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The First
WORLD CONGRESS
on
WILDERNESS MEDICINE

July 14-19, 1991

Whistler, British Columbia, Canada

91-12694

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Seventh Annual Scientific Meeting
of the
Wilderness Medical Society

23 September 1991 Proceedings

Wilderness Medical Society Congress

DAMD17-91-Z-1025

Douglas A. Gentile

Wilderness Medical Society
Vanderbilt University
Nashville, Tennessee 37212

U.S. Army Medical Research & Development Command
Fort Detrick
Frederick, Maryland 21702-5012

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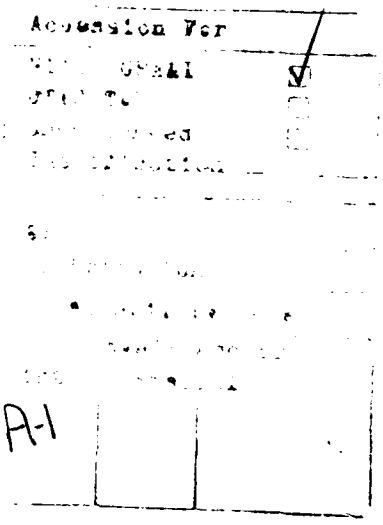
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The First

WORLD CONGRESS
on
WILDERNESS MEDICINE

Medicine and the Spirit of Adventure

July 14-19, 1991
Whistler, British Columbia, Canada





Seventh Annual Scientific Meeting
of the
Wilderness Medical Society
P.O. Box 397
Point Reyes Station, CA 94956
USA
(415) 663-9107



The Wilderness Medical Society was founded in January, 1983 as a non-profit organization which would promote educational and research programs that increase medical knowledge about human activities in wilderness circumstances. In eight years, the Society has grown to 2500 members with representation from around the globe.

The Wilderness Medical Society is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians.

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FIRST WORLD CONGRESS ON WILDERNESS MEDICINE

Medicine and the Spirit of Adventure

Chateau Whistler Resort

July 14-19, 1991

Whistler, British Columbia, Canada

Sunday, July 14 - LOCATION: Frontenac Ballroom

12:30 pm	**Welcome and Introduction** Dr. Gentile and Dr. Erb
12:45	**The World's Environments and the Spirit of Adventure** Mr. Bishop
1:35	**Wilderness Medicine - An Historical Perspective** Dr. Auerbach
2:00	**Rescue Operations in the Swiss Alps** Dr. Durrer
2:50	**BREAK-VISIT EXHIBITS - LOCATION: Empress Ballroom**
3:20	**Wilderness Wound Management** Dr. Zukin
4:10	**Lyme Disease - A Global Perspective** Dr Gentile
5:00	ADJOURN
5:30	**RECEPTION - LOCATION: Empress Ballroom**

Monday, July 15 - LOCATION: Frontenac Ballroom

7:00	**COFFEE SERVICE-VISIT EXHIBITS-LOCATION: Empress Ballroom**
8:00	**Travel Medicine** Dr. Shlim
9:00	**Controversies in Hypothermia: The Afterdrop Phenomenon** Dr.Hayward
9:50	**BREAK-VISIT EXHIBITS**
10:20	**Symposia (simultaneous sessions)**

A. Survival Medicine - LOCATION: Frontenac A
   Desert Survival - Dr. Otten
   Human Cooling Rates in Extreme Cold - Dr Hayward
   Cold Weather Survival - Dr. Bowman
   Cold Water Immersion: Operational Considerations-Dr. Bagian

B. Enhancing Performance for Wilderness Adventure - LOCATION: Frontenac B
Can you enhance capacity for performance at moderate altitudes? - Dr. Levine
Can you enhance capacity for performance in the cold? - Dr. Young
Can you enhance capacity for performance in the heat? - Dr. Pandolf
Assessment of performance capacity for wilderness adventure - Dr. Haskell

C. Wilderness Medicine Core Curriculum I - LOCATION: Frontenac C
Diving Medicine - Drs. Auerbach and Kizer
High Altitude Medicine - Drs. Hackett and Oelz

12:30 pm LUNCH (on your own)

2:00 Workshops/Demonstrations - (first session)

1. World Status of Wilderness Medicine I:
Mountaineering and Environmental Stresses
LOCATION: Frontenac A
Moderators: Drs. Hackett and Oelz

2. Wilderness Medicine Research I
LOCATION: Frontenac B
Drs. Shlim and Taylor

3. Winter Survival
LOCATION: Outdoor location TBA
Dr. Bowman

4. Litters and Litter Packaging
LOCATION: Outdoor location TBA
Mr. Callahan

5. Improvised Self Rescue for the Sport Climber
LOCATION: Outdoor location TBA
Mr. Johnson

6. Wilderness Wound Management
LOCATION: Montebello Room
Drs. Weiss and Zukin

7. Helicopter Rescue in British Columbia
LOCATION: Outdoor location TBA
Mr. Brink and Mr. Flann

3:30 Workshops/Demonstrations - (second session)
LOCATIONS: As indicated above

1. Wilderness Medicine Research II
Drs. Shlim and Taylor

2. Winter Survival
Dr. Bowman

3. **Litters and Litter Packaging**
   Mr. Callahan

4. **Improvised Self Rescue for the Sport Climber**
   Mr. Johnson

5. **Helicopter Rescue in British Columbia**
   Mr Brink and Mr. Flann

5:00 ADJOURN

7:30 **Evening Program: Diving the Rainbow Reefs**
LOCATION: Frontenac Ballroom
Dr. Auerbach

Tuesday, July 16 - LOCATION: Frontenac Ballroom

7:00 **COFFEE SERVICE-VISIT EXHIBITS-LOCATION: Empress Ballroom**

8:00 **Medicinal Plants of Amazonia**
Dr. Plotkin

9:00 **Neurologic Problems at High Altitude**
Dr. Hackett

9:50 **BREAK-VISIT EXHIBITS**

10:20 **Symposia (simultaneous sessions)**

A. **Aerospace Medicine - LOCATION: Frontenac A**
Medical Problems of Space Flight - Dr. Bagian
Designing a Health Care System - Dr. Boyce

B. **High Altitude Medicine - LOCATION: Frontenac B**
Recent Advances in HAPE - Dr. Oelz
Effects of High Altitude on Cardiovascular Diseases - Dr. Hultgren

C. **Wilderness Medicine Core Curriculum II - LOCATION: Frontenac C**
Backcountry Water Disinfection - Dr. Backer
Orthopedic Injuries - Dr. Serra

12:30 pm LUNCH (on your own)

2:00 **Workshops\Demonstrations (first session)**

1. **World Status of Wilderness Medicine II:**
   Aquatic Medicine and Environmental Stresses
   LOCATION: Frontenac A
   Moderators: Drs. Auerbach and Kizer

2. **Desert Survival**
   LOCATION: Outdoor location TBA
   Dr. Otten

3. **High Angle Rescue: Recent Developments**
   LOCATION: Frontenac B
   Mr. Callahan

4. **Basic Backcountry Evacuation and Transportation Skills**
   LOCATION: Outdoor location TBA
   Mr. Johnson

5. **Wilderness Fracture/Dislocation Workshop**
   LOCATION: Outdoor location TBA
   Dr. Serra

3:30 **Workshops/Demonstrations (second session)**
LOCATIONS: As listed above

1. **Desert Survival**
   Dr. Otten

2. **High Angle Rescue: Recent Developments**
   Mr. Callahan

3. **Basic Backcountry Evacuation and Transportation Skills**
   Mr. Johnson

4. **Wilderness Fracture/Dislocation Workshop**
   Dr. Serra

3:30 **Journal of Wilderness Medicine - Editorial Board Meeting**
LOCATION: Montebello Room (open to all WMS members)

5:00 ADJOURN

7:30 **Evening Program: Saving the World's Rainforests**
LOCATION: Frontenac Ballroom
Dr. Plotkin

Wednesday, July 17 - LOCATION: Frontenac Ballroom

7:00 **COFFEE SERVICE-VISIT EXHIBITS-LOCATION: Empress Ballroom**

8:00 **Free Scientific Presentations**
This session will consist of brief communications of current research in all areas of wilderness medicine.

9:50 **BREAK - VISIT EXHIBITS**

10:20 **Free Scientific Presentations (cont.)**

12:30 pm ADJOURN

Thursday, July 18 - LOCATION: Frontenac Ballroom

7:00 COFFEE SERVICE-VISIT EXHIBITS-LOCATION: Empress Ballroom

8:00 Global Health Issues: Impact of International Travel on Developing Countries
Dr. Rasori

9:00 Marine Envenomations
Dr. Williamson

9:50 BREAK - VISIT EXHIBITS

10:20 Symposia (simultaneous sessions)

A. Wilderness Toxinology - LOCATION: Frontenac A
Jellyfish Envenomation - Dr. Williamson
Advances in Crotalid Antivenom - Dr. Russell

B. Expedition Medicine - LOCATION: Frontenac B
Group Cohesion and the Selection of Expedition Members - Dr. Jones
Expedition Medicine: Practical Considerations-Dr. Donner

C. Wilderness Medicine Core Curriculum III - LOCATION: Frontenac C
Traveler's Diarrhea - Dr. Backer
Heat Illness - Dr. Weiss

12:30 pm LUNCH (on your own)

2:00 Workshops/Demonstrations (one session only)

1. World Status of Wilderness Medicine III:
Delivery of Services and Search and Rescue
LOCATION: Frontenac A
Moderators: Drs. Bowman and Durrer

2. Whitewater Safety and Rescue
LOCATION: Outdoor location TBA
Dr. Weiss

3. Avalanche Rescue: Search Dog, Probe and Beacon Techniques
LOCATION: Algonquin Room
Mr. Flann and Blackcomb Alpine Rescue Specialists

4. Field Recognition and Management of Exotic Envenomations
LOCATION: Frontenac B
Mr. Callahan

5. Field Water Disinfection Systems
LOCATION: Frontenac C
Dr. Backer

4:00 ADJOURN

7:30 pm **Banquet and Special Presentation**
LOCATION: Frontenac Ballroom

Preservation and the Spirit of Adventure
Mr. Rowell

Friday, July 19 - LOCATION: Frontenac Ballroom

7:00 **COFFEE SERVICE-VISIT EXHIBITS-LOCATION: Empress Ballroom**

8:00 **Wilderness Medical Liability**
Mr. Miller

9:00 **Dealing with Death: The Psychology of Rescue**
Dr. Jones

9:50 BREAK

10:20 **Arthropod Envenomations: Spiders and Scorpions**
Dr. Russell

11:10 **Bear Attacks: Prevention and Survival**
Dr. French

12:00 noon **Closing Session**
Summary of the World Status of Wilderness Medicine
Drs. Erb and Paton

12:30 pm FINAL ADJOURNMENT

ACKNOWLEDGMENTS

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THE WORLD'S ENVIRONMENTS AND THE SPIRIT OF ADVENTURE

Barry C. Bishop, Ph.D.
Washington, D.C.

In 1888, thirty-three geographers, geologists, explorers, teachers, lawyers, meteorologists, cartographers, military officers, and financiers founded the National Geographic Society. All were learned, well-traveled men distinguished by a love of knowledge and a thirst for discovery and achievement. All possessed a passionate sense of adventure which was manifested in their desire to establish a Society "for the increase and diffusion of geographic knowledge."

In all of these efforts it has pursued its mission by bringing the world's environments to its members beginning with the National Geographic Magazine along with an expanding array of periodicals, books, maps, and other educational materials. It has sought to instill the spirit of adventure in its members, as well as a sound understanding of the vast and changing array of our world's physical and cultural landscapes.

Since 1890 the Society has also supported more than 4,500 research projects and explorations, ranging from Robert E. Peary's North Pole expeditions to a systematic photomapping of the northern skies. Today the Committee for Research and Exploration, with an annual budget of more than $5 million, funds research projects in a broad range of disciplines, including geology, paleontology, geophysics, oceanography, biology, anthropology, ethnology, and geographic exploration.

At the same time, the Society has been a significant force in the conservation movement. Early in its history, it helped save the giant sequoias of California and establish the National Park Service.

Over the course of history, science and technology have rapidly advanced as our globe has shrunk. As a result, complex and escalating problems affecting our quality of life have become increasingly manifest. Hence, our adventures of today and the future are also our concerns that demand attention if we are to maintain and improve our quality of life.

NOTE: *Dr. Bishop preceded his remarks with an audiovisual presentation, World of Beauty, and concluded with another, Voyage of Discovery.*

RESCUE OPERATIONS IN THE SWISS ALPS

Bruno E.Durrer, Air Rescue doctor / Mountain guide,
CH - 3822 Lauterbrunnen, Switzerland

1. HISTORY

In the golden age of alpinism accidents in the high alps most likely ended fatally. At that time a rescue operation usually took several days of hard physical work. The older mountainguides in our villages could tell you tremendous stories of human tragedy and braveness.
The technic of mountaineering has certainly changed over the years and in mountain rescue similar drastic changes have also taken place.
In the early 1950`s many ground rescues were replaced by the utilisation of fixed wing aircraft. For a short period of time rescuers were even dropped by parachute in remote areas. In the mid 1950`s the first helicopters brought a new dimension into mountain rescue. Finally in the 1960`s the new and powerfull turbo- jet helicopters allowed winch rescue missions even at high altitudes and with adverse meteorological conditions.
For rescue operations we use mainly the french helicopters "Alouette III" and "Lama". The "Lama" beeing used for more difficult rescues.

2. RESCUE ORGANISATIONS IN SWITZERLAND

Switzerland is a very small, highly populated, mountaineous country with many helicopter companies. Throughout the country there are 18 helicopter rescue bases which allow to reach every site of accident within 15 minutes of flight after the alarm has been raised.
There are three helicopter rescue organisations: REGA (Swiss Air Rescue) a private, professional, nonprofit rescue company and Air Zermatt / Air Glaciers, two helicopter companies in the southwest (Valais), doing rescues besides their commercial flying work.
REGA runs 24 hours a day an alert-station and has a special radio rescue network. REGA rescue helicopters are airborn within 5 minutes during the day and 20 minutes at nighttime.
95% of all REGA missions are medic assisted. For the resting 5% paramedics are in charge. The medical equipment carried on board is sufficient to cover all traumatological and internal emergencies. The equipment consists of resuscitation kits, oxygen, chestdrains, special stretchers and matress for spinal injuries and mobile electrocardiograph / defibrillator units.

3. SPECIAL EQUIPMENT

Special equipment for difficult crevasse rescues includes a tripod winch and a compressor for digging a tunnel to the victim through the ice.
Avalanche accidents demand special equipment for search and evacuation. The avalanche rescue dog still has priority despite electronical devices.

For the rescue of stranded cable cars special rescue devices have been developped. There is also special equipment for fire fighting availabe.
For ground rescues and avalanche search there is a close collaboration with the Swiss Alpine Club. REGA is responsible for all their medical instruction.

4. SWISS RESCUE MISSIONS 1990

4.1. GROUND RESCUES IN SWITZERLAND

The ratio between ground and air rescues in the Swiss Alps has not changed within the last 10 years and still remains with over 90% beeing carried out by air. 5% are combined ground/air rescues and 5% are pure ground rescues. Quite often the helicopter rescue doctor is part of the ground rescue team.

4.2. AIR RESCUES

Every year we have at least 3000 helicopter rescue missions in the Swiss mountains, 1200 rescues for road accidents and 3000 hospital transfer flights.
Swiss Air Rescue (REGA) is responsible for about 2/3 of all helicopter rescue flights. The other 1/3 is carried out by the two companies Air Glaciers and Air Zermatt.

For 1990 exact data is only available from REGA. All persons rescued are registered according to medical and topographical index. In 1990 88% of all patients were rated NACA III- VII and 57% were rated NACA IV-VII, where medical (anesthesiological) assistance is defined to be compulsory. 2/3 of all rescue missions were topographically rated D - G.

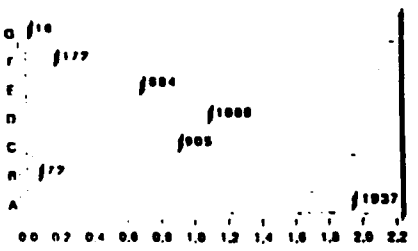
TOPOGRAPHICAL INDEX

SITE OF EMERGENCY

A Hospital
B Doctor's practice
C Road
D Field-path, ski slopes
E Easy accessible
F Difficult accessible
G Extremely difficult accessible

REGA HELICOPTER RESCUES 1990

TOPOGRAPHICAL INDEX N:4872 PAT.



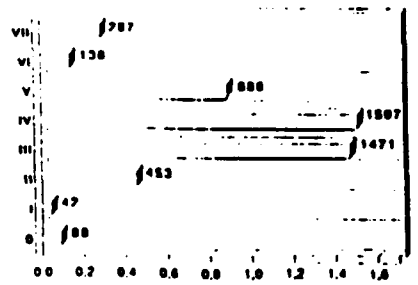
MEDICAL INDEX

MODIF. NACA-INDEX

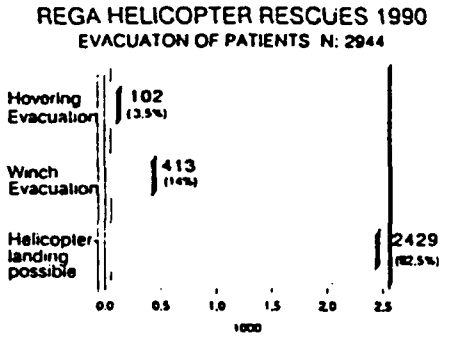
0 Evaluation, not injured
1 No acute therapy (Doctor) necessary
2 Ambulant therapy necessary
3 Stationary hospitalisation necessary
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5 Acute vital danger
6 Restitution of vital functions
7 Death (with or without resuscitation)

REGA HELICOPTER RESCUES 1990

NACA INDEX N:4872

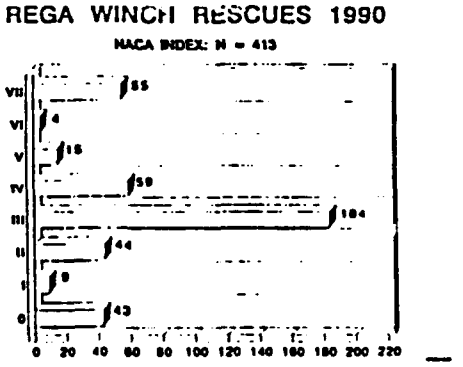


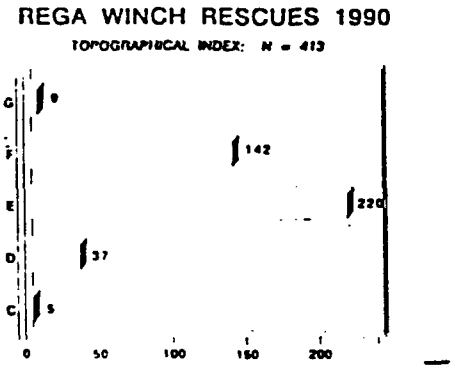
In over 80% of all primary missions the helicopter was able to land at the site of accident. Nearly 15% were winch evacuations and in max. 5% the helicopter hovered while the patient was lifted aboard. According to our experience the hovering percedure is a rather dangerous mission and we try to keep this rate as low as possible.



4.2.1. WINCH RESCUES IN SWITZERLAND

In 1990 about 600-700 (1983/84:n=450/Y) persons were rescued by helicopter winch in Switzerland (REGA n = 413).



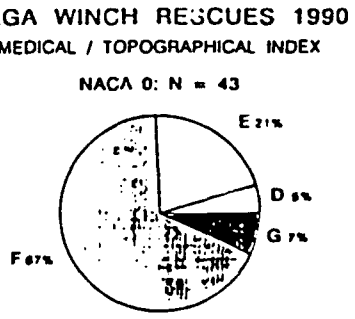


Over 75% of all REGA winch missions were rated NACA III-VII. Over 90% of all REGA winch rescues were rated topographically E - G. Over 2/3 of all patients rescued by winch were evacuated from accessible and 1/3 from difficult aaccident's site. 2% were extremely difficult winch rescues e.g. direct face rescues from the Eiger - Northface. Today almost every spot in the N-Face can be reached by extension of the winch cable.

4.2.2. PRACTICAL ASPECTS OF HELICOPTER WINCH RESCUE

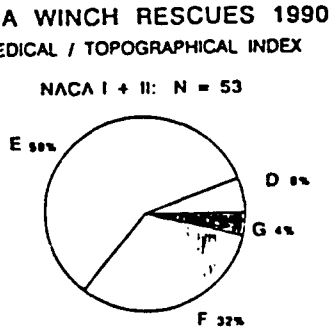
The practical consequences for the treatment and evacuation in winch rescues depend upon the rescue risks and the typ of injury. The rescue risks are determined by the meteorological, topographical and objective dangers of the mountains.
For a detailled discussion we have to look at the winch rescues in relation to the medical and topographical index:

NACA 0:



Almost 75% of all NACA 0 were rescued from difficult accessible area (TOPO F+G).
For the rescue of hikers stranded in difficult accessible areas we use special rescue belts or rescue jackets.
In extremely difficult areas the climbers are evacuated by winch on the climbing harness without problems. In some cases the blocked climber could hook himself to the winchcable, without lowering a rescuer first. This reduces the rescue risks considerably.
In the last two years paraglider accidents in the Alps have increased tremendously. Very often they are not injured but are found in very difficult situations.

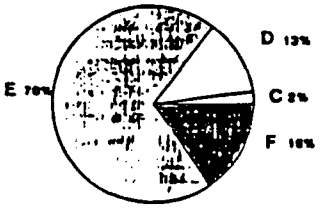
NACA I + II:



The winch rescue of NACA I+II patients usually causes no problems. These ambulant cases can be evacuated directly by the climbing harness or rescue belt and the rescuer has not to leave the winch cable. This percedure lowers the risk considerably whenever safe belays are lacking or objective dangers threaten the mission.
Patients with dislocated shoulder (NACA II) receive sufficient painkillers prior to the winch evacuation. Exceptionally we even relocate the shoulder immediately and winch the patient afterwards.

NACA III + IV:

REGA WINCH RESCUES 1990
MEDICAL / TOPOGRAPHICAL INDEX
NACA III + IV: N = 243



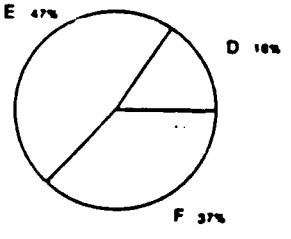
REGA WINCH RESCUES
TOPOGRAPHICAL INDEX / DIAGNOSIS

	HEAD/BRAIN	TX/SPINE	ABD/PELVIS
D	7	11	3
E	61	67	19
F	34	35	12
G	3	5	1

Over 50% of all winch rescued patients suffer of an injury rated NACA III + IV. A closer look at the the diagnosis of winch rescued persons shows a big number of injuries of head and trunc. Thus a proper treatment and fixation prior to the winch evacuation isessential.
In difficult accessible areas the rescue doctor often has the dilemma as to whether an immediate recovery or to administer immediate treatment at the site of accident has priority, especially when stone- or icefall threatens a mission.
On the other side severe headbrain- or spinal injuries demand immediate medical treatment. If the rescue risks allow it, the patients receive full first medical treatment at the site of accident. They are fixated and evacuated either in the horizontal-net or - bag with cervical collar and vacuum matress, if necessary.
In narrow crevasses the fixation device "KED" is very usefull. In difficult site of accident (Topo > F) additional helpers may be necesary for the fixation and the evacuation in the net or bag.
Prior to the evacuation the patient receives intravenous sedative medication.
During the winch evacuation the medic in charge accompanies the patient. According to our experience this reduces the psychological rescue stress of the patient considerably.

NACA V + VI:

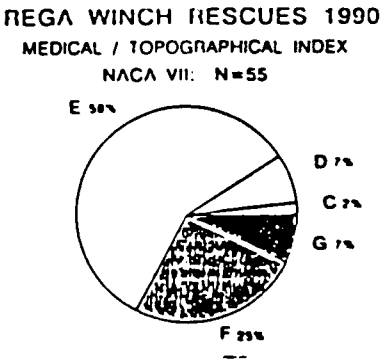
REGA WINCH RESCUES 1990
MEDICAL / TOPOGRAPHICAL INDEX
NACA V +VI: N = 19



An adequate first medical treatment for severely injured patients requires sufficient space to work and often additional helpers. In difficult accesible sites these patients generally are evacuated first and properly treated later.

However in desperate cases (1,5% of all winch-rescued patients) it can become necessary to intubate a patient immediately and evacuate him in the net.
In difficult areas such rescues are very delicate and demand usually additional helpers. This also applies for resuscitacions in pathless areas.

NACA VII:



Very often the helicopter can not approach close enough to the site of accident to determine whether a patient is still alive or not and the doctor has to descend.
As soon as the diagnosis of death is made, the recovery usually causes no problems. The victims arehooked to the winch and evacuated. Due to falling stones or ice some death-body recoveries take place only early in the morning.

5. RESCUE RISKS / DIFFICULT WINCH RESCUES

For difficult missions (low visibility, strong winds, night missions, direct faces, extension of the winch cable) the rescue risks have to be evaluated in relation to the seriousness of injury.
Due to the risk of rock- or icefall some rescues can be carried out only during the early hours of the day. However - often it can not be determined from the helicopter whether a patient is still alive or not.
Within the last 10 years 3 ambulance helicopters have been lost during rescue missions. There is considerable danger in the mountains from power cables and transport cables, especially with low visibility.

Occasionally for some difficult direct face rescues we operate "Lama"-helicopters, especially equipped with a convex "bubble-door". Thus the pilot has direct visual contact to the rescuer underneath. This know-how is derived from the helicopter pilot's experience in "helilogging" and allows long line rescues with an extension of up to 70 meters with an extraordinary precision.

