This may be caused by increased pressure in the capillaries from congestive heart failure, or from damage to the capillary walls, as in ARDS.

**Pulse Pressure:** The difference between systolic and diastolic pressures.

**Râles:** Soft crackles in the lungs. May be simulated by rubbing some of your hair between two fingers

(next to your ear). Generally signifies pulmonary edema.

**Renal Failure:** Kidney failure.

**Rh Type:** One of the most important blood types. A person's blood is RH positive or negative. People with RH negative blood will have an allergic reaction against RH positive blood if it is transfused.

**Rhabdomyolysis:** breakdown of skeletal muscle. This leads to release of myoglobin into the blood. Myoglobin is excreted in the urine (myoglobinuria) which may lead to kidney failure.

**Septic Shock:** A type of shock caused by massive infection. Sepsis is characterized as "warm" shock, because there is a high cardiac output and dilated peripheral blood vessels.

**Shunting:** A shift in blood supply from one area to another.

**Sympathetic Nervous System:** The fight or flight mechanism. The sympathetic nervous system, along with its companion parasympathetic nervous system, make up the autonomic nervous system.

**Urine Output:** Normal urine output is 1-2 cc/kg/hr for children, or about 30-50 cc/hr for an average adult.

**Volume Depletion:** Same as hypovolemia. Low blood volume.

## References

1. Committee on Trauma ACOS. *Advanced Trauma Life Support Instructor Manual*. Chicago: American College of Surgeon, 1989:3.
2. Campbell JE. *Basic trauma life support: Advanced prehospital care*. 2nd ed. Bowie, MD: Brady, 1988:108.
3. Williamson JE, Reader R, eds. *Accidents in North American Mountaineering 1983*. New York, NY: American Alpine Club, 1983:29-33,45,46,49,68.
4. Williamson JE, Reader R, eds. *Accidents in North American Mountaineering 1981*. New York, NY: American Alpine Club, 1981:17,21,24,28.
5. Williamson JE, Reader R, eds. *Accidents in North American Mountaineering 1983*. New York, NY: American Alpine Club, 1983:35-44,50-67.
6. Williamson JE, Reader R, eds. *Accidents in North American Mountaineering 1981*. New York, NY: American Alpine Club, 1981:28-40.
7. Safety Committee AAC. *Accidents in North American Mountaineering 1960*. New York, NY: American Alpine Club, 1960:35-6.
8. Safety Committee AAC. *Accidents in North American Mountaineering 1969*. New York, NY: American Alpine Club, 1969:32.
9. Safety Committee AAC. *Accidents in North American Mountaineering 1973*. New York, NY: American Alpine Club, 1973:31.