For the winch rescue of para- or deltagliders an extension of the winchcable of up to 70 meters is also necessary to avoid further fall due to the downwash of the helicopter. Such a rescue can be very delicate and demands an experienced crew.

Winch rescues at night (1990 REGA: 58 rescued persons) demand excellent meteorological conditions and a highly experienced crew. They take place only if exact information about the site of accident is available. REGA helicopters are equipped with special night vision devices and search lights.

6. FUTURE OF HELICOPTER RESCUE IN SWITZERLAND

For the past ten years 80% of all REGA flights were medic-assisted and only 20% were acompanied by paramedics. For legal reasons today there is the tendency towards 100% medic-assistance.

The single engined Alouette III has been in service for over 25 years now. For safety reasons a modern,twin engined helicopter is needed as replacement. REGA evaluated for this reason three different types of helicopters in the high alps. Finally the Italian Agusta AK 107 was chosen. This should result in safer and more efficient rescue operations in the future.

7. CONCLUSIONS:

1. Due to the fact that many climbers and paragliders now carry walky-talkies, the time lapse between accident and alert has been considerably reduced. Consequently the medics are confronted more often with severly injured patients who would not otherwise have survived.

2. Over 75% of all winch rescued persons were rated NACA III - VII. For this reason we consider it as necessary to have a medic on board.

3. This medic has to be physically fit and must be trained in alpine technics as well, since 2/3 of all 1990 rescue missions (n:2944) were topographically rated D - G.

4. If the rescue risks allow it and the typ of injury (NACA > III) demands it, we start to treat injured persons even in difficult accident`s sites.
   The assignment of trained air rescue doctors improved the efficiency of first treatment at the site of accident even in difficult and extremely inaccessible mountain areas.

TOPOGRAPHICAL INDEX

SITE OF EMERGENCY

A Hospital

B Doctor's practice

C Road

D Field-path, ski slopes

E Easy accessible
(Alpine huts, climbing < UIAA 3)

F Difficult accessible
(Climbing UIAA 3+4, crevasse)

G Extremely difficult accessible
(Climbing > UIAA 5, north faces, narrow crevasse)

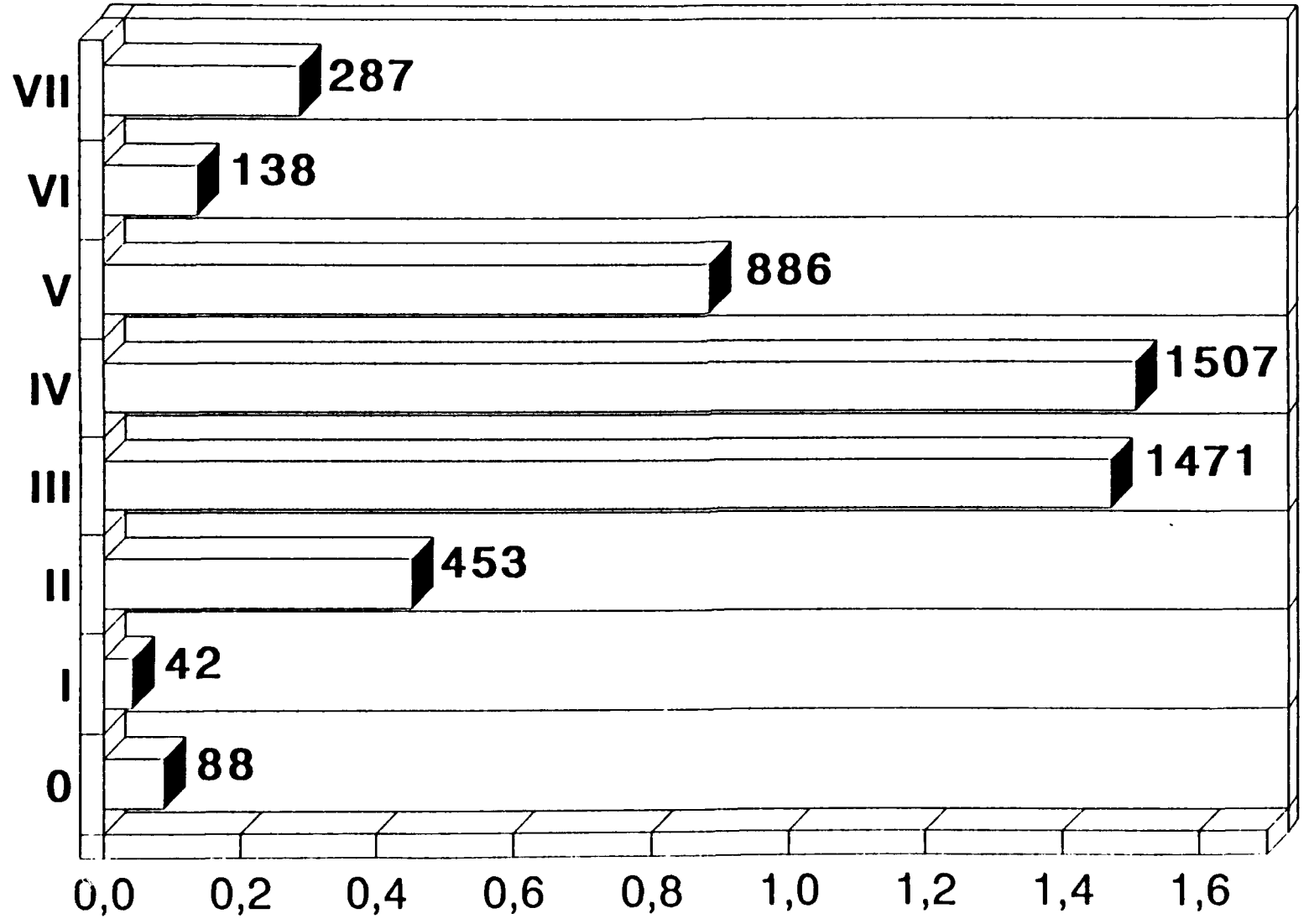
MEDICAL INDEX

MODIF. NACA-INDEX

0 Evacuation, not injured
1 No acute therapy (Doctor) necessary
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3 Stationary hospitalisation necessary
4 Vital danger possible
5 Acute vital danger
6 Restitution of vital functions
7 Death (with or without resuscitation)

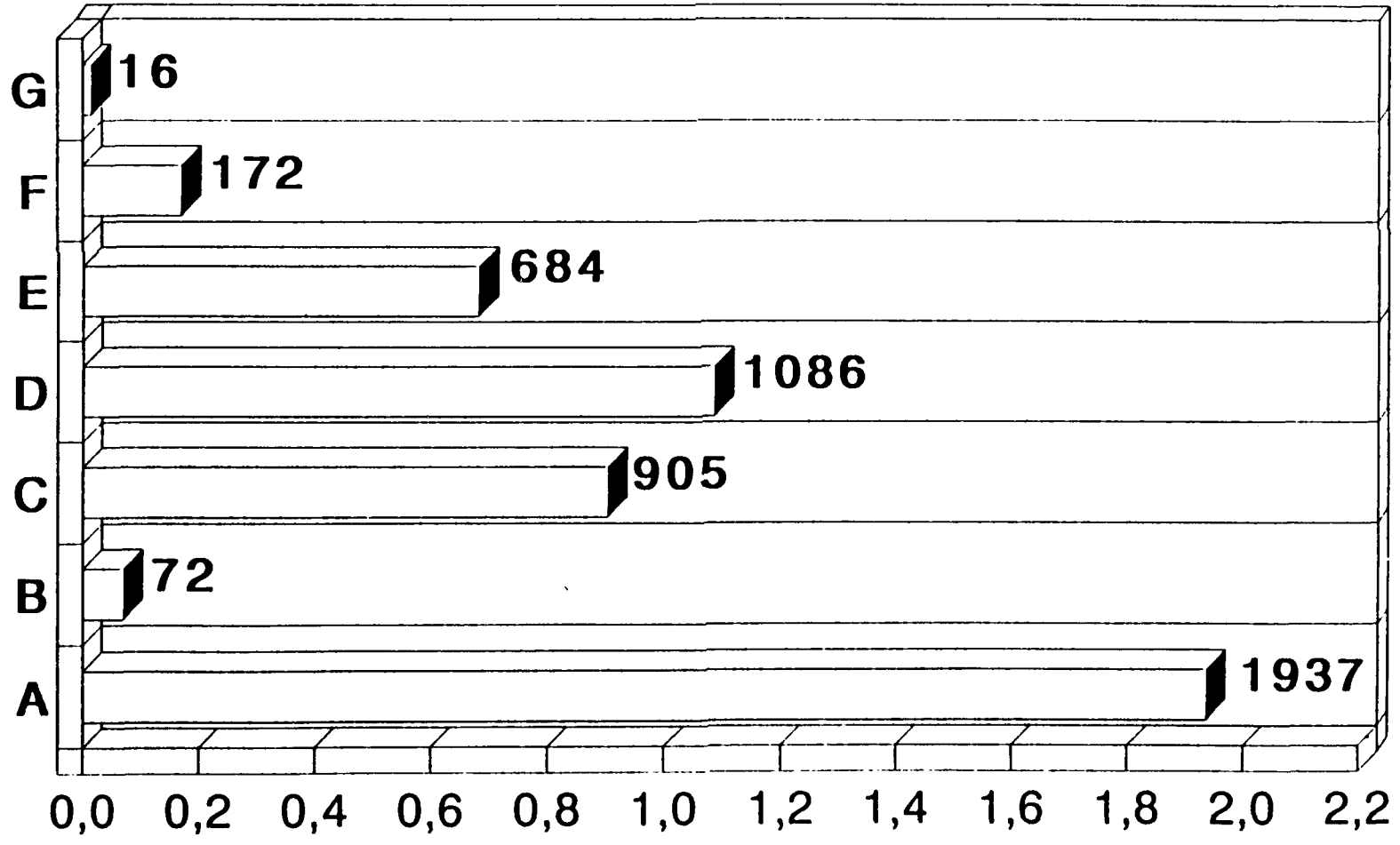
REGA HELICOPTER RESCUES 1990

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REGA HELICOPTER RESCUES 1990

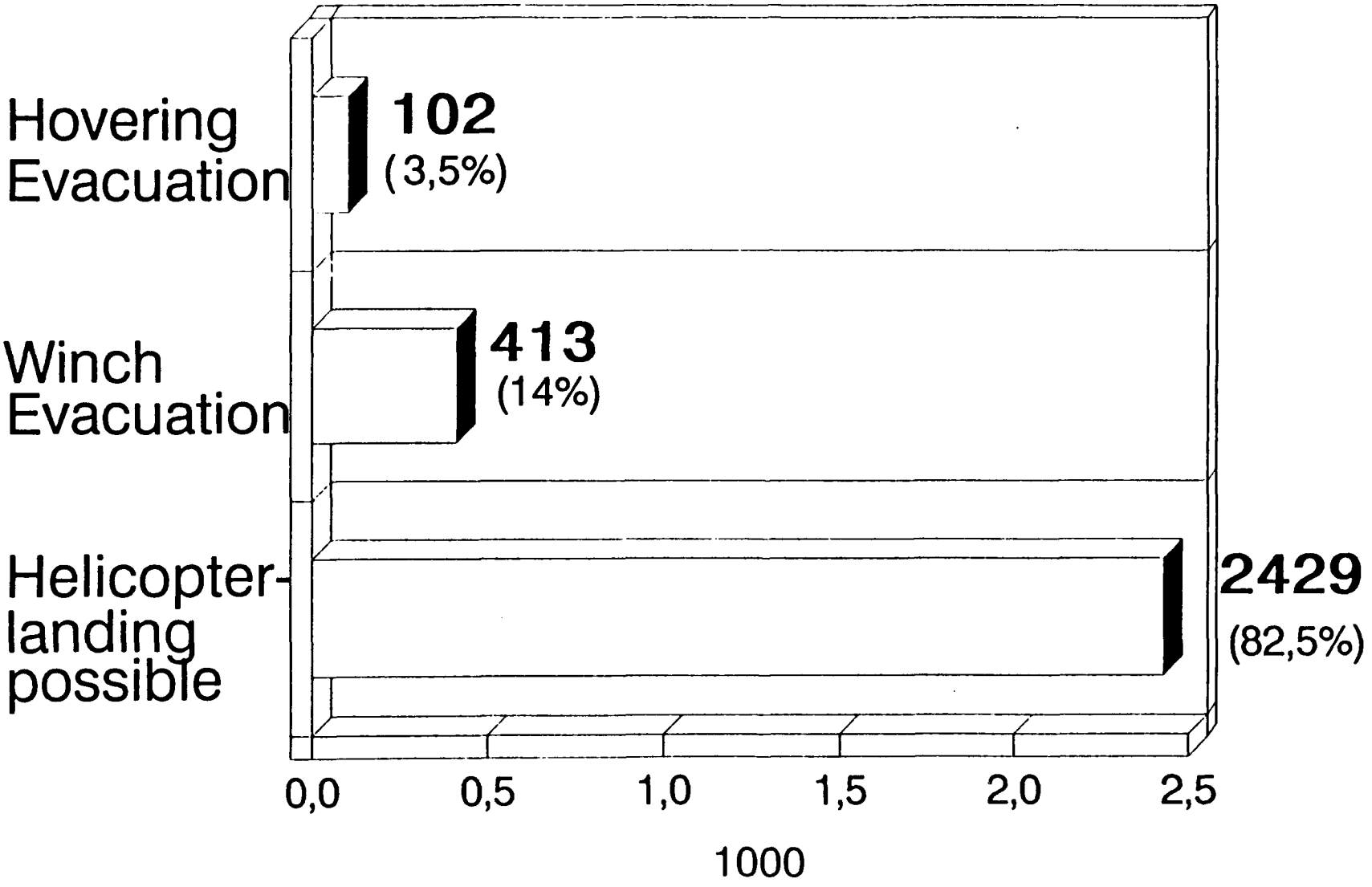
TOPOGRAPHICAL INDEX N:4872 PAT.



TOPOGRAF

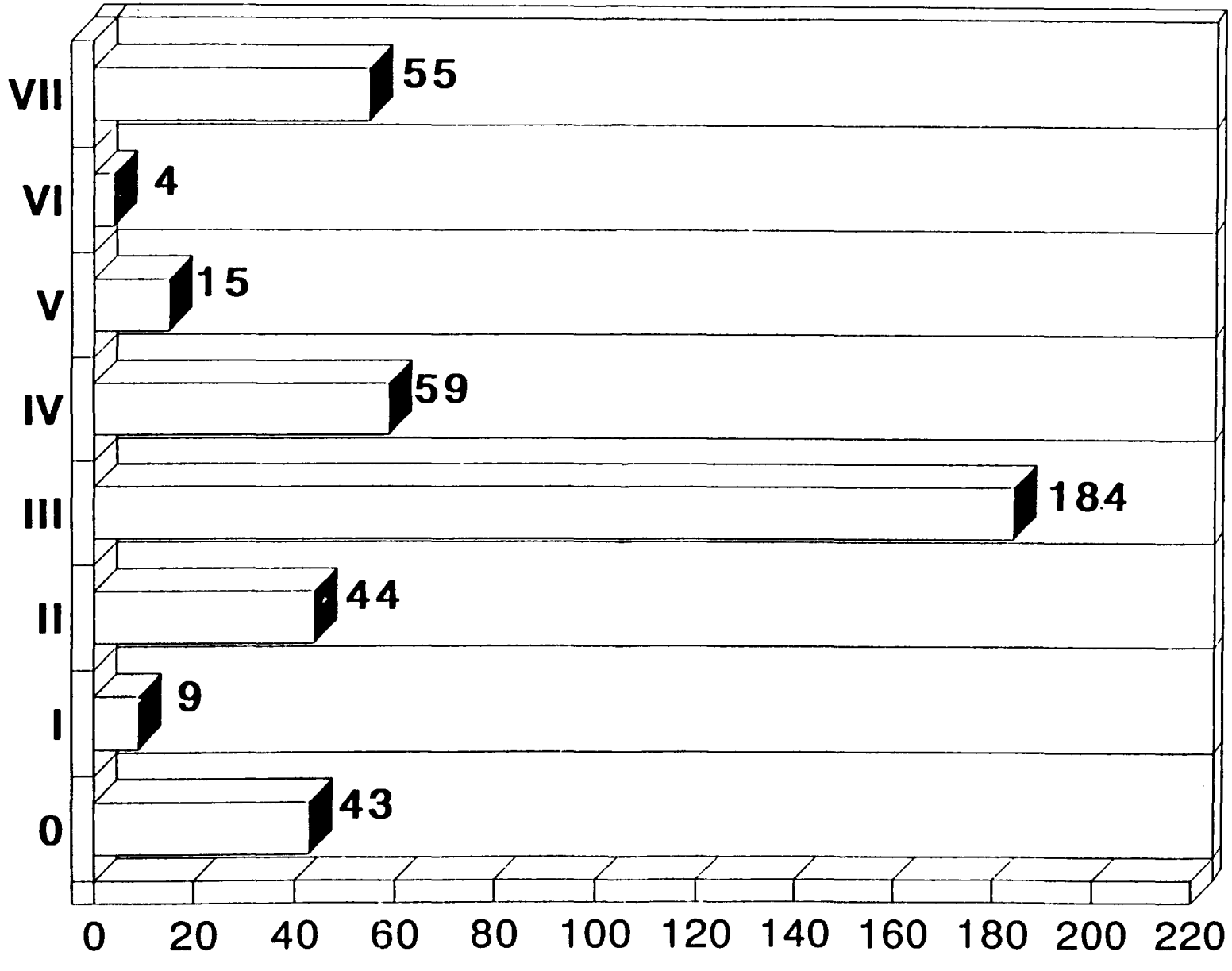
REGA HELICOPTER RESCUES 1990

EVACUATON OF PATIENTS N: 2944



REGA WINCH RESCUES 1990

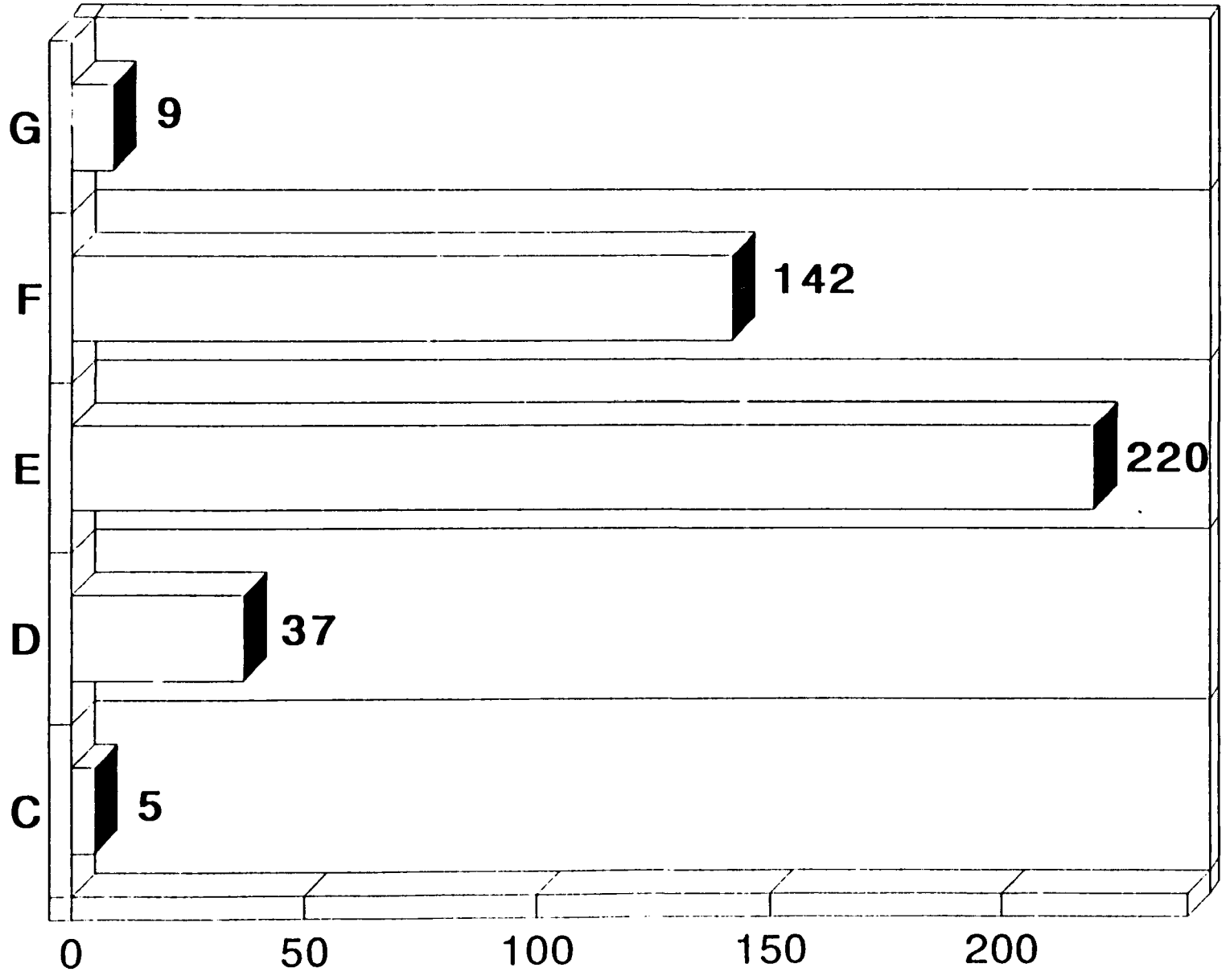
NACA INDEX: N = 413



WINDENGR

REGA WINCH RESCUES 1990

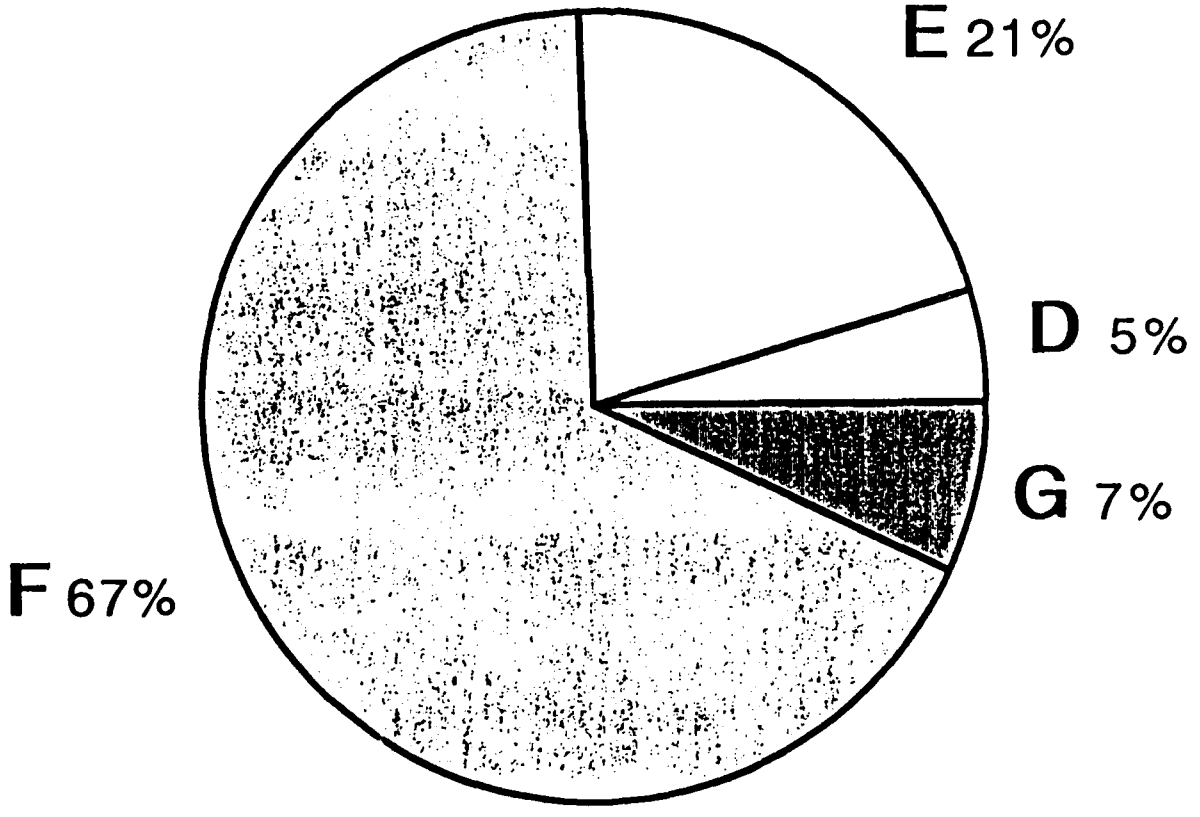
TOPOGRAPHICAL INDEX: N = 413



REGA WINCH RESCUES 1990

MEDICAL / TOPOGRAPHICAL INDEX

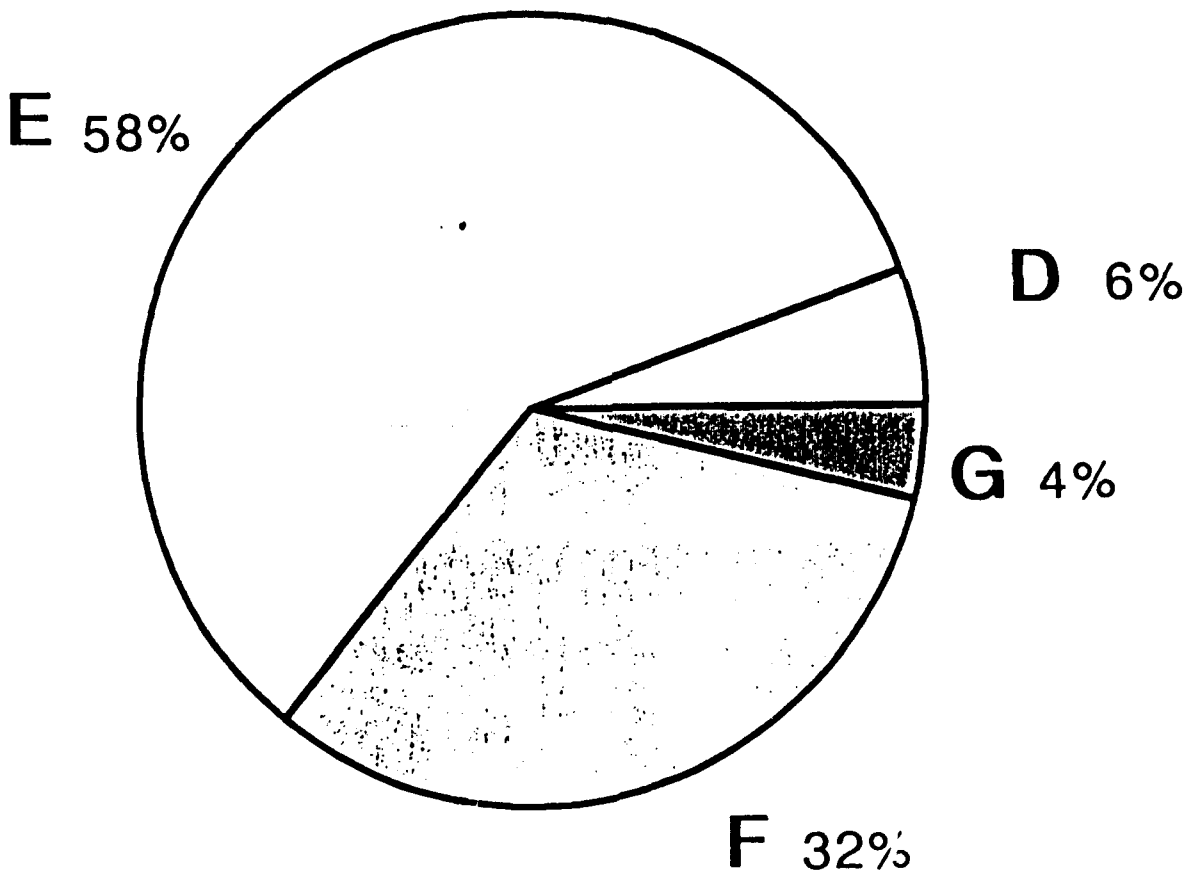
NACA 0: N = 43



REGA WINCH RESCUES 1990

MEDICAL / TOPOGRAPHICAL INDEX

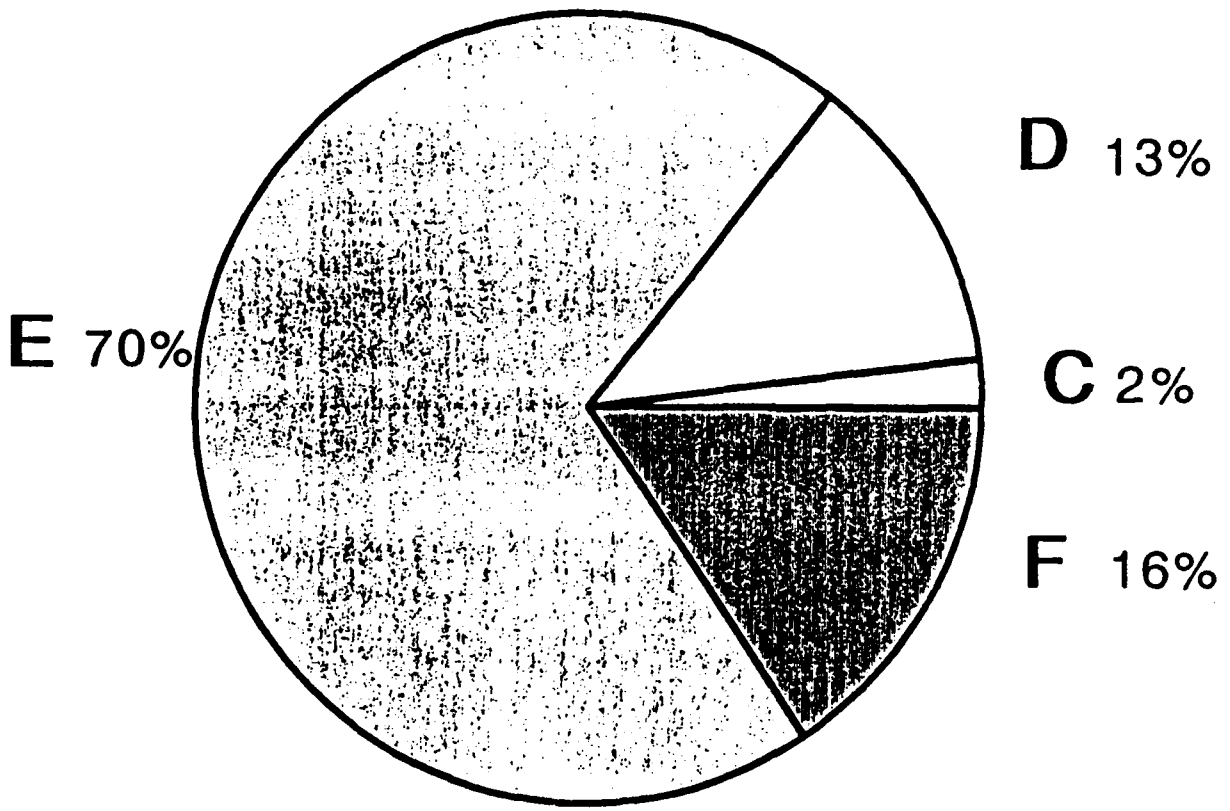
NACA I + II: N = 53



REGA WINCH RESCUES 1990

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NACA III + IV: N = 243



WINCH34

REGA WINCH RESCUES

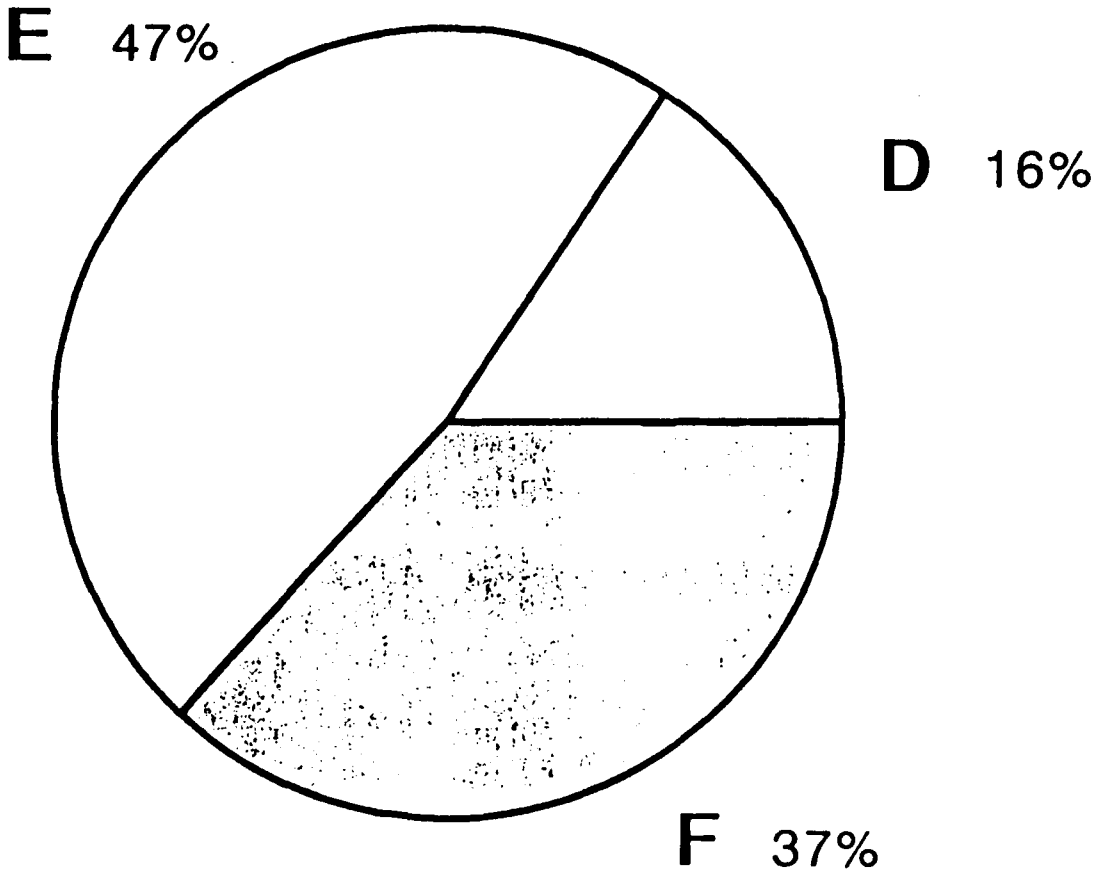
TOPOGRAPHICAL INDEX / DIAGNOSIS

	HEAD/BRAIN	TX/SPINE	ABD/PELVIS
D	7	11	3
E	61	57	19
F	34	35	12
G	3	5	1

REGA WINCH RESCUES 1990

MEDICAL / TOPOGRAPHICAL INDEX

NACA V +VI: N = 19

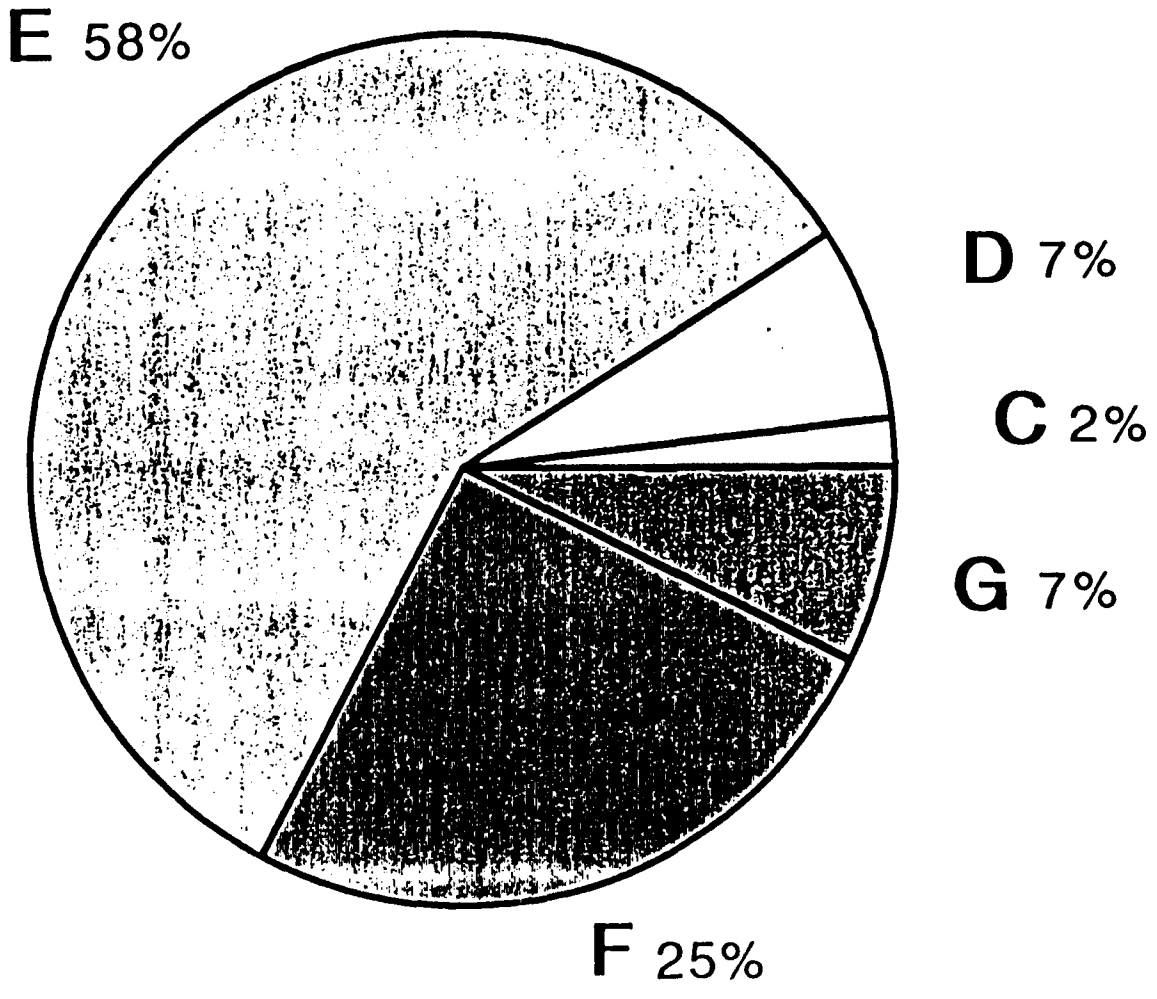


WINCH56

REGA WINCH RESCUES 1990

MEDICAL / TOPOGRAPHICAL INDEX

NACA VII: N=55



WOUND CARE MANUAL

WILDERNESS MEDICAL SOCIETY

SUMMER, 1991

D. D. Zukin, MD

CONTENTS

I. Normal Repair of Lacerations
II. Types of Wounds
III. Factors Affecting Wound Infection Rate
IV. Materials
V. General Evaluation of the Patient
VI. Wound Pre-Care
VII. Wound Closure
VIII. Aftercare
Illustrations

Introduction

The basic principles of wound cleansing and closure remain the same, even out in the wilds. The main problems faced in the wilderness are related to limited supplies. With a minimum of equipment, however, one can close the majority of simple lacerations. This manual sets forth the basic principles of wound closure. The reader will have to modify these guidelines based upon the resources which he or she has at hand.

I. NORMAL REPAIR OF LACERATIONS

A. RE-EPITHELIALIZATION.

The epidermis, the epithelium of the skin, protects the underlying dermis from both desiccation, and invasion by bacteria. When the skin is injured new epidermal cells migrate out to close over gaps. When a lacerations is repaired so that the wound edges evert, the basal cells of each wound edge come into direct contact and a new layer is formed in as little as 12-18 hours; but with inverted edges the new layer will take approximately 72 hours to form (SEE ILLUSTRATION).

Re-epithelialization occurs deep to any necrotic tissue, and hence debrides away non-viable

tissue.

B. NEOVASCULARIZATION.

Neovascularization begins at day 2-3, and peaks at day 4. Capillary buds grow out from each wound edge, and eventually join in the middle. The proliferation of blood vessels gives the healing wound an erythematous hue, but with minimal pain or induration.

Be careful not to mistake neovascularization for early infection.

C. CONNECTIVE TISSUE REGENERATION.

Fibroblasts and macrophages, like capillary buds, begin to appear in about 2-3 days. New collagen is laid down, and increasingly stronger strands bridge the wound gap. The collagen fibers are continually remodeled as the wound matures. In one study a preparation of normal skin could withstand a load of 18 pounds. A similar sample, 10 days after repair could withstand a load of only 1 pound; at 20 days 2 pounds; at 40 days 6 pounds; and at 5 months 14 pounds. The final strength of the wound, once healing is complete, is only about 80% that of normal tissue. Similarly, extensibility in a healed wound is not as great as in intact skin.

II. TYPES OF WOUNDS

A. LACERATIONS

1. Shear (sharp cuts caused by metal or glass).

Little tissue damage immediately adjacent to the laceration. Has the highest resistance to wound infection. Minimal edema during the healing process. Minimal foreign material and ground in dirt.

2. Tension wounds (i.e. a blow to a skin without a bone immediately below the skin).

Irregular edges, increased damage of the tissue directly adjacent to the laceration itself. Higher infection rate and scarring potential than shear wounds.

3. Compression wounds (a bone beneath the skin, struck by blunt object, i.e. stellate wound that occurs when the forehead strikes the ground).

Increased edema, infection potential and scarring than shear wounds.

4. Flaps.

Distal flaps (i.e. attached distally, detached proximally) have a more tenuous blood supply and hence have a higher potential for necrosis than do proximal flaps (i.e. flaps attached proximally). However, flaps in children, especially of the distal fingertips, have an amazing capacity of recovery.

B. PUNCTURE WOUNDS.

Puncture wounds may contain foreign material . For example, bits of fabric and plastic may become imbedded in the foot when a patient steps on a nail while wearing running shoes. Puncture wounds of the foot deep enough to reach the bone can result in osteomyelitis, frequently with pseudomonas as the causative organism.

C. ABRASIONS.

Loss of some or all of the epidermis. Repair is by migration of new epidermal cells from adjacent intact skin edges as well as from epidermal appendages (i.e. sweat and sebaceous glands) and hair follicles.

D. FOREIGN BODIES.

Suspect foreign body in wounds which become infected. Foreign bodies are difficult to detect prior to closure without the use of xray.

III. FACTORS AFFECTING WOUND INFECTION RATE

Several factors affect the wound infection rate. Highly vascular regions, such as the face and scalp, are more resistant to infection. Shear lacerations have a lower infection rate than compression injuries. Wounds repaired with 8-10 hours of the time of injury have a lower infection rate than wounds sutured later than 8-10 hours. However facial lacerations frequently heal without infection even when sutured closed more than 24 hours after the injury.

Suturing technique also influences the infection rate. Wounds with tight sutures and wounds with inverted edges are more prone to infection. Wounds that are grossly soiled, such as those resulting from animal or human bites have higher infection rates than clean wounds.

IV. MATERIALS

A. SUTURE THICKNESS-- the more O's, the finer the suture:

Largest 0, 00, 000, 4-0, 5-0, 6-0 Smallest.

B. SUTURE MATERIALS.

1. Absorbable.

a. Vicryl and Dexon (polyglycolic acid).

Lowest infection rates of absorbables, because breakdown products inhibit bacterial growth. Lowest tissue reactivity of absorbables. Good tensile strength. Braided, so hold knots well, but makes gradual cinching down of ties more difficult. Can take 40 or more days to absorb, but usually lose tensile strength within 14 days. Primarily used for deep and subcuticular closures.

Come both dyed and undyed. For emergency department use, **choose undyed**, especially when used near the skin surface.

b. Chromic.

A gut suture which has been treated with chromic ion to increase its strength. High tissue reactivity and slightly higher infection rate. Lower tensile strength than vicryl or dexon. Stiffens when dry, hence packed in liquid to make it easier to handle. Non-braided. Takes 14-17 days to absorb.

c. Plain gut.

Most tissue reactivity of the absorbables. Same infection potential as chromic. Low tensile strength, so breaks easily if knot cinched tightly. Absorbs in 4-10 days, so useful where rapid absorption required, such as inside the mouth.

d. Fast -Absorbing Gut.

Heat-treated plain gut loses its tensile strength even faster than plain gut, and is thus useful for placing sutures in areas where suture removal will be difficult.

e. PDS (Polydioxanone) and **Maxon** (GTMC)

PDS and Maxon, like Vicryl and Dexon, are degraded primarily by hydrolytic action, and possess a similar low tissue reactivity. Both are monofilaments. Monofilament materials possess a smoother passage through tissue, and, in studies, a lower incidence of infection rate.

PDS and Maxon are ideal for back-packing trips and other expeditions because, although they are absorbable, they retain tensile strength for more than two weeks and thus are also suitable for simple skin closure. Therefore one can take along just one suture material, and still be able to close both the deep and the skin layer.

2. Non-absorbable.

a. Silk. Standard for all sutures in terms of ease of tying. Braided, so holds knot well. Unfortunately, the higher infection potential of silk sutures in comparison to monofilaments limits its usefulness.

b. Nylon (Ethilon & Dermalon) Monofilament. Low tissue reactivity, low infection rate. Harder to use than silk. Knots tend to unravel, hence use 4- 5 "throws" per knot.

c. Polypropylene (= Prolene). Monofilament. Similar features to nylon. Slightly easier to handle than nylon. Blue prolene is especially useful for repairing scalp lacerations in dark haired individuals, because the sutures are easy to locate for removal.

d. Dacron. Braided, easy to use, low tissue reactivity. Comes in a coated form which pulls through tissue more easily than the uncoated type.

C. NEEDLES. Look on the package.

1. **Curves.** Straight, 3/8th circle (for most uses), 1/2 circle (for web spaces of fingers and toes.

2. **Needle cross-sections.**

a. Cutting. Triangle with flat base on the outer circumference and cutting point on the inner circumference.

b. Reverse Cutting. Triangle with sharp cutting edge on the outer perimeter, and flat surface on the inner perimeter, hence less cutting of skin in region where suture tension is the greatest. In many cases reverse cutting needles are labeled simply as cutting needles. Choose this needle configuration for most ER suturing situations.

c. Taper. Circular. For special situations, such as tendon repairs.

3. **Needle classes.**

Note, the picture on the box reflects the actual size and curvature of the needle. The letters and numbers stand for different things depending on the manufacturer. For example, M is a series number used with ethicon. FS = for skin,honed 12 times. P = plastic, honed 24 times. PS = plastic surgeon's, intermediate honing between FS and P. Larger numbers generally refer to smaller needle sizes. For facial lacerations, use a P3 needle.

Recommendations: For expeditions, choose cutting needles of various sizes, small for the face and the fingers (such as the P-3 needle by Ethicon), and larger needles for the arms, legs and trunk (such as the FS-2 by Ethicon).

D. INSTRUMENTS

1. **Skin hooks.**

Decrease local tissue damage but do not offer the control of forceps.

2. **Forceps.**

Smooth and rat-toothed. Toothed forceps cause less crushing of skin because less pressure is required to secure the skin edge.

3. **Needle holders.**

Hold the needle at the junction of the middle and proximal 1/3rds, near to where the suture attaches to the needle. Smooth and corrugated surfaces. Smooth best for 5-0 and 6-0 suture material. Hold the clamp with the ring finger in a loop and the index finger along the shaft (= **"palm" the needle holder**).

4. **Scissors.**

Iris scissors are quite adequate for debriding wound edges.

5. **Scalpels**

Scalpels are primarily used for incising specific patterns for plastic wound closures that are

best not attempted on expedition.

V. GENERAL EVALUATION OF THE PATIENT

Remember not to be distracted by the open wounds of a multiply-injured patient. The course of resuscitation still should be:

1. **A-C (Airway + Cervical spine immobilization)**
2. **B (Breathing)**
3. **C (Circulation)**

Once the patient is stable, then proceed to care for the open wounds. Check the status of the circulation, motor function and sensation (CMS) of the injured area. Be sure to check the sensory-motor examination before administering the local anesthetic.

Tetanus prophylaxis: Follow the guidelines at the end of the handout. **In an un-immunized patient, both tetanus immune globulin (250 IU given IM) and tetanus toxoid (0.5 cc IM) must be given for tetanus prone wounds such as punctures and deep lacerations.**

VI. WOUND PRE-CARE

A. SHAVING.

Leave your razor blades at home. There is a higher incidence of infection in operative wounds prepped with shaving as compared to clippers. Therefore clipping away bothersome hairs with a scissors is the preferred method of hair removal.

NEVER SHAVE THE EYEBROWS.

Eyebrows are slow to grow back, and in addition they serve as valuable landmarks during suturing.

B. LOCAL ANESTHESIA.

Whenever possible, local anesthesia should precede irrigation, for the sake of the patient's comfort.

1. Local infiltration.

There is no greater infection rate injecting through the open wound edge as opposed to through intact skin, and it is much less painful to the patient. Raise a weal in the dermis.

1% lidocaine is adequate for most purposes.

1cc of 1% lidocaine contains 10 mg of the drug. **Do not exceed 4 mg/kg of lidocaine or 0.4 cc/kg of the 1% solution (with a maximum dose of 300 mg). Do not exceed** 7 mg/kg of lidocaine with epinephrine or 0.7 cc/kg of the 1% solution (with a maximum dose of 500 mg). The addition of epinephrine both prolongs the duration of anesthesia, and slows the oozing of blood in vascular areas such as the face and the scalp.

EPINEPHRINE SHOULD NOT BE USED ON THE FINGERS, TOES, TIP OF THE NOSE OR THE PENIS, OR IN WOUNDS WITH TENUOUS PERFUSION (SUCH AS FLAPS) BECAUSE IRREVERSIBLE ISCHEMIA CAN RESULT.

If you are going to bring one anesthetic solution, bring 1% plain (without epinephrine) lidocaine.

Bupivacaine (= Marcaine) 0.25% or 0.5% lasts for several hours, and hence is a useful agent for prolonged procedures. The dose is 1-2 mg/kg, or about 1/2 cc/kg of the 0.25% solution (contains 2.5 mg/cc). Use with caution. Do not give more than the recommended amount. **Always aspirate back on the syringe before injecting, because inadvertent intravascular injection has resulted in irreversible cardiovascular collapse.**

2. **Regional block** (i.e. the digital nerve block for finger lacerations, or the mental nerve block for lip lacerations) Ideal because there is no edema and secondary deformity of the area to be sutured.

C. IRRIGATION.

Forceful (one the order of 7 psi of pressure) irrigation of wounds effectively washes away both bacteria and foreign material.

THE METHOD OF CHOICE FOR IRRIGATING OUT SOILED LACERATIONS IS TO FIT A 20 CC OR 30 CC SYRINGE WITH A 16 GAUGE OR 18 GAUGE ANGIOCATH AND PRESS DOWN FIRMLY ON THE PLUNGER.

Irrigation from an iv bag or bulb syringe irrigation simply will not generate the pressure necessary to remove foreign material and bacteria. Syringes and catheters are lightweight, and can easily be brought along. Saline can be made by adding 1/4th teaspoon salt per cup of boiling water. (Let the water cool before attempting irrigation.)

Clean wounds, such as those caused by sharp metal or glass, are generally NOT highly contaminated with foreign material and do not require high pressure irrigation.

Use caution to ensure that none of the irrigation fluid splashes into your own eyes, placing you at risk for catching hepatitis virus (or worse).