10. Safety Committee AAC. *Accidents in North American Mountaineering* 1974. New York, NY: American Alpine Club, 1974:22.
11. Williamson JE, ed. *Accidents in North American Mountaineering* 1976. New York, NY: American Alpine Club, 1976:20.
12. Williamson JE, Whalley E, eds. *Accidents in North American Mountaineering* 1977. New York, NY: American Alpine Club, 1977:50.
13. Williamson JE, Whalley E, eds. *Accidents in North American Mountaineering* 1978. New York, NY: American Alpine Club, 1978:44.
14. Williamson JE, Reader R, eds. *Accidents in North American Mountaineering* 1981. New York, NY: American Alpine Club, 1981:62.
15. Williamson JE, Reader R, eds. *Accidents in North American Mountaineering* 1982. New York, NY: American Alpine Club, 1982:70.
16. Williamson JE, Reader R, eds. *Accidents in North American Mountaineering* 1983. New York, NY: American Alpine Club, 1983:80.
17. Williamson JE, Whitteker J, eds. *Accidents in North American Mountaineering* 1988. New York, NY: American Alpine Club, 1988:74.
18. Williamson JE, Whitteker J, eds. *Accidents in North American Mountaineering* 1989. New York, NY: American Alpine Club, 1989:86.
19. Williamson JE, Whitteker J, eds. *Accidents in North American Mountaineering* 1990. New York, NY: American Alpine Club, 1990:74.
20. Williamson JE, Miskiw O, eds. *Accidents in North American Mountaineering* 1991. New York, NY: American Alpine Club, 1991:64.
21. Williamson JE, Miskiw O, eds. *Accidents in North American Mountaineering* 1991. New York, NY: American Alpine Club, 1991:65-6.
22. Schussman LC, Lutz LJ, Shaw RR, Bohnn CR. The epidemiology of mountaineering and rock climbing accidents. *J Wild Med* 1990;1(4):235-48.
23. McLennan JG, Ungersma J. Mountaineering accidents in the Sierra Nevada. *Am J Sports Med* 1983;11(3):160-3.
24. Paton BC. Health, safety and risk in Outward Bound. *J Wild Med* 1992;3(2):128-44.
25. Breisch RL, ed. *American Caving Accidents - 1967-70*. Albuquerque, NM: Speleobooks, 1974.
26. Breisch RL, ed. *American Caving Accidents - 1972*. Albuquerque, NM: Speleobooks, 1975.
27. Breisch RL, ed. *American Caving Accidents - 1973*. Albuquerque, NM: Speleobooks, 1975.
28. Breisch RL, ed. *American Caving Accidents - 1974*. Albuquerque, NM: Speleobooks, 1976.
29. Breisch RL, ed. *American Caving Accidents - 1975*. Albuquerque, NM: Speleobooks, 1977.
30. Knutson S, ed. *American Caving Accidents - 1976-79*. *NSS News* 1981;39(5 [Part 2]):A1-82.
31. Knutson S, ed. *American Caving Accidents - 1980-81*. *NSS News* 1983;41(3):99-118.
32. Knutson S, ed. *American Caving Accidents - 1982*. *NSS News* 1983;41(10):255-65.
33. Knutson S, ed. *American Caving Accidents - 1983*. *NSS News* 1984;42(11 [Part II]):347-58.
34. Knutson S, ed. *American Caving Accidents - 1984*. *NSS News* 1985;43(11 [Part II]):355-67.
35. Knutson S, ed. *American Caving Accidents - 1985*. *NSS News* 1986;44(11 [Part II]):399-410.
36. Knutson S, ed. *American Caving Accidents - 1986*. *NSS News* 1987;45(11 [Part II]):386-98.
37. Knutson S, ed. *American Caving Accidents - 1987*. *NSS News* 1988;46(12 [Part II]):475-90.
38. Knutson S, ed. *American Caving Accidents - 1988*. *NSS News* 1989;47(12 [Part II]):314-34.
39. Knutson S, ed. *American Caving Accidents - 1989*. *NSS News* 1990;48(12 [Part II]):331-49.
40. Knutson S, ed. *American Caving Accidents - 1990*. *NSS News* 1990;48(13 [Part II]):330-57.
41. Simeone FA. Shock. *Christopher's Textbook of Surgery*. Ed. Davis L. Philadelphia: Saunders, 1964:58.
42. Mattox KL, Moore EE, Feliciano DV. *Trauma*. Norwalk, CT: Appleton & Lange, 1988:139.
43. Committee on Trauma ACOS. *Advanced Trauma Life Support Instructor Manual*. Chicago: American College of Surgeon, 1989:59-62.
44. Committee on Trauma ACOS. *Advanced Trauma Life Support Instructor Manual*. Chicago: American College of Surgeon, 1989:65.
45. Mannix FL. Hemorrhagic shock. *Emergency medicine: Concepts and clinical practice*. Rosen P, Baker FJ II, Barkin RN, Braen GR, Dailey RH, Levy RC. St. Louis: C.V. Mosby Company, 1988:195.
46. Maningas PA. Resuscitation with 7.5% NaCl in 6% dextran-70 during hemorrhagic shock in swine: Effects on organ blood flow. *Crit Care Med* 1987;15(12):1121-6.
47. Maningas PA, Mattox KL, Pepe PE, Jones RL, Feliciano DV, Burch JM. Hypertonic saline-dextran solutions for the prehospital management of traumatic hypotension. *Am J Surg* 1989;157:528-34.
48. Landau EH, Gros O, Assalia A, Krausz MM. Treatment of uncontrolled hemorrhagic shock by hypertonic saline and external counter-pressure. *Ann Emerg Med* 1989;18(10):1039-43.
49. Abramson NS. Brain resuscitation. *Emergency medicine: Concepts and clinical practice*. Ed. Rosen P, Baker FJ II, Barkin RN, Braen GR, Dailey RH, Levy RC. St. Louis: C.V. Mosby Company, 1988:143.
50. Better OS. Traumatic rhabdomyolysis ('crush syndrome')—updated 1989. *Isr J Med Sci* 1989;25(2):69-72.
51. Better OS. The crush syndrome revisited (1940-1990). *Nephron* 1990;55(2):97-103.
52. Collins AJ, Burzstein S. Renal failure in disasters. *Crit Care Clin* 1991;7(2):421-35.

53. Odeh M. The role of reperfusion-induced injury in the pathogenesis of the crush syndrome. *Mechanisms of Disease*. Ed. Epstein FH. *N Engl J Med* 1991;324(20):1417-22.
54. Better OS, Stein JH. Early management of shock and prophylaxis of acute renal failure in traumatic rhabdomyolysis. *N Engl J Med* 1990;322(12):825-9.
55. Odeh M. The role of reperfusion-induced injury in the pathogenesis of the crush syndrome [letter]. *N Engl J Med* 1991;325(19):1383-4.
56. Petty TL. A historical perspective of mechanical ventilation. *Crit Care Clin* 1990;6(3):494-8.
57. Bates B. *A guide to physical examination and history taking*. Fourth Edition. Philadelphia: J.B. Lippincott, 1987.
58. Patt A, McCroskey BL, Moore ED. Hypothermia-induced coagulopathies in trauma. *Surg Clin North Am* 1988;68(4):775-85.
59. Aprahamian C, Gessert G, Bandyk DF, Sell L, Stiehl J, Olson DW. MAST-associated compartment syndrome (MACS): A review. *J Trauma* 1989;29(5):549-55.
60. Pattison J, Hirsch N. Morbidity associated with antigravity suits [letter]. *Anaesthesia* 1989;44(10):867.
61. Parra RO, Farber R, Feigl A. Pressure necrosis from intermittent-pneumatic-compression stockings [letter]. *N Engl J Med* 1989;321(23):1615.