D. TOPICAL ANTISEPTICS

1. Betadine Prep Solution (= 10% Povidone-iodine in an aqueous base)

1% povidone-iodine solution (= a 1:10 dilution of the stock 10% Betadine prep solution) is the topical antiseptic of choice for use in the emergency department. Dilute povidone-iodine solution applied for 30-60 seconds into the open wound significantly lowers the incidence of wound infection without perceptibly altering the course of wound healing.

So, along with your syringes and catheters, bring a small plastic bottle of betadine solution to leak onto your carefully packed clothes.

Unlike Normal Saline, the Betadine Prep solution is dabbed on the wound, not irrigated under pressure.

The same solution is used to paint the surrounding intact skin just prior to draping.

NOTE: The reason for the hesitancy on the part of many to employ povidone-iodine solution to the open wounds is that **povidone-iodine detergent (= Betadine Surgical Scrub, found in both bottles and the ubiquitous scrub brushes with the attached sponges) is terribly cytotoxic.** The fact is that all detergents are highly cytotoxic, so as a general rule:

IF IT LATHERS, DON'T USE IT IN THE SAME ROOM AS AN OPEN WOUND (or at least in the same tent)!

As with most rules, there are exceptions. Green soap is recommended for the decontamination of bite wounds from potentially rabid animals such as skunks, raccoons, foxes and cows (the cow, by the way, is now the most commonly rabid domestic animal in the state of California).

Povidone-iodine, with repeated use can be absorbed systemically causing mild alterations in thyroid function tests (most notable an elevated TSH with a normal T-4) Hence the solution should be used with caution in pregnant women, and in infants.

2. Betadine Surgical Scrub (= 10% povidone-iodine in a detergent base).

As mentioned above, Betadine Scrub is cytotoxic, and hence should only be used to cleanse intact skin (i.e. washing your own hands.)

3. Phisohex.

Like betadine scrub, phisohex contains a detergent and consequently should not be

employed near an open wound.

4. Alcohols.

Alcohols are highly cytotoxic, essentially fixing the tissues. Like detergents, alcohols probably shouldn't be used in the same room as an open wound.

5. Hydrogen peroxide.

All physicians have watched with pleasure the way hydrogen peroxide bubbles away blood stains from their scrub suits and white jackets. Unfortunately, peroxide causes the same effervescence in capillaries, leading to a complete standstill in capillary blood flow when applied into an open wound. In addition, peroxide is a poor antiseptic. Therefore peroxide should not be in open wounds.

6. Shur-clens.

Shur-clens is a non-toxic surface active agent (even when administered intravenously) effective for cleaning off dirt or grease. It probably offers little advantage over normal saline.

E. DEBRIDEMENT.

All foreign material must be removed, however tedious this may seem at times. In addition, carefully trimming irregular wound edges will result in a finer final result. Grossly devitalized tissue should be removed. Debridement can be carried out with either scalpel or scissors.

F. HEMOSTASIS.

The following steps are useful for obtaining hemostasis:

1. Apply direct pressure for 10 to 20 minutes (after all clots first removed.)

2. Epinephrine 1:1000, 1cc diluted with 4-5 cc of saline on a 4X4 gauze, held over the bleeding region for 5 minutes will stop small dermal bleeders. Larger vessels must be ligated, however, because the vasoconstriction is temporary. Contraindicated in regions where epinephrine is contraindicated (see Anesthesia).

3. Suture the wound. Very effective in wounds such as scalp lacerations which continue to slowly bleed even after local pressure. The wound must be observed for several minutes for hematoma formation, prior to bandaging.

4. Locate, clamp and ligate arterial bleeders. Pressure over a main artery (such as with a BP cuff inflated proximal to an extremity laceration), may facilitate the ligation of arterial bleeders.

Avoid ligating arterial bleeders in the hand because the digital nerves run in close proximity to the arteries.

VII. WOUND REPAIR

A. WOUND TENSION.

The tension within the skin suture loop can be decreased by first bringing together the subcutaneous tissues with buried sutures. The tension can be further decreased by undermining (see Undermining, below).

Sutures cinched with too much tension strangulate the tissue causing local necrosis. Hence the familiar surgical aphorism:

APPROXIMATE, DON'T STRANGULATE

B. UNDERMINING.

Undermine underneath the subcutaneous tissue in the natural fascial plane. Use scissors, entering with the blades closed, then opening the blades to dissect. Ideally, undermine to the width of the wound gape on both sides. Especially useful in situations where there has been tissue loss, as well as in regions where the skin is normally taught, such as the lower leg or forearm.

C. SKIN SUTURES.

1. General.

Use **monofilament nylon** or **prolene** (Maxon and PDS are acceptable substitutes). Enter the skin at about 90 degrees to the skin surface to insure that there will be as much suture at the base as the top of the suture loop, to aid in eversion.

Use only enough tension to bring the wound edges together.

In complex wounds, first suture the most difficult region.

2. Simple Interrupted Sutures (SEE ILLUSTRATION).

Double loop the first throw of the knot, and single loop the subsequent throws. Lift straight up on the sutures just prior to cinching down on the first throw as this simple maneuver serves to enhance wound edge eversion. With the second throw, pull the knot to one side of the wound edge. Use a total of 4-5 throws for monofilaments. Be sure that the suture intertwines in opposite directions with each throw, or else the knots will not square.

For cosmetic closures, use more sutures per centimeter. For facial lacerations place the sutures 2 mm from the wound edge and 2-3 mm apart. However, in the extremities use larger bites and place the sutures further apart.

3. Running Sutures (SEE ILLUSTRATION).

The first loop is identical as for the interrupted technique, but then one continues down the wound without cutting and tying until the wound is closed. This technique is quicker, and yields excellent results with practice

4. Vertical Mattress Suture (SEE ILLUSTRATION).

The vertical mattress suture insures wound edge eversion and is therefore useful in regions such as the web-space between the thumb and index finger where inversion is often a problem.

5. Horizontal Mattress (SEE ILLUSTRATION).

Few indications. May be helpful in regions of thinning skin, such as lower legs in elderly patients.

6. Half-Buried Horizontal Mattress (SEE ILLUSTRATION).

The half-buried horizontal mattress stitch goes through the subcuticular portions of angulated flaps in stellate lacerations. It is the method of choice for repairing such injuries because it preserves the vascular supply to the flap (remember that the skin vessels run in the dermis, and not at the dermal- epidermal junction).

D. BURIED SUTURES.

The deep layer serves three vital functions in insuring the optimal cosmetic outcome of a sutured facial laceration. First, the deep layer provides up to six weeks additional support to the wound after the skin sutures are out. Thus the scar is less likely to widen with time. Second, the deep layer avoids the development of unsightly pitting in the injured region caused by lack of healing of the deep portions of the wound. Third, the deep layer serves to preserve the normal functioning of the muscles of facial expression. However, when rapid closure of a wound is necessary, deep sutures can be kept to a minimum or eliminated entirely.

In the extremities, and the hand in particular, the deep sutures increase the risk of infection, and have the potential of damaging vital nerves, arteries and tendons. Hence, as a rule:

AVOID USING DEEP SUTURES IN THE EXTREMITIES. NEVER USE DEEP SUTURES IN HAND LACERATIONS.

The suture materials most commonly used for the deep layers are dexon , vycril, PDS and Maxon. Use the minimum number of sutures to close the deep tissue. The more foreign material, the greater the risk of infection.

There are two basic deep stitches used in laceration repairs:

1. The Buried Knot Stitch (SEE ILLUSTRATION).

Begin and end at the base of the wound, so as to bury the final knot. In some cases the deep sutures along a wound must all be placed prior to tying, because the tying of one suture can make the placement of the subsequent deep sutures more difficult.

2. The Buried Horizontal Mattress Stitch (SEE ILLUSTRATION).

Be careful that both sides of the wound are sutured at even depths. Do not pull tightly, or the wound will pucker.

E. SKIN TAPES (= STERI-STRIPS)

Skin tapes are useful for shallow, non-gaping wounds. Tapes are not practical for use in toddlers and young children, who have a tendency to pull them off. Never use steri-strips over the joints.

Prior to applying skin tapes, wait for full hemostasis. Tapes will not adhere to a moist areas. Next prepare adjacent skin with tincture of benzoin to enhance sticking. Benzoin is available in tiny, unit dose containers practical for most expeditions. Allow the benzoin to dry, and become tacky and then apply the strips. Finally, cover with a protective dressing.

F. SKIN STAPLES.

Disposable skin staplers are significantly faster for repairing lacerations than conventional sutures because knot tying is not needed. Staplers are light-weight, pre-sterilized and pre-loaded with staples. Disposable staple removers are also required.

Staples are easy to place, and therefore are ideal during adverse conditions.

G. Skin Glue (Tissue Adhesives)

Although available in Europe and Canada, tissue adhesives are not yet available for routine use in the United States. The two most commonly used adhesives are fibrin glue and cyanoacrylate. In a study of 1500 children, Mizrahi et al noted excellent cosmetic results using Histoacryl Blau, a butyl cyanoacrylate (J Ped Surg 23:312-313; 1988). Skin glue is commonly used by mountain climbers to cover hand wounds. Skin glue may increase slightly the risk of wound infection.

In using skin glue, first hold the wound edges together. Then apply a thin layer of glue. The glue sets in 1-2 minutes. There may be heat production during polymerization. Use extreme caution near the eyes because the glue will adhere to the cornea and lids if it runs into the eye.

H. TREATMENT OF ABRASIONS

Ground in foreign matter must be removed or else an unsightly road tattoo will result.

Abrasions should be dressed either with a topical antibiotic salve such as neosporin or silvadene, or else with a semi-occlusive dressing such as Duoderm, Tegaderm, Xeroform, or even a simple band-aid.

I. TREATMENT OF MINOR 2ND DEGREE BURNS

The treatment of minor burns is similar to that described already for abrasions. If the blister is intact, it should not be opened, because re-epithelialization is faster is the blister is not unroofed. If the blister is already un-roofed, then the open area can be cleaned with saline, and the wound covered with bacitracin, silvadene, or a xeroform dressing. Xeroform can usually be left in

place until the burn has healed.

VII. AFTER CARE.

A. CLEAN DRESSING.

Conventional band-aids are fine for small lacerations. Slight pressure will aid in hemostasis and decrease local edema. Wounds that are kept covered with an occlusive bandage heal slightly faster than wounds left open to the air.

B. ELEVATE THE WOUNDED REGION (when practical).

C. SPLINTS.

Lacerations over joints should be splinted for 5 - 7 days.

D. ANTIBIOTICS.

Prophylactic antibiotics sound like a good idea, but unfortunately they have not been proved effective in decreasing the incidence of wound infections in simple lacerations. Antibiotics have a place in certain animal and human bites.

E. WOUND CHECK.

In any case with a high potential for infection, check the wound every 2 days for signs of infection. In the case of animal bites, the first check should be at 24 hours.

F. SUTURE REMOVAL. (SEE CHART)

Unsightly suture marks occur when epithelial cells grow down the tracks of skin sutures. These tracks can form on the face of children in as little as 5 days. Hence the need for prompt removal of facial sutures. Most suture marks regress and disappear with time, however.

Suture Chart

REGION	SUTURE		SUTURE REMOVAL (days) CHILD	ADULT
THE FACE	6-0 NYLON OR PROLENE (SKIN) 5-0 VYCRIL OR DEXON (DEEP)		4	4-5
THE SCALP	3-0, 4-0, OR 5-0 NYLON OR PROLENE		5-7	7
THE HAND	5-0 OR 6-0 NYLON OR PROLENE NO DEEP SUTURES	JOINT OTHER	10-14 7-10	12-16 7-12
EXTREMITIES	4-0 OR 5-0 NYLON OR PROLENE (SKIN) 4-0 VYCRIL OR DEXON (DEEP)	JOINT OTHER	10-14 7-10	12-20 7-12
THE TRUNK	4-0 OR 5-0 NYLON OR PROLENE (SKIN) 4-0 VYCRIL OR DEXON (DEEP)		7	7-10
ORAL MUCOSA AND TONGUE	6-0 VYCRIL OR DEXON OR 4-0 OR 5-0 PLAIN		ABSORBABLE	

Because of the low tensile strength of the wound during the first 10 -20 days, lacerations on the face, and over joints should be re-enforced with skin tape following suture removal.

TETANUS PROPHYLAXIS

CLEAN WOUNDS (Clean, superficial abrasions and lacerations)

Tetanus Toxoid Immunizations	Give Tetanus Toxoid	Give Tetanus Immune Globulin
Three or more, last within 10 yr	No	No
Three or more, last > 10 yr	Yes	No
Fewer than three or unknown	Yes	No

TETANUS-PRONE WOUNDS (Contaminated, deep punctures, tenuous blood supply, extensive lacerations)

Tetanus Toxoid Immunizations	Give Tetanus Toxoid	Give Tetanus Immune Globulin
Three or more, last within 5 yrs	No	No
Three or more, last > 5 yrs	Yes	No
Fewer than three or unknown	Yes	Yes

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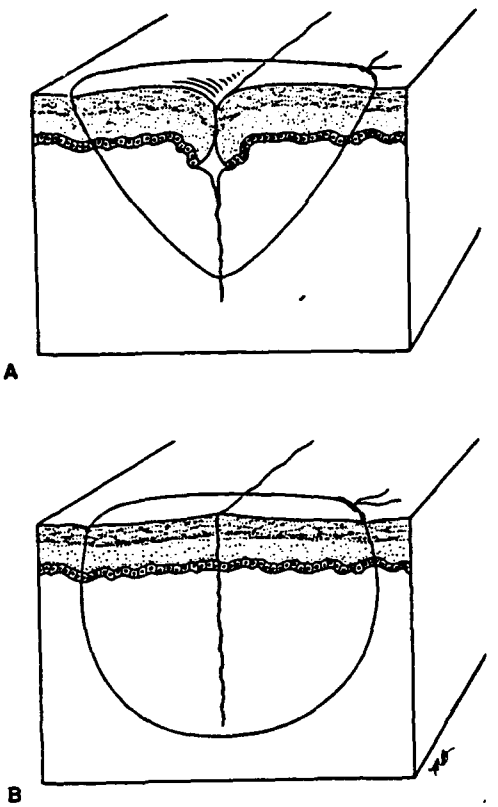
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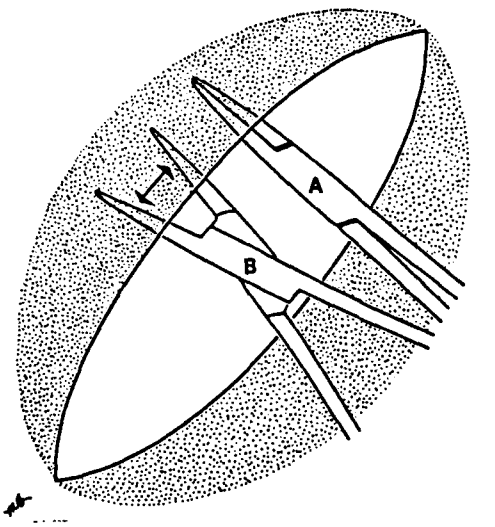
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A. The base of the loop is not broad enough, and consequently there is inversion of the wound edges. Notice how basal, regenerative epidermal cells on either side of the wound do not come into contact, thus delaying the healing. B. There is a broad base to the suture loop, and consequently the edges evert. Notice that in this instance the basal epidermal cells do come into contact, thus facilitating healing.

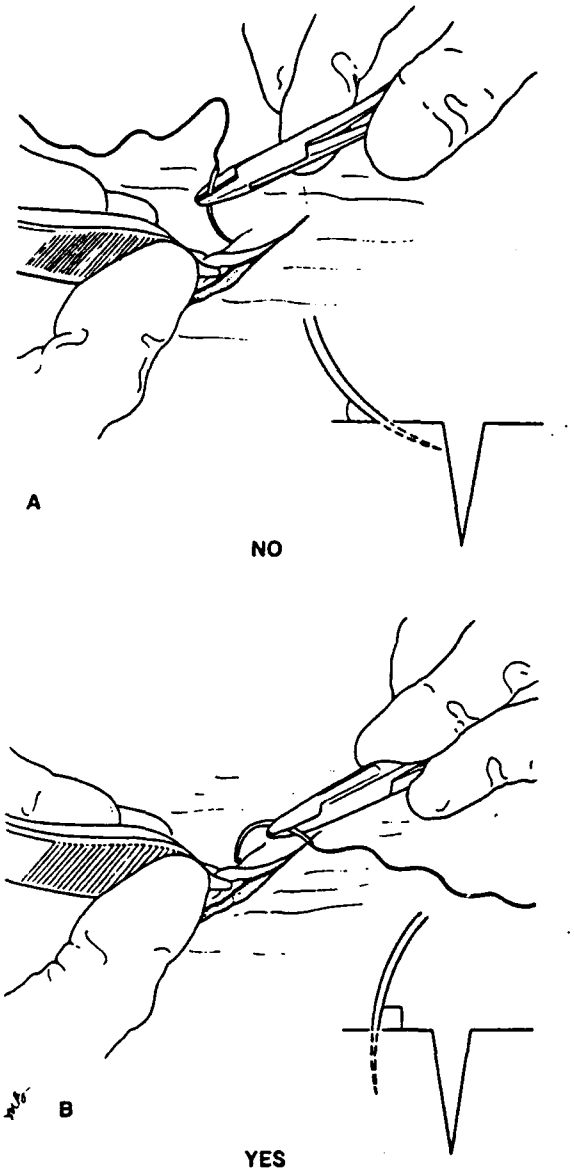
INVERSION VS EVERSION



Technique of undermining using a mosquito clamp. The extent of undermining (hatched area) on both sides of the wound should roughly equal the gape of the wound. First the clamp enters to the desired depth (A). Next the blades are opened to bluntly dissect open the tissue plane (B). An iris scissors can be used also.

UNDERMINING

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A. Do not enter the skin with the needle at a tangent, as this will yield too little tissue at the base of the suture loop. B. The skin should be entered at a 90 degree angle.

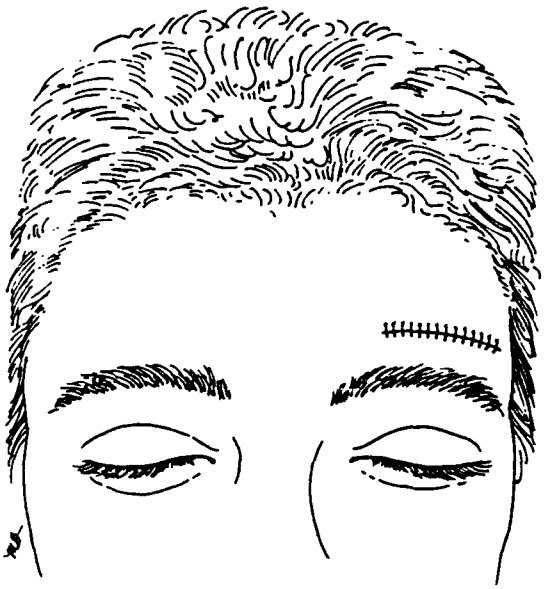
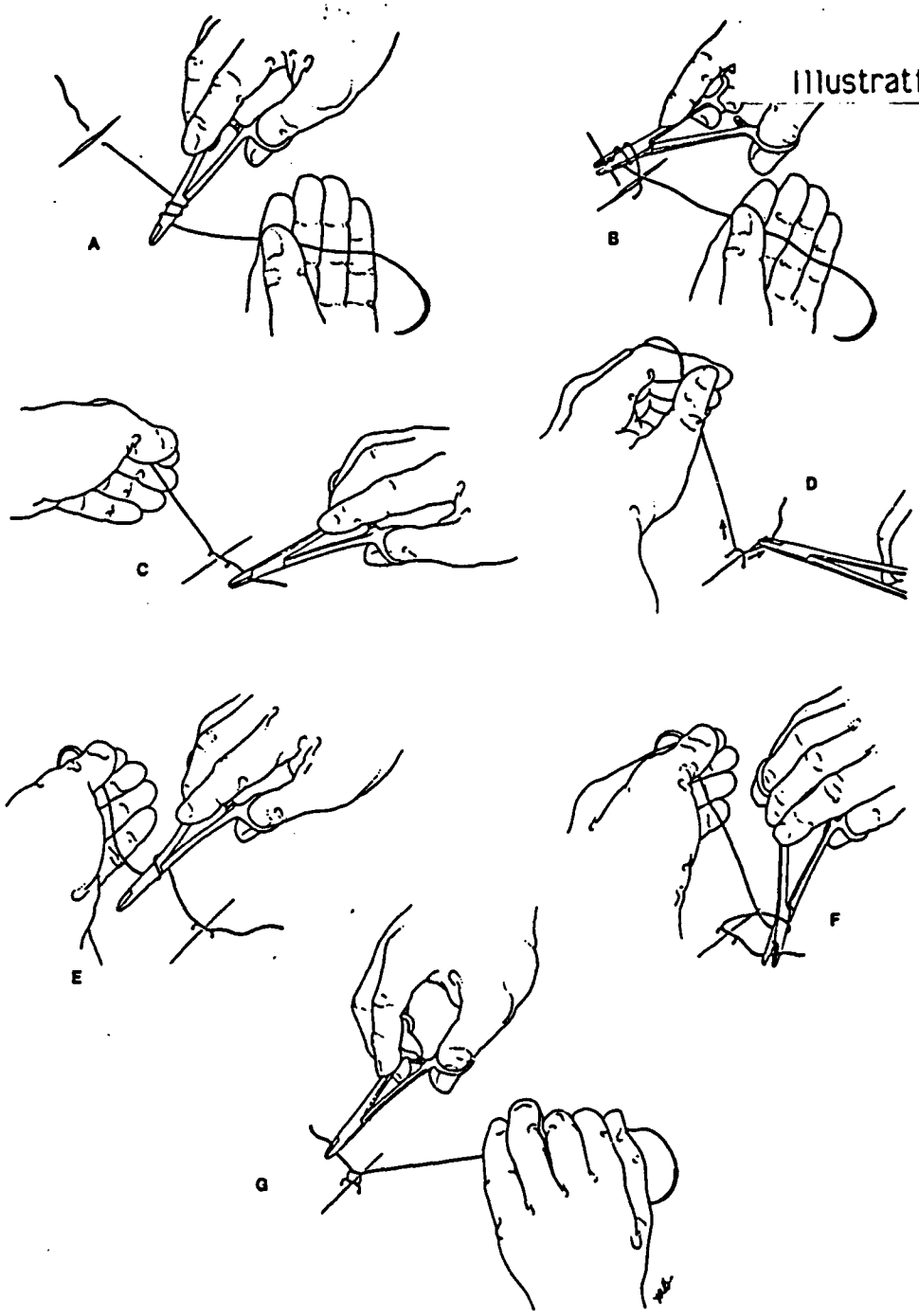


Figure 5-16 Spacing of sutures. On the face the sutures should be placed approximately 2 mm from the wound edge and 2 to 3 mm apart.

SIMPLE SUTURES, TECHNIQUE AND SPACING

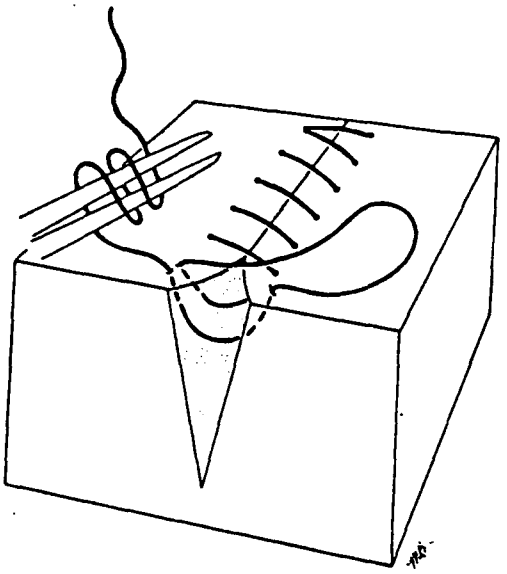
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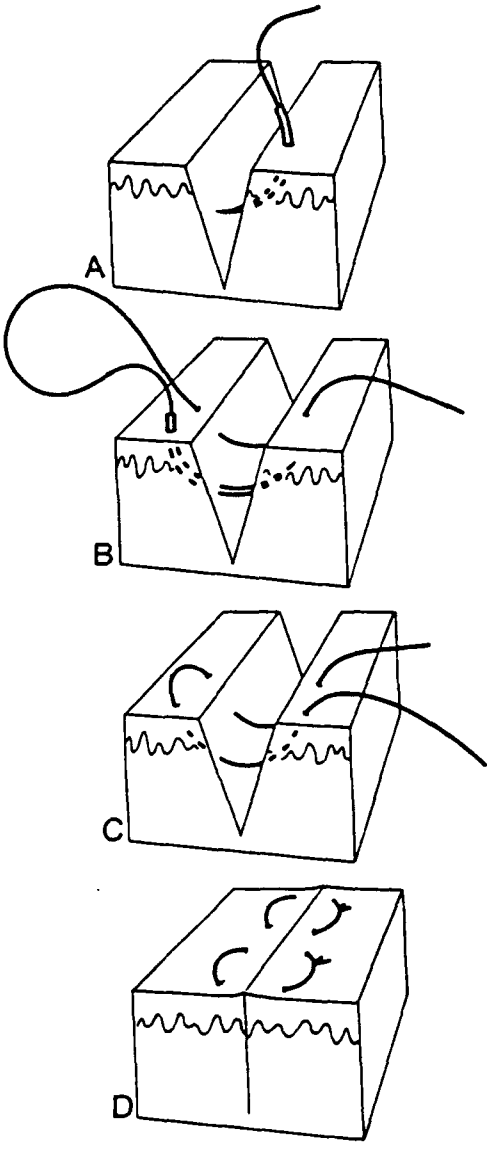
The instrument tie. During the instrument tie, manipulate the needle end of the suture with the nondominant hand and the needle holder with the dominant hand. To start with, the suture enters the far side and exits the near side of the wound in the usual fashion. A. Lay the needle holder on the suture on the near side, and then wrap the suture around the needle holder twice. B. Next reach back with the needle holders and grab the free suture end on the far side. C. Cross hands, pulling the free end back toward the near side and bringing the needle end of the suture to the far side. D. Lift up both suture ends and cinch down the first throw. E. Now lay the needle holder on the suture on the far side and loop once. F. Reach back and grab the free suture end. G. Cross hands and the knot will square. Repeat this pattern for a total of four to five throws.

SIMPLE SUTURE, INSTRUMENT TIE

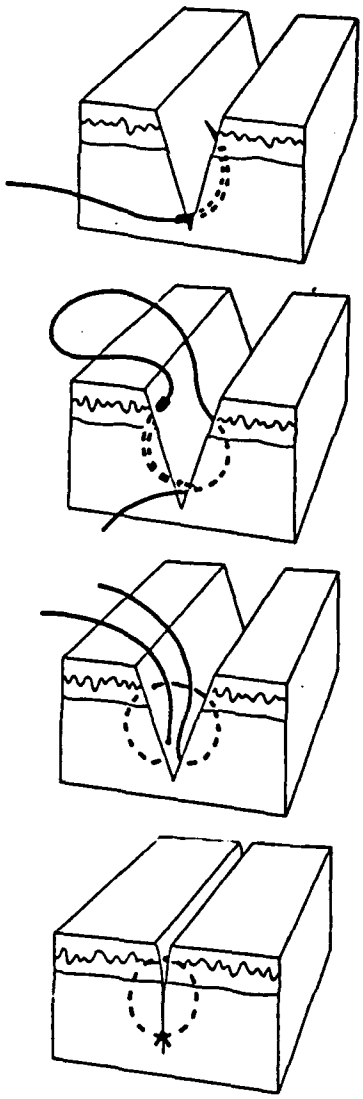
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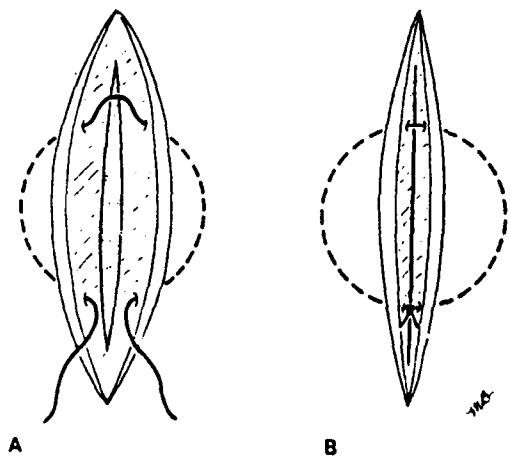
RUNNING SUTURE
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The horizontal mattress stitch. See text for discussion. *Source:* Reprinted from *Procedures and Techniques in Emergency Medicine* (p 292) by RR Simon and BE Brenner with permission of the Williams & Wilkins Company, Baltimore. © 1982.

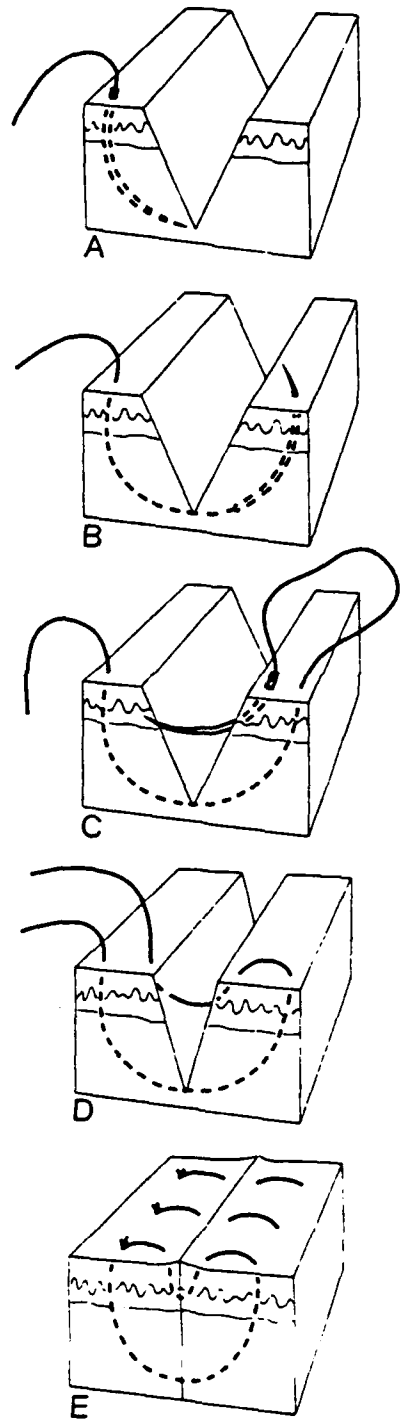


The buried knot suture. See text for discussion. *Source:* Reprinted from *Procedures and Techniques in Emergency Medicine* (p 295) by RR Simon and BE Brenner with permission of the Williams & Wilkins Company, Baltimore, © 1982.

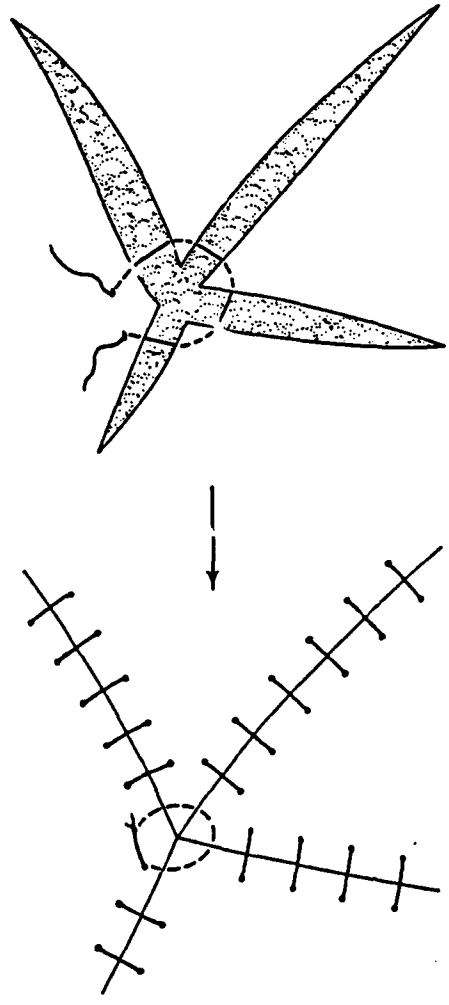


The buried horizontal mattress suture. See text

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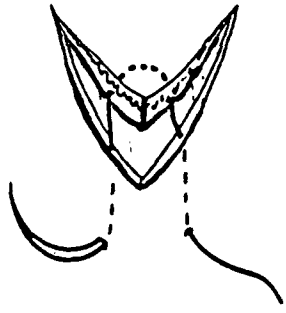


The vertical mattress suture. See text for discussion. *Source:* Reprinted from *Procedures and Techniques in Emergency Medicine* (p 291) by RR Simon and BE Brenner with permission of the Williams & Wilkins Company, Baltimore, © 1982.



Repair of a stellate laceration using a half-buried horizontal mattress stitch.

HALF-BURIED HORIZONTAL MATTRESS
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HALF-BURIED HORIZONTAL MATTRESS

LYME DISEASE: A GLOBAL PERSPECTIVE

Douglas A. Gentile, MD

OBJECTIVES:

At the completion of this presentation, the participant should have a clear understanding of:

1. The epidemiology of Lyme disease
2. The clinical manifestations of Lyme disease
3. The principals of diagnosis of Lyme disease
4. The recommendations for treatment of various manifestations of Lyme disease

I. Introduction
  A. Epidemic form of oligoarticular arthritis recognized in 1975 in Lyme, Connecticut
    1. Originally diagnosed as JRA
  B. Epidemiologic evidence suggested arthropod vector
    1. Geographic clustering
    2. Rural setting
    3. Temporal clustering - spring and summer
  C. Tick vector
    1. Erythema migrans - linked to European syndromes
      a. Ixodes ricinus - 1909
    2. Epidemiologic studies implicated Ixodes dammini
    3. Response to penicillin
  D. 1982 Burgdorfer isolated spirochete from mid-gut of Ixodes dammini ticks
    1. Antibodies in patients with Lyme disease
  E. Spirochetal etiology confirmed when isolated from blood, CSF and skin lesions
    1. Borrelia burgdorferi

II. Borrelia burgdorferi
  A. Fastidious, slow growing
    1. Difficult to isolate from patients
  B. Outer membrane unique
    1. Genes encoding located on plasmids
    2. Major antigens - OSP-A, OSP-B
  C. Differences between American and European isolates
    1. Morphology, outer surface proteins, plasmids
    2. May account for clinical variations

III. Vector and animal hosts
  A. Transmitted primarily by Ixodes ricinus complex
    1. I. dammini - northeastern and midwestern US
    2. I. pacificus - western US
    3. I. ricinus - Europe
    4. I. persulcatus - Asia
    5. I. scapularis - southeastern US
      a. Competent laboratory vector
      b. Naturally infected ticks recovered NC
  B. Other ticks and arthropods implicated
  C. Lifecycle and preferred hosts
    1. Critical that immature stages feed on same host
    2. White-footed mice major reservoir
      a. Tolerant to infection
      b. Spirochetemic throughout summer
    3. As tick feeds organism becomes systemic
      a. Organisms injected with saliva?
        (1) Ribeiro, et al - systemic infection after attachment, before engorgement
        (2) Burgdorfer
          (a) <5% of adults with systemic infection

(b) Studies show regurgitation of gut contents - tracers
(3) Animal infected without evidence of systemic infection in ticks
b. Ticks often must remain attached 24-48 hours before transmission of organism
(1) 1 of 14 at 24 hours, 5 of 14 at 48, 13 of 14 at 72
4. Transmission to humans - larva
a. Small size
b. No history of tick bite in 70%
5. Preferred adult host - white-tailed deer
a. Critical to high tick density
b. Deer population correlates with disease
c. Not involved in lifecycle of B. burgdorferi
d. Great Island - decrease in ticks after deer eliminated
6. When all features present high infection rates
a. Parts of Northeast over 60% of ticks infected
(1) 1 per square meter on well kept lawns in Westchester County
b. West Coast 1-2% I. pacificus infected

IV. Epidemiology
A. 1989 - 8333 cases reported
1. Most common tick-borne infection
B. Most from 3 endemic areas
1. Northeast, Midwest and Pacific Coast
2. Sporadic cases identified 43 states

V. Clinical manifestations
A. Multisystem involvement
B. Occurs in stages
1. Stage I - early, localized
2. Stage II - disseminated
3. Stage III - persistent
C. Patients may have one or all stages
D. Mimic other diseases

VI. Early localized infection (Stage I)
A. Characterized by erythema migrans
1. Develops 60-80% of patients
2. B. burgdorferi spreads locally in skin
B. Description of EM
1. Annular, usually centrifugally expanding plaque
2. Begin as red macules, maybe nodules
3. May have target configuration
4. Central area clear, erythematous, vesicular
5. Usually asymptomatic
a. May be pruritic or painful
6. Size variable
a. 2-60 cm diameter
b. Median 15cm at 2 weeks
7. Usually resolve within 3-4 weeks
a. "chronicum" misnomer

C. May be accompanied by regional lymphadenopathy, fever and minor constitutional symptoms

VII. Disseminated infection (Stage II)

A. Within days or weeks spirochete may spread to many sites via blood or lymph
   1. Recovered from blood during this stage
   2. Probably initially to all organs
   3. Sequesters in certain niches

B. Hematogenous spread
   1. Secondary annular skin lesions
      a. Occur in 20-50% of patients in US
      b. Resemble EM but smaller and migrate less
   2. Other skin lesions
      a. Diffuse erythema or urticaria
      b. Evanescent lesions
      c. Malar rash
   3. Excruciating headache, mild neck stiffness common
      a. Short attacks lasting hours
      b. CSF normal
   4. Migratory pains in joints, bursae, tendons, muscle and bone
      a. Last hours or days in given location
      b. No joint swelling, frank arthritis
   5. Severe malaise and fatigue

C. Sequestered infection
   1. Neurologic system
   2. Heart
   3. Joints

VIII. Neurologic involvement (Stage II)

A. 15-20% of patients in US
   1. Like other spirochetes adept at invading NS

B. Classic triad
   1. Lymphocytic meningitis
   2. Cranial neuritis
   3. Radiculoneuritis

C. Central nervous system involvement
   1. Aseptic meningitis
      a. Headache, stiff neck
      b. Lymphocytic pleocytosis
         (1) 100 cells
         (2) Elevated protein, normal glucose
   2. Encephalopathy
      a. Fatigue, memory and cognitive difficulty
         (1) Most nonspecific assoc. with febrile illness
         (2) Some with evidence for mild encephalitis

D. Cranial neuritis
   1. Facial nerve palsy common
      a. May be only abnormality
      b. Usually with CSF pleocytosis
   2. Also II, III, IV, V, VI, VIII

E. Peripheral nervous system

1. Multifocal axonal neuropathy
    a. Painful radiculitis
    b. "Guillain Barre-like"
    c. Brachial/lumbosacral plexitis
    d. Mononeuritis multiplex
2. Carpal tunnel syndrome

IX. Cardiac (4-8%)
A. Fluctuating A-V block
1. First degree, Wenckebach, complete heart block
    a. Usually brief
        (1) Complete heart block rarely longer than one week
        (2) May require temporary pacemaker
    b. Require hospitalization, ECG monitoring
B. Myopericarditis
1. ECG changes - ST and T-wave
2. Left ventricular dysfunction

X. Arthritis
A. 60% of patients
B. Usually several months after onset (mean 6)
C. Localized intermittent musculoskeletal pain
1. Joints, periarticular areas, tendons, bursa, enthesis, muscle, bone
2. Individual episodes short
    a. Hours to days
3. Migratory - up to 10 different regions
4. Often asymptomatic between episodes
D. Transient arthritis with synovial thickening/effusion
1. Duration days to months (mean 3 months)
    a. Declines over time
2. Oligoarticular 70%
    a. Knee, shoulder, ankle, elbow, TMJ, wrist, hip, small joints
3. Synovial fluid
    a. 5,000 to >100,000 cells (median 25,000)
    b. PMN's 80%
    c. Immune complexes present

XI. Less common features
A. ARDS, hepatitis, myositis, osteomyelitis, iritis
B. In Europe lymphocytoma

XII. Stage III - persistent infection
A. Skin
B. Musculoskeletal system
C. Neurologic system
D. Eyes

XIII. Skin
A. Acrodermatitis chronica atrophicans
B. Primarily Europe

XIV. Chronic arthritis
A. 10% of patients with untreated EM
B. One year or more of continual joint inflammation
C. Begins mean of 12 after EM
D. Knees most common - also hips, shoulder, wrist, elbow

E. Severe cases radiographic changes
    1. Erosion of cartilage and bone
F. Immunogenetic basis
    1. HLA-DR4

XV. Chronic nervous system involvement
A. Europe
    1. Progressive encephalomyelitis
        a. Spastic paraparesis
        b. Ataxia
        c. Cognitive impairment
        d. Bladder dysfunction
        e. Cranial neuropathy
    2. Intrathecal IgG to B. burgdorferi
B. U.S.
    1. Months to years after EM
    2. Three syndromes (often overlap)
        a. Encephalopathy
            (1) Affects memory, mood and sleep
            (2) Occasional language disturbance
        b. Polyneuropathy
            (1) Sensory symptoms
                (a) Distal paresthesia
                (b) Radicular pain
            (2) EMG - denervation
        c. Leukoencephalitis
            (1) MRI - peripheral white matter and periventricular lesions
            (2) Spasticity, bladder dysfunction
            (3) Normal evoked potentials, absence of myelin basic protein

XVI. Chronic fatigue, keratitis

XVII. Congenital infection
A. Most pregnancies normal
B. Fetal transmission of B. burgdorferi does occur
    1. Fetal death may ensue
C. Parenteral antibiotics?

XVIII. Diagnosis
A. Serology only practical laboratory aid
B. Stage I
    1. Minimal mononuclear cell response
    2. Specific antibody evolves slowly
        a. Within 2-4 weeks IgM to 41-kd flagellin
        b. Capture IgM ELISA more sensitive
    3. Diagnosis largely clinical
C. Stage II
    1. Heightened mononuclear cell response
    2. Polyclonal activation of B-cells
        a. Increased total serum IgM
    3. Gradually specific IgG antibody develops
        a. Between second and third months
        b. Flagellar and OSP antigens
        c. May persist for years
    4. ELISA more sensitive and specific than IFA
        a. Elevated in almost all patients after

first few weeks
5. Serologic tests must be correlated with clinical abnormalities
a. Serologic testing not yet standardized
(1) Significant variability within and between laboratories

D. False negatives
1. Early Lyme disease
2. Early antibiotics
a. Suppress humoral response
b. T-cell proliferative response

E. False positives
1. Cross-reactivity
a. Relapsing fever
b. Leptospirosis
c. Syphilis
(1) No anti-cardiolipin Ab
(2) VDRL normal
(3) Cross reaction with FTA-ABS
d. RMSF
e. Rheumatoid arthritis, SLE
2. Persistent antibody
a. Asymptomatic infections
3. Atypical cases or serology to r/o Lyme
a. High false positive rates

F. Improvements
1. Standardization
2. Western blot analysis
3. Specific antigens
4. Polymerase chain reaction
a. Amplify B. burgdorferi genome sequences

G. Recommendations
1. Early Lyme disease
a. IgM ELIZA
(1) Specific Ag if available (flagellum)
(2) Capture antibody
2. Stage II or III
a. IgM and IgG ELIZA
3. Paired sera to same lab at same time
4. Indeterminate results - confirm with Western blot

XIX. Treatment
A. Antibiotic sensitivities to B. burgdorferi
1. Highly sensitive
a. Tetracycline
b. Ampicillin
c. Ceftriaxone
d. Erythromycin
(1) In vivo < in vitro
2. Moderately sensitive
a. Penicillin
(1) Penicillin G > penicillin V
b. Chloramphenicol
c. Oxacillin

B. Early Lyme disease (Stage I, hematogenous Stage II)
   1. Tetracycline 250mg 4x daily, 10-30 days
   2. Doxycycline 100mg 2x daily, 10-30 days
      a. Better tissue levels
      b. Longer half life
      c. Less GI upset
   3. Amoxicillin 500mg 3x daily, 10-30 days
      a. Preferred to penicillin V
   4. Erythromycin 500mg 3x daily, 10-30 days
      a. Alternative for children with PCN allergy

C. Neurologic abnormalities
   1. Ceftriaxone 2g IV daily, 14 days
      a. Long half life
      b. Crosses blood-brain barrier readily
      c. Effective in some patients who did not respond to PCN
   2. Penicillin G, 20 million units IV, 6 divided doses daily, 14 days
   3. Doxycycline 100mg orally 2x daily, 30 days
   4. Chloramphenicol 1 g IV 4x daily, 14 days

D. Facial Palsy alone (normal CSF)
   1. Oral regimens may be adequate

E. Cardiac abnormalities
   1. First degree A-V block (PR < 0.3 sec)
      a. Oral regimens
   2. High-degree block
      a. Ceftriaxone, Penicillin G

F. Arthritis
   1. Doxycycline 100mg 2x daily, 30 days
   2. Amoxicillin and probenecid, 500mg each 4x daily, 30 days
   3. Ceftriaxone
   4. Penicillin G

G. Acrodermatitis
   1. Oral regimens for 30 days

H. Stage III Lyme disease
   1. Response less predictable, slower

I. Pregnancy
   1. Penicillin G, 20 million units IV, 14 days
   2. Erythema migrans alone - amoxicillin 500mg 3x daily, 21 days

LEARNING FROM EXPERIENCE: TRAVEL MEDICINE IN KATHMANDU

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Learning from Experience

Abstract

To understand the risks of illness in a population of travelers and expatriates in Nepal, we recorded the diagnoses of every patient who utilized our clinic from 1987 to 1991. There were 19,616 diagnoses recorded. Visits for gastrointestinal illness accounted for 31% of the patient visits; respiratory illness accounted for an additional 21%. Visits for diarrheal disease were more common among travelers than foreign residents. The diarrheal disease rate showed a distinct seasonality, with a peak in April, May, and June each year. Febrile illnesses such as dengue fever and enteric fever showed marked longitudinal variations in illness rates over the four years. Malaria was an uncommon diagnosis, with only 12 laboratory proven cases (all Plasmodium vivax). Typhoid vaccine efficacy was studied which showed good protection from the parenteral vaccine, but cast doubt on the efficacy of the oral typhoid vaccine. Immune serum globulin appeared to offer excellent protection against hepatitis A.

Learning from Experience

Introduction

Travel medicine is growing rapidly as an area of special interest within medicine. Most clinics that devote themselves to travel medicine give routine immunizations and prophylaxis advice to people before they travel. The majority of returning travelers who are ill still seek care from their personal physicians rather than from these new clinics. Thus, it is difficult to gather data as to what actually happens to travelers when they are traveling, except by utilizing questionnaires that rely on the patients memory and perhaps incorrect diagnoses abroad.

Until recently, most advice regarding travel-related illnesses was based on short-term focused studies, or data that related to local populations. Traveler's illneses may vary over time, and travelers themselves may differ from local populations in significant ways. These differences include the ideas that: 1) Travelers may be generally healthier and better able to withstand illnesses; 2) They make more effort to avoid illness; 3) They tend to present earlier in the course of their illnesses; and 4) They have access to more expensive medications. Learning which illnesses travelers acquire most often, how they present, and which treatments are most effective will help establish travel medicine as a distinct discipline.

Learning from Experience

Methods

The CIWEC Clinic is an expatriate and tourist oriented outpatient facility in Kathmandu, Nepal, that has been operating since 1982. It is ideally located to study foreigners in Nepal, since the vast majority of foreign residents reside in Kathmandu, or return there for their medical care. Ninety per cent of tourists enter and exit the country from Kathmandu. All serious illnesses or accidents are evacuated to Kathmandu from the rest of Nepal.

From 1 March 1987 through 28 February 1991 we kept a database which recorded the diagnoses of every patient who utilized the clinic. The data was recorded by the practitioners at the clinic each day as they saw the patients. Many diagnoses were subjective, but important diseases were documented by stool examination, blood culture or serology. Multiple visits for the same illness were recorded as one diagnosis. If the same patient returned later with a separate illness, it was recorded as a separate visit. The database was entered into a computer using a commercially available database system. For the purposes of the study a "resident" is a person who was not a Nepalese citizen who was residing in Nepal. A "tourist" is any person from a country other than Nepal who is temporarily visiting in Nepal, usually for less than three months.

Results

Diagnoses were recorded for 19,616 patient visits during the four years. Citizens from 76 countries were seen at the clinic, of which 49% were from European countries, and 40% were from North America. Only 3% of the patients were Nepalese citizens. Age and sex data were not recorded. The overall disease categories for residents and tourists are shown in Figure 1. There was a significant difference between the percentage of tourists who presented with a diarrheal illness and the percentage of residents who presented with a diarrheal illness ($p<0.00001$).

The seasonality of three diarrheal diseases is shown in Figure 2. The transmission of all three illnesses was increased in the period prior to the peak of the monsoon in July. The Giardia and Entamoeba hisolytica peaks are delayed about one month from the bacterial dysentery peak. This is probably due to the longer incubation period and the longer time that people tolerate the symptoms of these two protozoal illnesses before seeking medical care.

The monthly tourist arrival rate has peaks in the spring and fall seasons, with significant drops in between. About 200,000 tourists a year arrived in Nepal during the study period. Even though the fall season sees more tourists than the spring season, there was no concomittant peak in diarrheal illness. Two other enterically-transmitted diseases also peaked just before the heaviest

monsoon rains in July: enteric fever, and hepatitis A. Both these illnesses had their highest rates in June.

Thus, every enterically transmitted illness that we studied had a peak transmission rate in the three months preceding the monsoon rains.

The febrile illnesses in which we were most interested were enteric fever, dengue fever, malaria, and hepatitis. Malaria was rare, with only 12 cases recorded during the four years, all of which were Plasmodium vivax.

The monthly and yearly distribution of enteric fever is shown in Figure 3. There was a large increase in the number of enteric fever cases in the fall of 1987. Since then the diagnosis has been made less frequently. In 1990, we did 48 blood cultures for suspected enteric fever, with only 2 positive results.

The vaccine status of the enteric fever cases was studied from 1987 to 1988, and the results reported elsewhere.[1] We compared a mostly non-vaccinated group of Israeli travelers (6% received typhoid vaccine), to a mostly vaccinated group of other Western travelers (90% received typhoid vaccine). The Israelis had culture proven enteric fever at a rate of at least 14 times the other vaccinated Westerners. Only 10% of Western travelers had used the oral typhoid vaccine (Berne Vivotif), but 4 of 7 (57%) of the culture-proven Salmonella typhi infections in vaccinated people occurred in those using the oral vaccine.

Learning from Experience

Dengue fever cases tended to be clustered in the fall season. Dengue is not endemic in Nepal, and all cases were imported from Northern India or Thailand. The fall of 1990 showed a marked increase in cases over the previous three years, even though tourism was not increased that year.

Viral hepatitis serology was obtained from all clinically diagnosed hepatitis patients. In a separate study from 1986 to 1988, 52 hepatitis patients were identified, of which 22 were travelers.[2] Foreign residents accounted for 30 cases of hepatitis, of which 5 were proven to be hepatitis E. One of the 22 travelers died of fulminant hepatitis A. None of the patients diagnosed as having hepatitis A had received immune serum globulin within the four months prior to illness.

Discussion

Diarrheal illness, respiratory infection, and dermatologic problems account for 75% of traveler's medical visits in Nepal. Respiratory illness is common (21%), and antibiotic treatment of the complications of respiratory infection is also common. Travelers to Nepal should receive advice on how to treat the complications of colds, and carry appropriate medication in their first aid kits.

Diarrheal disease proved to have a distinct seasonality, affecting many more people in the spring trekking season than in the fall. All forms of enteric illness had increased transmission in these same months, suggesting a common vector of transmission. The hot season prior to the monsoon is associated with the greatest amount of fly activity, and contaminated flies may well account for the increased diarrheal disease at this time of year. The heaviest monsoon rains occur in July, and during that month, the rate of diarrheal illness drops dramatically, and remains at that lower rate throughout the rest of the year. The number of flies decreases after the heavy rains, but this has not been formally studied. Flies have been shown to be capable of carrying enteric pathogens,[3] and reduction of flies in an army camp setting resulted in decreased diarrheal illness.[4]

The implication of these findings is that routine diarrhea prophylaxis advice to travelers may not be sufficient protection during times of increased fly

activity, as food could be contaminated right up to the time of serving. Hotels and restaurants in developing countries may have to be taught to exclude flies from their kitchens during these seasons of increased risk.

Two of the febrile illnesses that we studied showed both seasonal and longitudinal variations over four years. The longitudinal variation was most striking with the increase in enteric fever during the fall of 1987, and decreased diagnosis of that disease in 1990; dengue fever was also greatly increased in the fall of 1990. These wide variations of disease rates could prove to be confounders of short-term studies of returning travelers, leading to conclusions that either drastically over- or underestimate the risks of certain diseases. Long-term surveillance will be necessary to understand the risks to travelers of important diseases.

The efficacy of immunoprophylaxis advice was also able to be studied. Parenteral typhoid vaccine was shown to be highly effective, but questions were raised about the efficacy of the oral typhoid vaccine. Immune serum globulin (ISG) protection against hepatitis A appears to be highly effective, as we did not diagnose a case of clinical hepatitis A in anyone receiving ISG within the past four months.

As travel medicine begins to establish itself as a separate discipline, data such as we have gathered in Nepal will be necessary to provide a scientific foundation to the

advice that is given to travelers, and to facilitate the diagnosis of illness in returning travelers. Studies of disease risk, and the effectiveness of immunoprophylactic advice can be monitored worldwide if travel medicine clinics could cooperate in keeping a common database for all travelers utilizing these facilities. Studying travelers also offers the benefit of learning about the diseases themselves, as our data on the seasonality of enteric diseases demonstrates. Gathering data on travelers and foreigners abroad can help define the disease risks for travelers to specific destinations. Cooperative data gathering should be a priority as travel medicine looks to the future.

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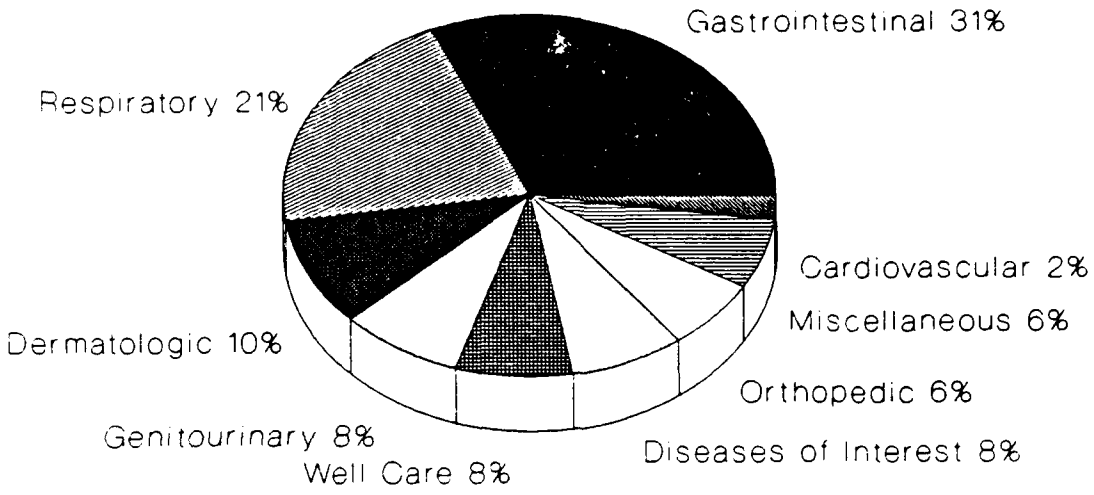


Figure 1: The percentage of visits for various diagnoses by category, for all patients. Only first visits for a particular diagnosis were counted for this chart. N=14, 859.

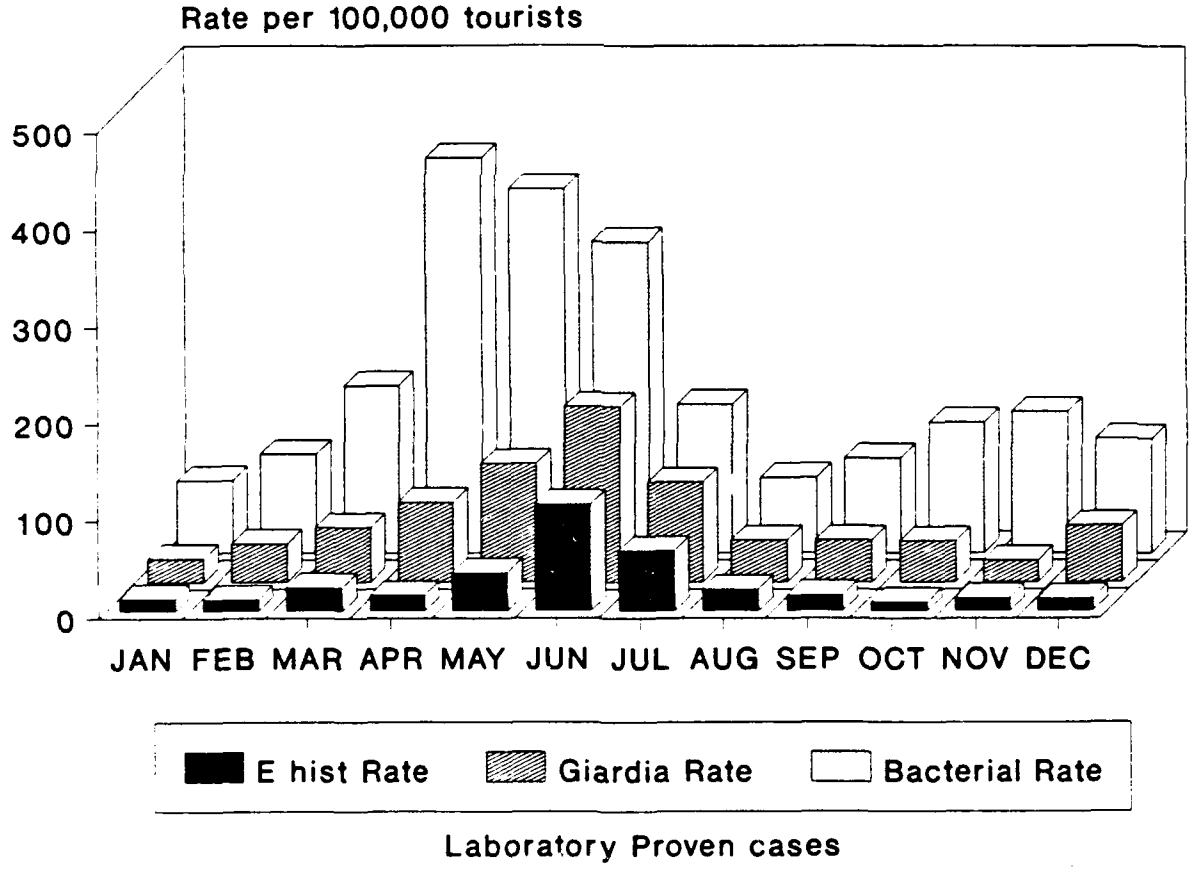


Figure 2: The rate of diarrheal illness by month for three types of diarrhea. The rate was achieved by dividing the number of laboratory proven cases by the average number of tourists entering Nepal during the specific month. This rate does not reflect the actual attack rate of diarrhea in Nepal, but should be a consistent way to compare each month. Bacterial diarrhea was assumed when a significant number of white cells were observed in a stool exam. Giardia and Entamoeba-histolytica cases were based on positive stool exams.

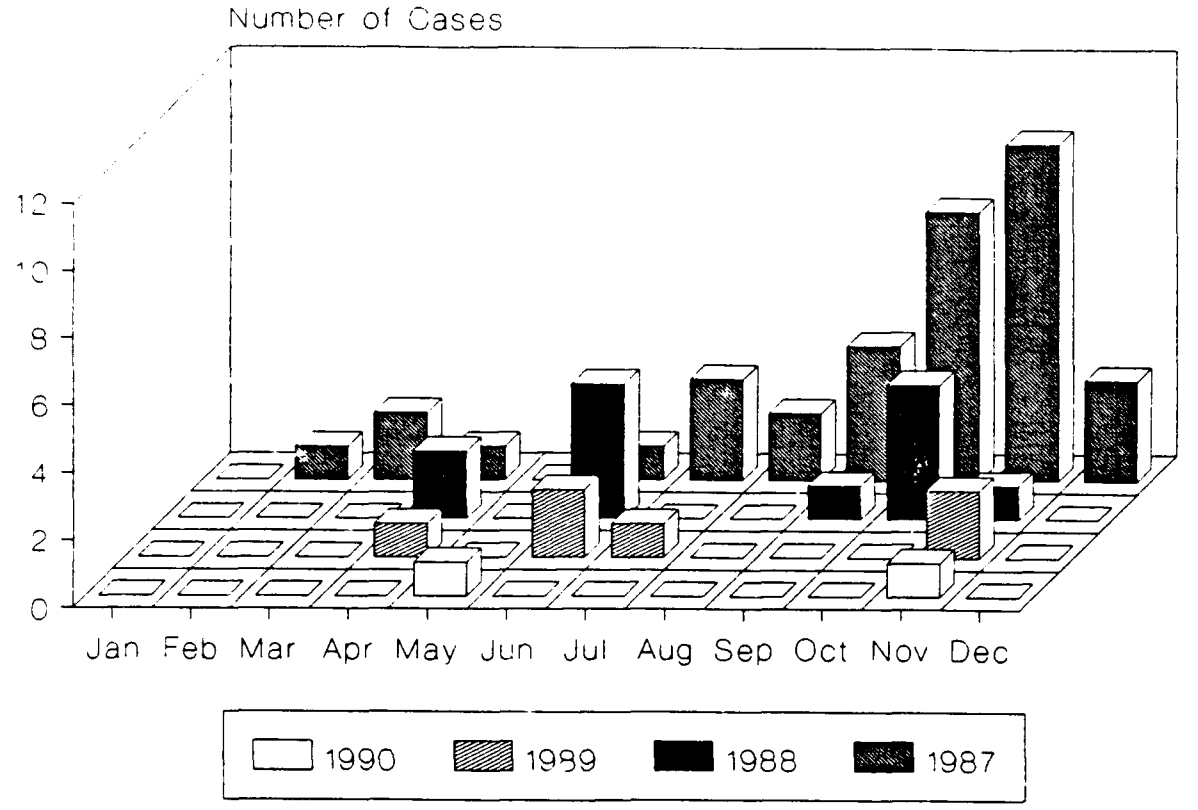


Figure 3: Blood culture proven enteric fever cases (infection with either Salmonella-typhi or Salmonella paratyphi-A) by month and year.

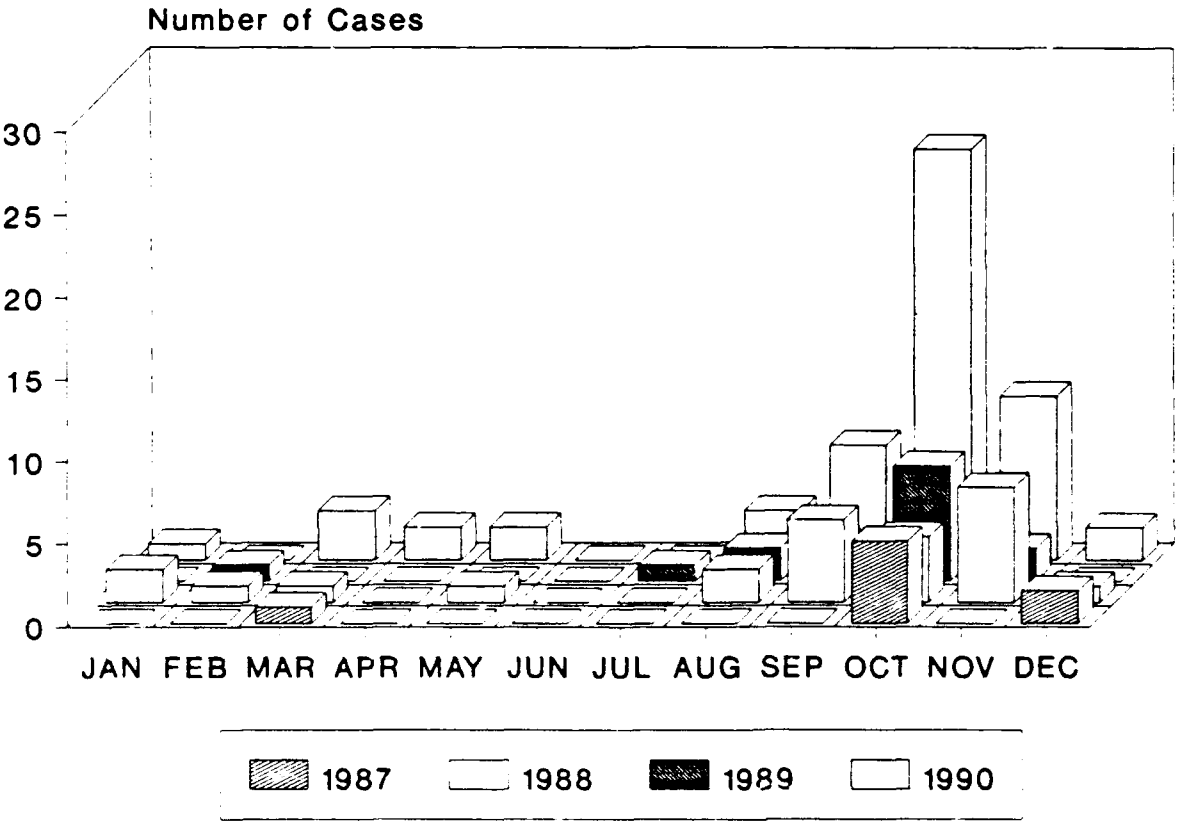


Figure 4: Dengue fever cases by month and year. Most cases were confirmed by acute and convalescent serology; other cases were very typical of dengue fever clinically.

CONTROVERSIES IN HYPOTHERMIA: THE AFTERDROP PHENOMENON

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Learning Objectives:

Following this presentation, participants will be able to understand why there is still confusion and controversy regarding protocols for first-aid treatment of accidental hypothermia in wilderness situations. With this understanding, participants should be able to improve their ability to evaluate various treatment strategies and implement a safe and effective protocol of their own.

Outline of Course Material:

- Need to concentrate on problems of pre-hospital care
- Need to concentrate on severe hypothermia
  - emphasis on "still alive" victims
- Physiological problems of first-aid treatment of severe hypothermia
- Emphasis on the basic thermal problem
- Limitations of experimental information
- Reasons for confusion and controversy
- Emphasis on need to understand "afterdrop"
  - the two causes and their interaction
  - afterdrop can be much larger than expected
    - case history and experimental evidence
- Primary thermal objective of first-aid: "stabilize" (arrest afterdrop) versus "rewarm"
- The "brown fat analogy"
- Heat-donation devices to be considered
- Basic protocol: the INSTANT response
- Need for improving treatment procedures in emergency transport vehicles (helicopters, ambulances, etc)
- Future research needs

DESERT SURVIVAL

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Following this presentation, participants will be able to identify the hazards associated with the desert environment and be familiar with the fundamentals of survival in general and desert survival in particular.

I. Geography

Deserts comprise about 15% of the earth's land area and approximately 8% of the land area of the United States or 300,000 square miles of desert. Most American deserts are adjacent to National Parks and Forests and are frequently visited, i.e., Grand Canyon, Great Basin, Big Bend, Arches, Zion, Organ Pipe, Joshua Tree, et al. An area that has less than 10 inches (25 cm) of rain, unevenly distributed throughout the year, is considered desert. Most deserts are found between 30 degrees South and 30 degrees North latitude, making them hot as well as dry, i.e., Sahara, Arabian, Australian, and Kalahari. There are several large areas of "cold" desert, i.e., the Gobi, Great Basin, and Patagonia that are found beyond 40 degrees North and South latitudes and have variable temperatures.

There are several climatic processes that produce desert areas. The most important of these are the six cells of air currents that descend at the poles and near the Tropic of Cancer and Tropic of Capricorn. These air currents, driven by the sun and the rotation of

the earth, create areas of relatively warm, dry climate. The second important process is the problem of rain shadows caused by mountain ranges along the western edge of the continents. These areas lie to leeward of the prevailing winds, and moist oceanic air is unable to rise over the mountains before it cools and loses its moisture on the western slopes. The dry air then rises over the mountain and descends to the land drying it. The Andes shadow the Patagonian Desert, the Sierra Nevada and Cascades shadow the Great Basin and Mojave Deserts, and the Great Dividing Range in Australia places most of that continent in a rain shadow. The dry land that is formed is unable to sustain a large amount of plant life. This lack of vegetation allows the sun's energy to directly heat the ground and rocks and concentrate in the soil and air directly above it. In a forested region the plants give off moisture via transpiration which, along with the plants themselves, absorbs most of the sun's energy before it can heat the soil. The large surface area of the vegetation also disperses the heat energy so that the temperature on a forest floor rarely rises above 100 F (39C). The combination of solar radiation, high winds, and hot temperatures causes an increase in the evaporation of any moisture that does reach the desert. The same factors that cause a high temperature during the day allow for a rapid loss of heat during the night. Temperatures may vary 40 to 45 degrees F in a single 24 hour period. It would seem that this climate would only allow for a sparse flora and fauna. This is not the case. Death Valley, where air temperatures have been recorded at 134 degrees F, has 600 species of plant, 30 species of mammal, 25 species of reptile, and two species of fish. The plants and animals that have evolved in this environment have developed ingenious methods for adapting to the aridity and extremes of temperature. Man, in order to survive in the desert, must adopt some of the same methods used by the indigenous organisms.

II. Preparation

Obviously, the more prepared someone is the more likely he or she will survive, all other things being equal. Things are never equal, however, and luck is probably the most important, albeit the most uncontrollable, factor. There are several controllable factors, namely physical conditioning, clothing, survival kit, and survival skills, that may prevent needless deaths in the desert.

A. Physical conditioning and acclimatization are probably just as important in desert travel as in mountaineering. The body's need for water cannot be lessened by these methods, but the amount of electrolytes lost and the efficiency of the sweating apparatus can be optimized. Lower body strength may help to prevent a minor injury such as an ankle fracture that in the desert environment could be fatal if the injured person was unable to get help or water. Prior to a trip to the desert for the unacclimatized individual, I recommend a level of fitness at least equivalent to an aerobic workout to 80% of maximum heart rate for 30 minutes four times a week. Upon arriving in the desert area, the individual should spend at least three days acclimatizing before starting out on any long hikes (more than five miles). This will allow for the increased intake of water which may be three to five gallons per day, the adjustment to the lack of vegetation and increased solar radiation, and the large temperature variations. Most activity should take place between dawn and 10 AM and between 3 PM and dusk. Between 10 AM and 3 PM, the hottest time of the day, it is best to stay in the shade and sleep, read, or handle domestic duties.

The most important conditioning is mental conditioning or "the will to survive." Throughout the survival literature this factor is constantly cited as the one thing that brought

survivors through their ordeal. Unfortunately, this cannot be taught. The potential, however, is in all of us but may only appear in extreme circumstances. Many medical personnel develop a sense of "aequanimitas" dealing with emergencies on a daily basis; jet pilots seem to have a similar trait. While the "will to survive" is not exactly the same as this trait, it may arise in unlikely individuals just as courage in battle does.

B. The clothing most suitable for the desert is similar to that worn in most wilderness areas, only the type of material may be different. Polypropylene, wool, pile, and goretex are the choices of the mountaineer whose enemy is hypothermia. While hypothermia is a possibility on the desert, hyperthermia and dehydration are more likely. Ripstop cotton is ideal due to its evaporative ability, and long sleeve shirts and trousers made from this material are excellent for desert conditions. A light color should be chosen to help reflect solar radiation. A pile jacket or wool sweater is needed at night in many desert areas. Most novices travelling in the desert remark on how cold it is at night and how ill prepared they were for it. A rule of thumb is "expose as little skin as possible." The skin must be protected from heat, ultraviolet rays, blowing sand, insects, and water loss. A hat is an absolute necessity and should be broad brimmed or a kepi to protect the neck and face. A cravat or large handkerchief can be used as an emergency hat if the other is lost. It can also be used as a towel or soaked in water and placed between the head and the hat to act as a solar air conditioner. Sunscreen and chapstick should be used frequently to protect exposed skin. Sunglasses or goggles are needed to protect the eyes. Ultraviolet keratitis similar to snow blindness can occur, especially at higher elevations. Corneal abrasions from blowing sand are quite common and preventable. Leather gloves protect the hands from hot objects as well as cactus spines and thorns. Footwear can be leather or

manmade materials and should be ankle high or higher. Low cut shoes will allow sand to enter and do not give adequate ankle support. Jungle boots with metal spike protection and running shoes may get extremely hot in the desert soil and are not good choices for desert travel. Polypropylene or polypropylene and wool socks seem to decrease the amount of blistering and give adequate cushioning and insulation to the feet.

C. The survival kit should have the necessary equipment and supplies to help you survive yet be small enough to be carried with you wherever you hike in the desert. The principle components should be water or the means of acquiring it. Unfortunately, water weighs eight pounds to the gallon and this limits the amount that an individual can carry to about one day's supply. A solar still should be carried along with water purifying tablets or iodine. The best way out of a survival situation is to be rescued, therefore signalling equipment is essential. Survival kit items should have multiple uses and they must be of high quality. Do not spare expense in purchasing equipment that your life may depend on. The following list is an example of a basic kit that may be carried in a relatively small pack:

Nylon canteen, 5 quart
Canteen cup
Clear plastic sheet, 5'x5'
Plastic tubing, 5'
Iodine crystals
Bandaids
Swiss Army knife
Waterproof matchbox
Penlight
Space blanket
Sunscreen
Signal mirror
Whistle
Sunglasses
Parachute cord, 50'
Compass

Insect repellant	Hard candy
Safety pins	Needle/thread

When weight is not a consideration, when travelling in a car for example, the following items should be carried:

Plastic water container, 5 gallon/person	Toilet paper
Folding shovel	First aid kit
Nylon tarp	Gloves
Extra clothing	Food
CB radio	

Necessity being the mother of invention, many items from vehicles can be used in an emergency: the rear view mirror can be used to signal, the hubcaps to collect water, batteries to start fires, oil to produce smudge for signalling.

D. The best survival kit is of no use if the person carrying it does not have the skills to use it correctly. These skills must be learned and practiced regularly or they will be lost. Until you have actually built a solar still you cannot anticipate the problems encountered. An experienced person can produce a solar still in 15 minutes. An amateur will take 60 minutes and lose more sweat than the still can produce in a week. Direction finding, fire building, shelter construction, and signalling are all necessary skills that must be learned before getting into a situation where your life may depend upon them. It is too late to learn to swim after you have fallen out of the boat. Priorities in a survival situation are based on the "rule of threes." You can live three minutes without oxygen, three hours

without warmth, three days without water, and three months without food. Assuming that there are no immediate medical problems or environmental hazards, e.g., fractured pelvis, landslide, flash flood, etc., the top priority in a desert survival situation will probably be water.

1. If there is a limited amount of water available, food should not be eaten unless the food contains a large amount of water. The metabolism of food digestion and excretion of waste products requires unnecessary usage of water. Water obtained from lakes, streams, wells, or springs should be considered contaminated and purified before drinking. Water may sometimes be found by digging at the outside bend of a dry riverbed or stream. Vomiting and diarrhea caused by contaminated water could be quickly fatal in the desert. Rainwater, dew, and water obtained from solar or vegetable stills is relatively pure. Urine, seawater, or brackish water should never be drunk in a survival situation. Liquid from radiators is contaminated by glycols and should never be drunk. Many plants such as barrel cactus and traveller's tree and animals such as the desert tortoise contain water which can be used in an emergency. A solar still can be made by stretching a 5'x5' piece of clear plastic over a hole dug in the ground into which vegetation, urine, or brackish water has been placed. The sun will cause water to evaporate and collect on the underside of the plastic and then drip back into a container at the bottom of the hole. A tube can be used to remove the water from the container without dismantling the still. The amount of water produced will depend on the amount of moisture in the hole; bone dry sand will not be very productive. Build the still at night to conserve water. Dew, rainwater, and edible animals may also be collected in the still as a bonus (see illustration).

2. Shelter is essential if the effects of the sun during the midday are to be

ameliorated. The temperature in the desert will vary both above and below the ground. The temperature at the ground surface will be the highest. It will decrease as one goes either below or above the ground. Therefore, a shelter that protects from direct solar rays and has within it either a trench 12 to 18 inches deep or a platform 12 to 18 inches high will be cooler than one in which you must be in contact with the ground. A second roof suspended 12 to 18 inches above the first will trap a layer of air and decrease the temperature within the shelter. Metal vehicles will be like ovens, and it is better to sit on a seat cushion in the shade of an automobile or under the wing of an airplane than to be inside. Try to build the shelter in the shadow of a cactus, tree, or large rock. Avoid dry stream beds (arroyo, wadi, or dry wash) that may be swept with killer floods in a matter of minutes after a cloudburst miles away. Desert animals will seek out shelter during the day also and may venture into your shade. Reptiles may be venomous and mammals may carry diseases such as rabies or plague. Most of them can be scared off with a stick or rock.

3. One of the earliest decisions that you will have to make is whether to wait for rescue or to attempt to find your way back to civilization. You need to look at all the factors that may decide your survival. What is the chance that you will be rescued? If you filed an itinerary or flight plan, if you have signalling equipment, a radio or emergency locator transmitter, water, shelter, and food, then you probably should stay where you are. Your chances for rescue will be much better if you are near an object such as an airplane or automobile that can be seen by SAR personnel. If you do decide to travel, mark a large arrow on the ground in the direction of travel and leave a note stating your direction of travel and plans. Travel in the cool of the night to conserve water, although the footing may be more hazardous. Before travelling in the desert or anywhere, obtain an up-to-date

topographic map of area and learn how to use a compass. Memorize major physical and manmade features so that if you get lost without your map and compass, you will still be able to find a road, river, or powerline that may lead to help. Direction can be approximated by using the shadow tip method or a watch during the day or the stars at night (see illustration).

Signalling can best be accomplished during the day using a mirror and reflecting the sun off the mirror towards the horizon in the direction of potential help. If an aircraft is spotted, do not shine the mirror directly on the aircraft but rather alternate flashing the mirror from the ground to the aircraft. A signal such as this can be seen at 30,000 feet. At night a fire or penlight can be seen at great distances also if shone from a high point. A mirror cannot be used up but penlight batteries and firewood can, so wait until you hear the sound of an aircraft or rescuers before signalling by these methods.

Nature is neutral, neither for us or against us. The game of survival like all games of life is based on our control of nature to the extent that we need to survive. This includes the nature inside of us as well as outside of us. The first step in any survival situation is to gain control of ourselves, then try to control those things outside ourselves such as climate and terrain.

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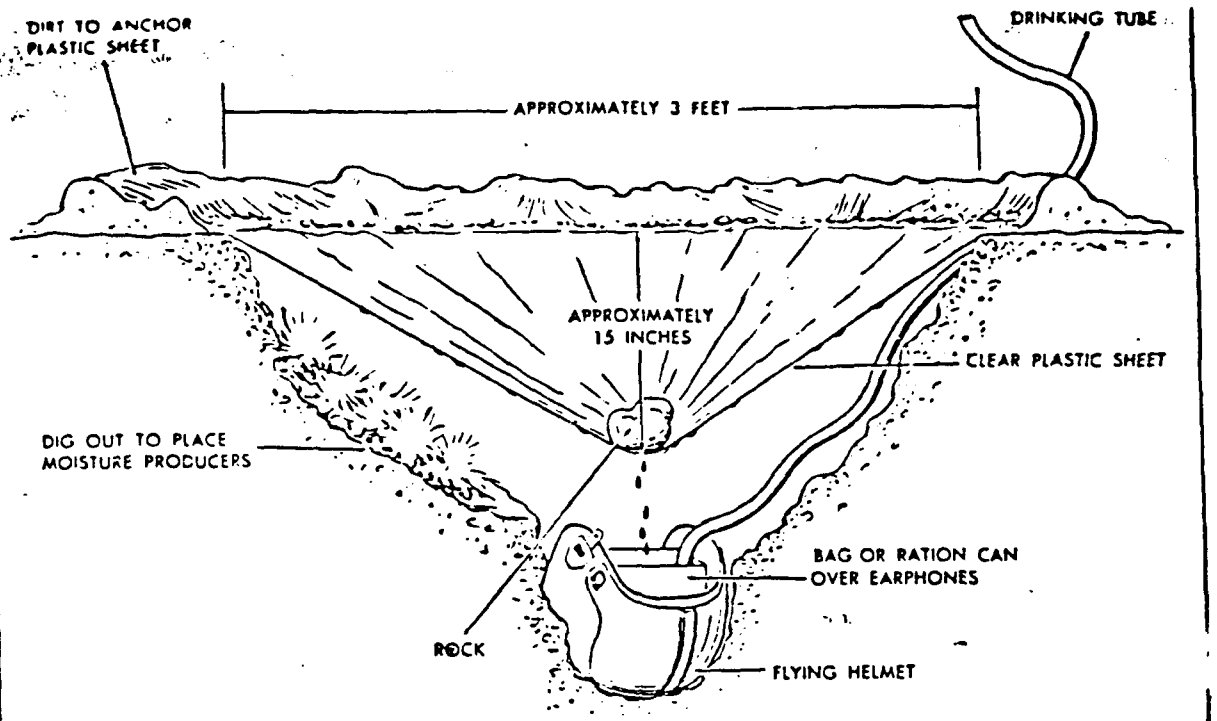
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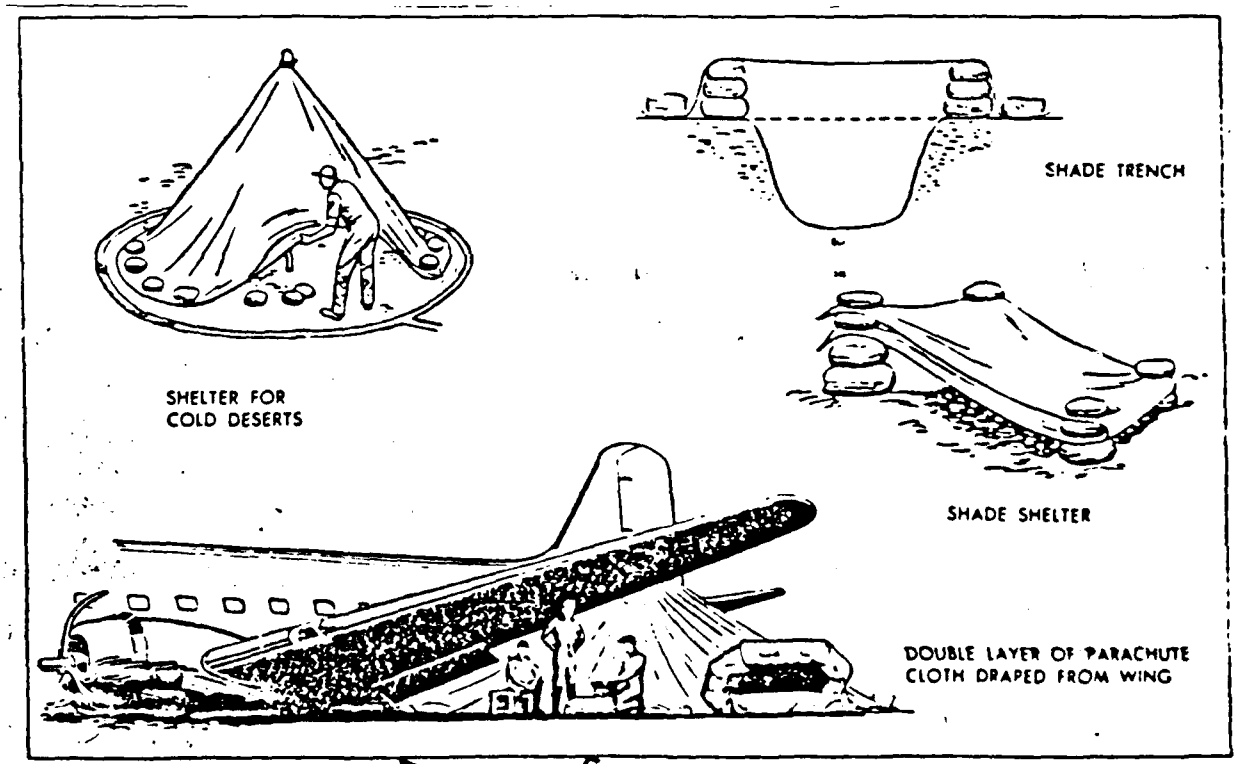
Dig a bowl-shaped hole in the soil about 40 inches in diameter and 20 inches deep. Add a smaller, deeper sump in the center bottom of the hole to accommodate the container. If polluted waters, such as body waste, are to be purified, a small trough can be dug around the side of the hole about half way down from the top. The trough insures that the soil wetted by the polluted water will be exposed to the sunlight and at the same time that the polluted water is prevented from running down around or into the container. If plant material is to be used, line the sides of the hole with pieces of the plant or its fleshy stems and leaves. Place the plastic film over the hole and put a little soil on its edges to hold it in place. Place a rock no larger than your fist in the center of the plastic and lower the plastic until it is about 15 inches below ground level. The plastic will now have the shape of a cone.

CAUTION

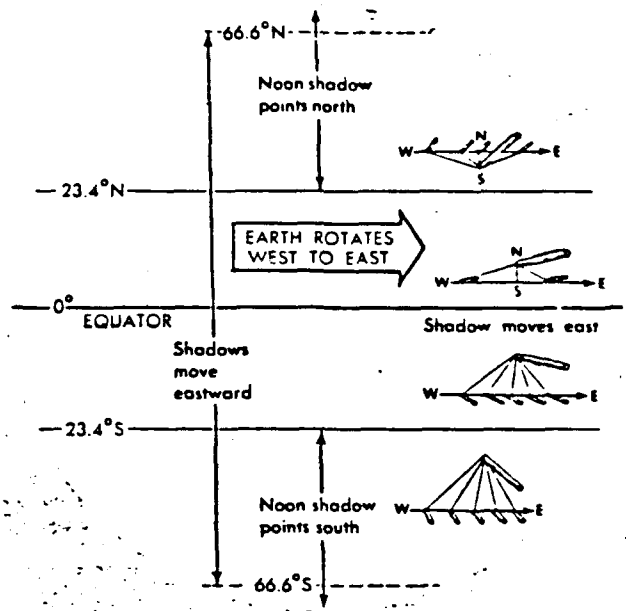
Make sure the plastic cone does not touch the earth anywhere causing loss of water.

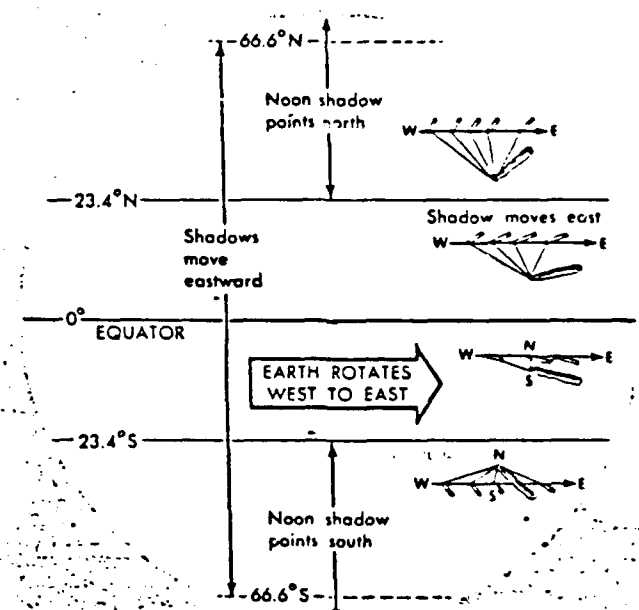
Put more soil on the plastic around the rim of the hole to hold the cone securely in place and to prevent water vapor losses. Straighten the plastic to form a neat cone with an angle of about 30 degrees so that the water drops will run down and fall into the container in the bottom of the hole. It takes about one hour for the air to become saturated and start condensing on the underside of the plastic cone.

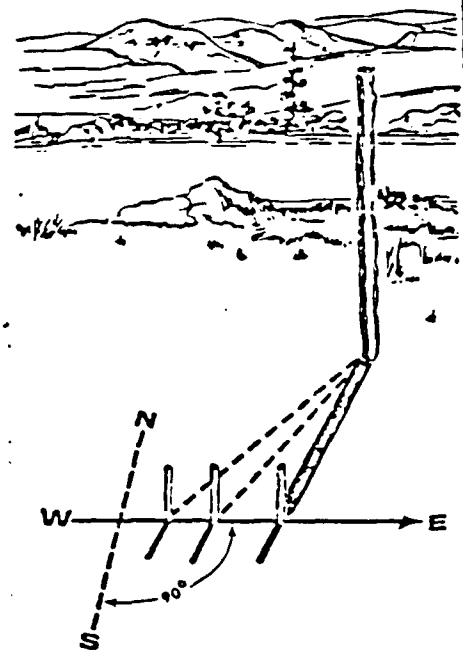
SOLAR STILL



DESERT SHELTERS







1. Put up a stick or rod as near to vertical as possible in a level place.
2. Mark the end of the shadow with small sticks or rocks allowing a short period of time between marks.
3. A line drawn at 90° to a line through the markers will be a north-south line.
4. The markers will progress toward the east during all seasons anywhere between the Arctic Circles (66.6°N to 66.6°S). In the Tropics (24.4°N-24.4°S), this indication of east direction is most useful because the noon shadow can be either north or south depending on the season. This determination of direction may be made anytime of the day.
5. The shortest shadow, which indicates local noon, will point north anywhere north of 24.4°N latitude and south anywhere south of 24.4°S latitude. The use of the NOON sun is necessary in the areas between the Arctic Circles and the poles.

Stick and Shadow Method to Find Direction

HUMAN COOLING RATES IN EXTREME COLD

John S. Hayward
Professor of Biology
Victoria, B.C., Canada

Learning Objectives:

Following this presentation, participants will be able to reasonably predict the actual cooling rates of humans in a variety of extremely cold situations. This knowledge will improve decision-making concerning: prevention of hypothermia; assessment of the seriousness of hypothermia in a wilderness situation; need for evacuation of a victim; choice of hypothermia-treatment protocol; and adequacy of existing search and rescue facilities with respect to hypothermia risk.

Outline of Course Material:

- The Wet-Cold situation
  - psychomotor consequences of peripheral cooling
  - exhaustion-dependent hypothermia
- Inadequacy of data on Dry-Cold situation.
- Cold water immersion
  - "average" cooling rates and survival times
  - sources of variation
    - swimming and postures
    - fatness
    - body size
    - age (children) and sex
    - special garments
    - rafts
- Other poorly-understood situations
  - trauma victims
  - alcohol and drug effects
  - starvation effect
  - interaction with hypoxia
- Future research needs

Cold Weather Survival

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Billings Clinic, Billings, MT, USA
National Medical Advisor, National Ski Patrol
Chairman, Medical Committee, National Association for Search & Rescue (NASAR)
Summer, 1991

Objectives: After attending this presentation, the student should be able to:

1. List the requirements for human survival
2. Discuss the voluntary and involuntary mechanisms that regulate body core temperature and how to use them to avoid dangerous degrees of cooling.
3. Discuss the requirements for cold weather clothing and give examples.
4. Understand the principles and techniques of the use of snow as insulation in constructing emergency snow shelters.
5. Describe the technique of fire building.
6. Discuss the principles of obtaining food and water in a cold weather survival situation.

For survival, the human body requires a constant supply of oxygen, a body temperature regulated between the relatively narrow limits of 75oF to 106oF (24oC to 41oC), an adequate supply of water and food, and a generous amount of self confidence, faith and the will to live. Physical conditioning and physical integrity (the absence of disease or illness) are also important. As a rough rule of thumb, man can survive less than 5 minutes without oxygen, perhaps 5 hours of severe weather without shelter, 5 days or so without water, and 5 weeks without food. Although the requirements for survival are listed (and discussed) separately, they are interrelated. Since most deaths in the outdoors in winter are due to injury, or hypothermia, or both, maintenance of body temperature and physical intregity (through accident prevention) are probably the most important requirements for cold weather survival. Dehydration, starvation, and exhaustion make temperature maintenance more difficult and interfere with the rational thought and agility required to prevent accidents. Hypoxia becomes a contributing factor at extreme altitude, or in the case of misfortunes such as suffocation due to avalanche burial or carbon monoxide poisoning due to cooking in an unventilated shelter. Abundant food and water will be of little value to the hypothermic dying from insufficient clothing and shelter. Lack of self-confidence, faith and the will to live will foster an attitude of panic and defeatism which tends to prevent the individual from taking timely survival actions such as preparing a shelter and lighting a fire. Poor physical condition or the presence of illness or

injury will interfere with heat production due to shivering; and hamper wood gathering, shelter building, and other physical activities needed to survive.

WARMTH AND SHELTER

The human body can be thought of as a heat generating and dissipating machine that must regulate its internal temperature closely for survival. This usually can be done successfully despite ambient temperatures which can vary more than 100oF (47oC) from the coldest to the hottest seasons of the year in temperate climates. Physiologic mechanisms that protect against excessive heat are better developed than those for protection against excessive cold. The most important organ for cold protection is probably the human brain, since voluntary actions such as donning clothing and seeking shelter are more important than involuntary actions.

Since death can occur if the human body core cools down to the temperature of a comfortable summer day, cold weather survival is an important topic for outdoor recreationists. Dangerous climactic conditions can exist in the arctic and the high mountains at all times of the year, and during the cooler months in temperate climates. It is of interest that more cases of hypothermia have been reported from moderately cold regions such as the British Isles and the State of Kentucky than from arctic regions, possibly because inhabitants of the latter areas are more experienced at protecting themselves.

Body core temperature can be influenced by both involuntary and voluntary processes. Resting heat production is the byproduct of basal biochemical reactions, amounting to 50-70 KCal./m2/h. It can be increased involuntarily by shivering, semiconscious activity such as foot stamping, and "nonshivering thermogenesis", which is probably mediated through increased secretion of thyroxin, epinephrine, and norepinephrine. Heat production can be increased voluntarily by muscular exercise, and to some extent by eating. Heat can be added from the outside by warm food and drink, and through a stove, fire or other heat source. Heat loss can be decreased involuntarily by decreasing sweating, shunting blood away from the skin, muscles and extremities; and by efforts to decrease body surface area as in curling up into a ball. It can be decreased voluntarily by putting on additional clothing and seeking shelter.

Since the most energy efficient method for avoiding dangerous body cooling is to decrease heat loss, it will be discussed in more detail. Heat loss and gain from the environment occurs through the five familiar physical mechanisms of conduction, convection, evaporation, radiation, and respiration. Heat loss through conduction and convection can be prevented by the intelligent use of insulation and wind-proof materials. Suitable materials fall into two general groups: woven fabrics and nonwoven fibers. Some nonwoven fibers, such as polyester pile, are incorporated into a fabric; others, such as down, are used as a filler to provide loft. Clothing should be worn in a number of thin layers so as to trap several layers of still air, which are warmed by body heat. The most effective

fabrics are those which trap air and prevent its motion, and whose effectiveness is not reduced by becoming wet. The best of these currently are wool, polypropylene, and such types of treated polyester as Capilene (R) and Thermax (R). Cotton has poor insulating value, especially when wet, and has no place in the cold outdoor environment. Orlon, polyester, acrylic, and similar synthetics are also good insulators. The above fabrics are typically worn as the innermost 2 or 3 layers---long underwear, shirt, sweater, and pants. Outer layers are normally chosen from fibers which produce loft, such as polyester and nylon pile and fleece, down, Dacron (R), Hollofil II (R), Quallofil (R), Thermoloft (R), Thinsulate (R) and Thermolite (R). These are made into jackets, vests, and overpants. The outermost layer is preferably of a windproof and water resistant fabric such as nylon/cotton blends, Gore-Tex (R), or similar material.

Heat loss by conduction is avoided by sitting on a log, foam pad or other poorly-conducting material rather than in the snow or on a cold rock, and by not touching metal or other good heat conductors with unprotected skin at low temperatures.

Heat loss by evaporation is avoided by wearing water-repellant outer garments to avoid wetting, by drying off quickly or changing to dry clothes when wet, and by removing layers when too warm, in order to avoid excessive perspiring. Heat loss by radiation is avoided principally by wearing a hat, since circulation to the head is not reduced in the cold, and up to 70% of body heat production, for example, can be lost at 5oF (-15oC) through an uncovered head. Special attention should be given to the protection of body parts with a large surface- area to volume ratio, such as the nose, ears, toes, fingers, and extremities in general. Mittens should be worn in preference to gloves. Tight boots and other garments which restrict circulation should be avoided. In cold, windy weather, a face mask, balaclava, or combination of a ski cap plus a "neck gaiter" should be worn in order to protect the face. In extreme conditions you should give up and seek shelter. Wind chill is very important; the adverse effects of wind when combined with low temperature must be experienced to be fully appreciated. Wind chill charts show the relationship between actual temperature, wind velocity, and "effective" temperature at the body surface. Heat loss is not linear, but is more proportional to the square root of the wind speed. The wind chill concept refers to the rate of cooling rather than the actual temperature reached.

Anyone who ventures out of doors in cold weather should carry clothing, either on the body or in the pack, for the most extreme environmental conditions which are likely to be experienced.

The ability to improvize a wind and cold-proof survival shelter is an essential survival skill. If the snow cover is adequate this should be constructed of snow, which is a very good insulator with heat conductivity 1/10,000 that of copper and about the same as wool felt. Every cold weather survival kit should contain a collapsable snow shovel of the small grain-scoop type, and a plastic or nylon tarp measuring about 8 by 10 feet. The shovel is used to dig a rectangular trench 3 to 4 feet deep, one end of which is roofed over with the tarp. A fire is built

in the other end.

The ability to build a fire under adverse conditions is another important survival skill which needs to be practiced. A cold weather survival kit should contain waterproof matches, a fire starter or candle, and a collapsable saw. The fire should be laid out of the wind--one end of a survival snow shelter is a good place. Tender is laid against a large, dead branch in lean-to fashion, with small, dry twigs such as those found on the dead lower branches of evergreens on the bottom and larger ones on top. All the tender is arranged so that air can reach each piece. A candle or fire starter is necessary if the wood is damp; if wet, the wood should be shaved or split as well. The amount of wood needed to last all night is easy to underestimate, and is two or three times more than you think.

FOOD

Although most persons in a survival situation worry more about food thasn anything else, food is usually less important than water or shelter. The body can last many weeks without food even in cold weather if water is available and exertion minimized. The amount of wild food found by an untrained individual will rarely provide enough calories to replenish the energy expended in searching for it. Bare ridges, high mountains above timberline, and dense evergreen forests are difficult places to find food, especially in winter. Success is more likely along river and stream banks, lake shores, margins of forests, and natural clearings.

WATER

Because of the fuel required to melt snow or ice for water, advantage should be taken of open water when found. It should be treated by filtration, chemical disinfection, or boiling if there is a question about its purity. Simply bringing water to a boil is probably effective at any altitude where contamination---other than human---is a problem. [The boiling point of water at 18,000 feet (5500 meters) is about 179oF (81.6oC)]. Small streams and springs coming down from high, uninhabited areas at right angles to the main valley drainage pattern and streams coming from untracked snowfields are more apt to be safer to drink untreated than larger streams running parallel to the long axis of a valley. When water supplies are limited, avoid overexertion with sweating (Ration your sweat, not your water!).

ADDITIONAL SURVIVAL CONSIDERATIONS

1. Keep in good physical condition with regular exercise.

2. Learn to swim well.

3. Become expert in the use of map and compass, and be able to find directions without a compass.

4. Know weather patterns in the area to be travelled.

5. Be familiar with wilderness and cold weather medicine, especially with the management of wilderness injuries, hypothermia, frostbite, and acute mountain sickness.

6. Carry a personal cold-weather survival kit (see below).

7. Persons stranded in cars or downed airplanes can survive using the equipment found in the vehicle. It is best to stay with or near the vehicle rather than try to go for help, since the vehicle is more visible to searchers than you are. Cars can be quite comfortable since they are upholstered, windproof and waterproof, but in very cold weather you may do better to build a shelter adjacent to the car so that a fire can be built. Since the fusilages of downed aircraft can be very cold, you are usually better off outside but near the aircraft. Batteries and cigarette lighters can be used as firestarters; oil and gasoline as fuel.

8. Have a working acquaintance with natural hazards which commonly create a survival situation, and how to predict and avoid them. These include such things as lightning strikes, snow avalanches, rockfall, cornice falls, flash floods, deadfalls, severe storms, undercut snowbanks along streams, hazardous animals and plants, etc.

9. Read and analyze accounts of actual survival experiences, noting particularly the ingenuous ways in which the victim handled the unexpected.

10. Never travel in the backcountry alone. Let someone know where you are going and when you will be back.

COLD WEATHER SURVIVAL KIT

Shelter Building Equipment:

Plastic or nylon tarp	Snow shovel
Nylon cord, 1/8", 50 feet	Folding saw

Fire Building Equipment:

Waterproof matches	Firestarter
Candle	Knife

Signalling Equipment:

Whistle	Signal mirror
Card with Ground-to-air Signals	Flashlight
Two quarters for phone	

Other:

Compass	Map
Metal Pot with Bale	First Aid Kit

Toilet Paper
Sunburn Cream
Spare Mittens and Socks
One Extra Layer: Parka and Pile Pants

Sunglasses
Sunburn Cream
Canteen (full)

Optional:

Piece of Ensolite or Thermorest
Stove and Fuel

Sleeping Bag
Small Ax (Hudson's Bay type)

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TECHNICAL NOTE

Insidious Hypothermia During Raft Use

JONATHAN W. KAUFMAN, M.S., B.Ch.E., and JAMES P. BAGIAN, M.D., B.S.M.E.

KAUFMAN, JW, BAGIAN JP. *Insidious hypothermia during raft use.* Aviat. Space Environ. Med. 1990; 61:569–75.

It is commonly thought that increasing protection against cold water results in greater physiological efficiency in maintaining core temperature. To examine the relationship of physiological changes resulting from cold exposures as a function of increased insulation, five subjects (4 males, 1 female) wearing anti-exposure ensembles covering the entire body and head were exposed in a pool to water temperatures (T_{water}) = 4.4°C and air temperatures (T_{air}) = 5.6°C. Trials consisted of subjects undergoing either head-out immersions with mean exposure time (t = 150 ± 9 S.E.M. min) or enclosed within a raft (t = 398 ± 126 min). Rectal temperatures were higher, and their relative change from baseline (ΔT_{re}) smaller, for 3 of the 5 subjects at minute 122 in the water than in the raft. While no correlation was found between mean weighted skin temperature (T_{sk}), hand temperature (T_{hand}), or foot temperature (T_{foot}) with T_{re}, close correlation was found between ΔT_{re} and forehead temperature (T_{fore}) ($r = 0.97$, $p < 0.05$) and change in forehead temperature ($r = 0.97$, $p < 0.05$). The results suggest that, during cold exposure, increased insulation, under specific conditions, may result in a lower T_{re}.

IT IS GENERALLY assumed that increasing insulation for persons exposed to cold will help them maintain core temperature. This in turn will translate into their ability to withstand cold exposure longer. Field reports (11) and laboratory studies (5,12,19) of insidious hypothermia have, however, raised questions regarding the impact of total body insulation in relatively warm water of 29°C. A recent evaluation of antiexposure protection produced evidence of insidious hypothermia occurring in people on rafts during exposure to 4.4°C water (1). What follows is an examination of the data from that study and its implications.

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This manuscript was received for review in June 1989. The revised manuscript was accepted for publication in November 1989.

Send reprint requests to Jonathan W. Kaufman, Naval Air Development Center, Environmental Effects Branch, Code 6023, Warminster, PA 18974-5000.

MATERIALS AND METHODS

Subjects: Four healthy males and one female (Table I) volunteered as subjects after being fully informed of the experimental protocol and associated risks (1).

Weight was recorded prior to each test run and the mean was calculated. Body surface area (SA) was calculated from the mean weight and height of each subject, and percent body fat was determined from estimates of body density (1).

Materials: The clothing ensemble used in this study was the NASA Launch Entry Suit (LES) (Table II) (1). The LES consists of a laminated PTFE membrane shell, which allows the passage of water vapor but not liquid, coupled with pressure bladders and controllers designed to provide protection against loss of cabin pressure. Integrated flight gloves were designed for use in low pressure environments, but provided minimal thermal protection. A number of survival mittens were evaluated in this study, with a neoprene/PTFE type ultimately selected.

The raft used was a variant of the U.S. Navy LRU-18/U one-man raft, modified by the inclusion of a canopy. The canopy consisted of a waterproof fabric cover running the length of the raft and designed to come over the head with a drawstring for sealing the face. To permit raft entry, the canopy was split, from the head to the foot of the raft, into two flaps of material. These flaps were designed to be secured along the raft mid-line by means of a two inch wide Velcro strip. After the first raft trial, subjects were instructed to sit on the water packets supplied as part of the LES survival equipment in an attempt to increase the insulation between themselves and the raft bottom.

The raft bailing system was a small wedge-shaped scoop with a relatively square opening, and fitted with a strap designed to slide over a survival mitten. To accomplish nearly complete bailing of a raft, a small hand pump was also provided which consisted of a squeeze bulb connected to pieces of tubing. The addition of the hand pump facilitated bailing when the raft canopy was closed and allowed for more complete removal of water from the raft while retaining trapped metabolic heat under the canopy.

TABLE I. PHYSICAL CHARACTERISTICS OF SUBJECTS.

Subject	Age (yrs)	Height (m)	Weight (kg)	% Body Fat	Surface Area (m^2)
A	36	1.82	75.4	9.3	1.96
B	44	1.73	71.5	12.6	1.85
C	31	1.83	87.4	14.4	2.10
D	34	1.88	90.3	18.9	2.17
E	32	1.65	69.8	29.5	1.77

TABLE II. CLOTHING WORN AS PART OF THE LAUNCH ENTRY SUIT (LES) CONFIGURATION WORN DURING TESTS.

a. Parachute harness
b. Life vest
c. Parachute pack
d. Life raft pack
e. LES helmet
f. LES gloves
g. Various survival mittens
h. Capilene underwear
i. Polypropylene sox
j. Urine collection device
k. Flyer's boots
l. Flotation device

Methods: Subjects employed each configuration once. The minimum time interval between tests for a given subject was 2 d in order to minimize acclimatization effects.

Each subject reported to the laboratory on the morning of a test and was given a physical examination by the attending flight surgeon. A urine specimen was collected and a urinalysis performed. Each subject's baseline weight was obtained, and ECG electrodes were placed on the subject. Heat flux/temperature transducers were attached to the following 10 body sites: (A) forehead; (B) left anterior upper chest; (C) left distal upper arm; (D) dorsum of left hand; (E) right anterior thigh; (F) left posterior thigh; (G) right shin; (H) right foot; (J) right proximal upper arm; and (K) left lower back. These transducers consisted of a thermopile heat flux transducer with a thermistor located in the center. A rectal thermocouple was inserted at least 8–10 cm anterior to the anal sphincter. A detailed description of the instrumentation is available elsewhere (1).

Subjects dressed and type T thermocouples were placed on external LES surface sites corresponding to the skin surface sites upon which the heat flux/thermistors transducers were placed. Thermocouple outputs, including the rectal probe, were measured with isolated signal conditioners (Ben-Dec, Santa Ana, CA, model TC.4). Upon completion of dressing, the subject was weighed, followed by a rest period of 20 min which enabled his temperature and heart rate to return to a resting condition before commencing that day's trial. The subject was cooled with a ventilator connected to the LES during this 20-min rest period. Laboratory temperature was maintained at approximately 20°C to minimize thermal stress during dressing.

Following the rest period, subjects entered the pool. Testing was performed in chamber conditions of water temperature (T_{water}) = 4.4 ± 0.2°C and air temperature (T_{air}) = 5.6 ± 0.1°C, with wind velocities of 6.7–11.7 km/h, overhead spray, and approximately 1-ft, choppy waves. Runs consisted of either immersion for the entire exposure period or a relatively short immersion followed by entry into a raft. Trials using only a personal flotation device consisted of subjects entering the water and attempting to remain immersed for up to 6 h.

Raft tests required the subject to remain in the water for 2 min (during a trial in which the raft sank, the subject was initially in the water for 10 min), after which he or she was handed a raft with its primary air chambers inflated. The subject then boarded the raft and inflated the secondary air chambers. Bailing the raft was then initiated, first by use of the canopy, which served to remove large quantities of water quickly, then by means of hand-held bailers; this activity was continued until the subject decided sufficient water had been removed to justify closing the raft canopy, after which bailing was by means of the small hand pump. Type T thermocouples were then passed through the raft opening located at the feet (which was used for all leads) to measure changes in the air and water temperatures within the raft. Subjects were instructed to remain in the raft for 24 h or until the trial was terminated.

Runs were terminated early due to a rectal temperature (T_{re}) = 35°C; hand temperature (T_{hand}) = 10°C ; foot temperature (T_{foot}) = 4.4°C (i.e., equal to T_{water}); heart rate (HR) exceeding 90% of the maximum predicted for age; or if the subject, flight surgeon, or principal investigator requested termination. Potable water was available to subjects in packets carried in the LES, but no food was provided to subjects.

Mean weighted skin temperature (T_{sk}) was calculated using the equation:

$$T_{sk} = 0.1(T_A) + 0.125(T_B + T_K) + 0.07(T_J + T_C) + 0.06(T_D) + 0.125(T_E) + 0.15(T_G) + 0.125(T_E + T_F)/2 + 0.05(T_H) \quad \text{(Eq. 1)}$$

where T_i are the measured skin temperatures at locations i = A–K (16). Unweighted mean skin temperature ($T_{sk,\,un}$) was also calculated by summing each of the individual sites, with the S.E.M. intended to act as a measure of skin temperature uniformity. Mean weighted skin surface heat flux (HF) was calculated from the equation:

$$HF = 0.1(HF_A) + 0.125(HF_B + HF_K) + 0.07(HF_J + HF_C) + 0.06(HF_D) + 0.125(HF_E) + 0.15(HF_G) + 0.125(HF_E + HF_F)/2 + 0.05(HF_H) \quad \text{(Eq. 2)}$$

where HF_i are the measured heat fluxes at locations i = A–K (14,15,16). Cumulative energy losses from the body were calculated in Joules by:

$$\Sigma Q = \Sigma(HF \times SA) \quad \text{(Eq. 3)}$$

where ΣQ is the total heat energy and SA is the body surface area.

Statistical Analysis: All data, except exposure duration, were analyzed at minute 122, as this was the maximum time for which all subjects endured exposures in both configurations. Correlation between physiological indices was analyzed using the nonparametric Spear-

man rank correlation test. Significance was determined at a probability level of 0.05. Comparison of populations (i.e. with or without evidence of insidious hypothermia) were not analyzed for statistical differences due to the very small sample sizes (i.e. n = 3 and n = 2, respectively). Mean values are given without S.E.M. for the same reason when comparing these populations.

RESULTS

Three of the five subjects (A, B, and D) had higher Tre's and smaller ΔTre's in the immersion trials than in the raft trials after 122 min had elapsed (Table III). This difference was established after approximately 20 min into the runs for subjects A, B, and D and remained until termination, while subject E did not achieve a higher Tre in the raft than in the immersion trials until after approximately 110 min had elapsed (Fig. 1, 2). Mean Tre's (and ΔTre's) for the immersion and raft runs for subjects A, B, and D, i.e. those subjects who demonstrated insidious hypothermia (I), were respectively 36.9°C(−0.4°C) and 35.8°C (−1.6°C). This compares with means for subjects C and E, i.e. the subjects who did not produce evidence of insidious hypothermia (NI), of 36.1°C (−1.1°C) and 36.9°C (−0.8°C) for the immersion and raft trials. Mean immersion Tre minus raft Tre was positive for I (1.0°C) while negative for NI (−0.7°C) (Table III). An inverse relationship was observed for the mean immersion ΔTre minus raft ΔTre, which was negative for I (−1.2°C) and positive for NI (0.4°C) (Table III). These differences among subjects did not correlate with either subject weight or percent body fat.

Mean exposure duration for the immersion runs was 150 min while the raft runs were 398 min (Table IV). The wide disparity in exposure duration observed with the raft trials did not correlate with which subjects displayed higher or lower Tre's in the raft vs. immersion trials measured at 122 min. Subject B endured the longest (801 min) followed by subject E (587 min). All immersion trials were terminated due to subject discomfort, with a mean final Tre = 36.5°C. The raft runs for subjects C and D were terminated when Tre reached 35.0°C, subject A reached a Tre of 35.1°C before equipment problems caused termination of the run, and the runs of subjects B and E, the two longest runs of the study, were terminated because of subject discomfort.

TABLE III. RECTAL TEMPERATURE (Tre) BY SUBJECT, ALONG WITH THE MEAN ($\bar{X}$) AND STANDARD ERROR OF THE MEAN (S.E.M.), MEASURED AT 122 MIN DURING IMMERSION AND RAFT TRIALS. THE CHANGE IN Tre (ΔTre) IS CALCULATED FROM VALUE AT 122 MIN MINUS INITIAL VALUE

	Tre (°C)		ΔTre (°C)	
Subject	imm.	raft	imm.	raft
A	36.8	36.3	−0.5	−1.3
B	37.0	35.3	−0.1	−1.9
C	35.4	36.1	−1.5	−1.1
D	36.8	35.9	−0.7	−1.6
E	37.3	37.6	−0.7	−0.4
$\bar{X}$	36.7	36.2	−0.7	−1.3
S.E.M.	0.3	0.4	0.2	0.3

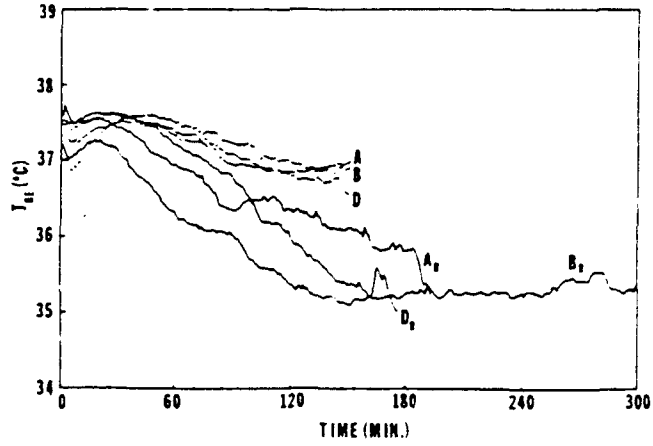


Fig. 1. Rectal temperature (T_{re}) vs. time for subjects A, B, and D. A higher T_{re} was observed for these subjects in immersion trials compared with raft trials. The subscript R denotes raft trials.

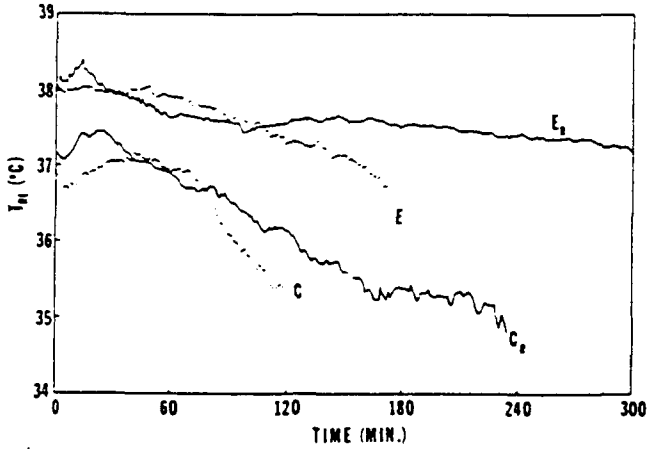


Fig. 2. Rectal temperature (T_{re}) for subjects C and E. A lower T_{re} was observed for these subjects in immersion trials compared with raft trials. The subscript R denotes raft trials.

No correlation with either Tre or ΔTre was observed for Tsk, ΔTsk; Tsk,un, or S.E.M. of Tsk,un in either immersion or raft runs (Fig. 3, 4, Table IV). This lack of correlation indicates that neither Tsk nor the skin temperature gradient measured by S.E.M. of Tsk,un are related to the insidious hypothermia observed in this study. Other temperature gradients were examined to determine if they might contribute to the observed phenomena. No correlation between the skin/garment temperature gradient (Tsk–Tsuit) and either Tre or ΔTre was observed (Table V). This was also true for the change in the (Tsk–Tsuit) gradient, obtained by subtracting the value at minute 122 from the initial value (Table V). The core/skin temperature gradient (Tre–Tsk) and the observed gradient change also displayed no correlation with either Tre or ΔTre (Table V). ΣQ, which is related to the garment/environment temperature gradient, was also found to be uncorrelated with either Tre or ΔTre (Table V).

Extremity temperatures were also examined for correlation with Tre or ΔTre, as it had been suggested that these may be the driving mechanism for thermoregulatory responses to cold (5,19). No correlations were

TABLE IV. EXPOSURE DURATION, MEAN WEIGHTED SKIN TEMPERATURE (T_{sk}), UNWEIGHTED MEAN SKIN TEMPERATURE ($T_{sk,un}$), AND THE STANDARD ERROR OF THE MEAN OF $T_{sk,un}$ (S.E.M., $T_{sk,un}$) FOR IMMERSION AND RAFT TRIALS. THE CHANGE IN T_{sk} (ΔT_{sk}) IS CALCULATED FROM VALUE AT 122 MIN MINUS INITIAL VALUE. VALUES ARE GIVEN AS OVERALL MEAN (X_{all}) ($N = 5$), STANDARD ERROR OF THE MEAN (S.E.M.), MEAN FOR SUBJECTS A, B, AND D (X_{IS}) ($N = 3$), AND MEAN FOR SUBJECTS C AND E (X_{NI}) ($N = 2$).

	Exposure Duration (min)		T_{sk} (°C)		ΔT_{sk} (°C)		$T_{sk,un}$ (°C)		SEM, $T_{sk,un}$ (°C)	
	imm.	raft	imm.	raft	imm.	raft	imm.	raft	imm	raft
X_{all}	150	398	22.7	29.0	−10.0	−4.2	21.1	28.0	2.0	1.5
S.E.M.	9	126	0.9	0.4	2.0	1.9	0.4	0.5	0.2	0.2
X_I	151	390	23.4	28.7	−9.2	−4.4	21.3	27.8	2.1	1.5
X_{NI}	150	410	21.6	29.6	−11.3	−4.0	20.9	28.2	1.7	1.5

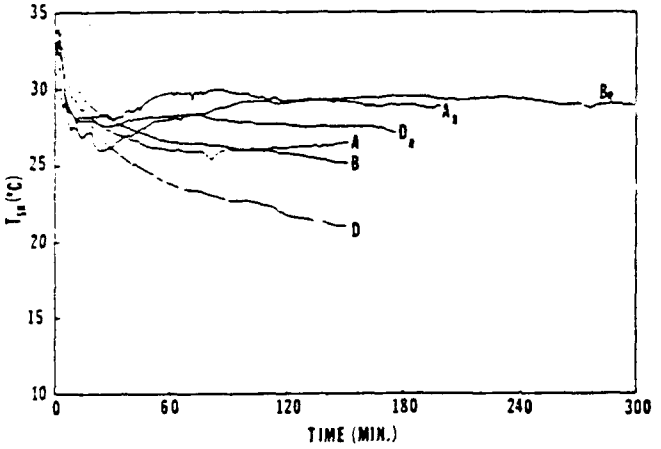


Fig. 3. Mean weighted skin temperature (T_{sk}) time for subjects A, B, and D. A higher T_{re} was observed for these subjects in immersion trials compared with raft trials. The subscript R denotes raft trials.

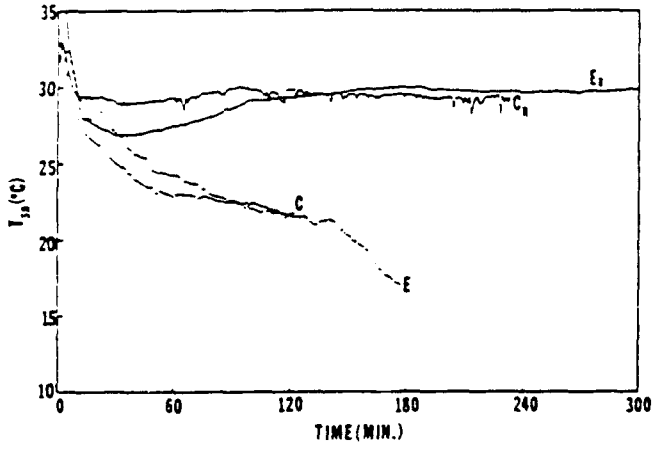


Fig. 4. Mean weighted skin temperature (T_{sk}) vs time for subjects C and E. A lower T_{re} was observed for these subjects in immersion trials compared with raft trials. The subscript R denotes raft trials.

found for Thand, ΔThand, or ΔTfoot in either immersion or raft trials, though Tfoot was correlated to ΔTre in the immersion runs ($r = 0.97$, $p < 0.05$) (Table VI).

While the existing literature does not suggest a role for forehead temperature (Tfore) in insidious hypothermia, correlations with ΔTre in both the immersion and raft runs were observed for both Tfore (respectively, $r = 0.97$, $p < 0.05$ and $r = 0.9$, $p < 0.05$) and ΔTfore (respectively, $r = 0.97$, $p < 0.05$ and $r = 0.9$, $p < 0.05$) (Table V). In a pattern duplicating that for Tre and ΔTre, the mean immersion Tfore minus raft Tfore was positive for I (2.0°C) and negative for NI (−7.3°C), i.e. Tfore at 122 min was greater during immersion than raft runs for I and lower for NI. An inverse relationship was observed for the mean immersion ΔTfore minus raft ΔTfore, which was negative for I (−3.9°) and positive for NI (+12.2°C), indicating a greater change in Tfore occurred during raft runs for I and immersion runs for NI.

DISCUSSION

The observations described have shown for the first time the existence of insidious hypothermia in a nondiving environment and in a colder ambient environment than studied previously. A lower Tre observed in a raft than in an open water exposure over a comparable time for I suggests that these subjects experienced insidious hypothermia while in the raft. It is striking that in the severe test environment used in this study, the enhanced insulation provided by the raft could elicit such a response. Previous cases of accidental or experimentally induced insidious hypothermia have been at water temperatures above 29.0°C, compared with the 4.4°C water of this study. Therefore, the question which should be addressed is: by what mechanism might the raft induce insidious hypothermia and does this represent a threat to survival?

It has been postulated that the mechanism of insidious hypothermia involves a mild temperature gradient across which heat is lost from the body without eliciting afferent cold receptor responses (5,11,19). A lack of afferent cold receptor signals to the core would be responsible for minimal metabolic response to the net loss of body heat (5,12,19). The lack of a correlation in this study between ΣQ, Tsk − Tsuit, and Tre − Tsk with Tre or ΔTre, and thus no apparent relationship to the observed insidious hypothermia, points to metabolic inhibition as the mechanism by which insidious hypothermia occurs. If decreased cutaneous vasoconstriction were responsible, one would expect distinctive changes in the thermal gradients and in heat losses. Coleshaw *et*

TABLE V. TEMPERATURE GRADIENTS, I.E. TEMPERATURE GRADIENT ACROSS SUIT ($T_{sk} - T_{suit}$) AND BODY TEMPERATURE GRADIENT ($T_{re} - T_{sk}$), ALONG WITH SUMMED TOTAL HEAT LOSSES (EXCLUDING RESPIRATORY HEAT LOSSES) (ΣQ) MEASURED AT 122 MIN DURING THE IMMERSION AND RAFT TRIALS. THE CHANGES IN TEMPERATURE AND GRADIENTS ARE CALCULATED FROM VALUE AT 122 MIN MINUS INITIAL VALUE. VALUES ARE GIVEN AS OVERALL MEAN (X_{all}) (N = 5), STANDARD ERROR OF THE MEAN (S.E.M.), MEAN FOR SUBJECTS A, B, AND D (X_I) (N = 3), AND MEAN FOR SUBJECTS C AND E (X_{NI}) (N = 2).

	$T_{sk} - T_{suit}$ (°C)		$\Delta(T_{sk} - T_{suit})$ (°C)		$T_{re} - T_{sk}$ (°C)		$D(T_{re} - T_{sk})$ (°C)		ΣQ (kJ)	
	imm.	raft	imm.	raft	imm.	raft	imm.	raft	imm.	raft
X_{all}	16.4	13.8	[illegible]	−2.9	14.0	7.2	9.3	3.0	−155	−125
S.E.M.	1.4	0.7	[illegible]	1.1	0.9	0.5	0.9	1.0	32	26
X_I	17.7	14.5	1.5	−1.7	13.4	7.2	8.8	2.8	−136	−114
X_{NI}	14.6	12.8	−0.7	−4.7	14.8	7.2	10.2	3	−185	−143

TABLE VI. EXTREMITY TEMPERATURES, I.E. HAND TEMPERATURE (T_{hand}), FOOT TEMPERATURE (T_{foot}), AND FOREHEAD TEMPERATURE (T_{fore}), MEASURED AT 122 MIN DURING THE IMMERSION AND RAFT TRIALS. THE CHANGES IN TEMPERATURES ARE CALCULATED FROM VALUE AT 122 MIN MINUS INITIAL VALUE. VALUES ARE GIVEN AS OVERALL MEAN (X_{all}) (N = 5), STANDARD ERROR OF THE MEAN (S.E.M.) MEAN FOR SUBJECTS A, B, AND D (X_I) (N = 3), AND MEAN FOR SUBJECTS C AND E (X_{NI}) (N = 2).

	T_{hand} (°C)		ΔT_{hand} (°C)		T_{foot} (°C)		ΔT_{foot} (°C)		T_{fore} (°C)		ΔT_{fore} (°C)	
	imm.	raft	imm.	raft	imm.	raft	imm.	raft	imm.	raft	imm.	raft
X_{all}	19.0	26.1	−14.5	−8.1	18.6	27.6	−13.5	−6.3	25.2	27.7	−7.1	−6.2
S.E.M.	1.5	1.0	1.2	0.9	1.8	2.9	1.0	1.7	2.2	2.2	2.0	2.3
X_I	17.2	25.4	−15.5	−8.5	16.5	28.8	−14.0	−4.9	26.9	24.9	−5.1	−9.0
X_{NI}	21.8	27.1	−13.1	−7.4	21.9	25.8	−12.8	−8.5	22.7	32.0	−10.2	−2.0

al. (5) and van Someren *et al.* (19) demonstrated that local cooling of the hands and feet could elicit metabolic heat production in individuals otherwise experiencing insidious hypothermia. Keatinge *et al.* (12) demonstrated a similar metabolic response by the fluctuation of skin temperatures during insidious hypothermia.

Keatinge *et al.* (12) and van Someren *et al.* (19) have suggested that uniformity of surface temperatures may be the cause of inadequate metabolic responses to excessive body heat losses. It was postulated (12,19) that the spatial temperature gradients existing between the extremities and torso are necessary for maintaining thermoregulatory responses in relatively mild temperatures. The results of this study did not support this theory, since no correlation was found. This was clearly demonstrated by two of the subjects (A and B) exhibiting evidence of insidious hypothermia, and one who did not (C) having increased skin temperature uniformity of relatively equal magnitude in the raft compared with immersion.

Similarly, it was expected that some relationship between Thand, Tfoot, or Tsk with Tre would be found among subjects experiencing insidious hypothermia (12,19). It was with some surprise, therefore, that such a relationship was not found for the subjects in this study. The results for Tsk, Thand, and Tfoot do not agree with the previously stated hypothesis regarding the driving mechanism for core temperature preservation. It would be expected that I would have higher Tsk, Thand, and/or Tfoot in the raft trials compared to NI. This would be consistent with the postulated mechanisms involved in suppressing metabolic responses to cold (5,11,12,19). The results of this study do not correspond to these models, as no discernible relationship between elevated Tsk, Thand, or Tfoot and subjects experiencing insidious hypothermia was observed.

The difference among subjects might also be explained by NI having relatively higher Tsk, Thand, Tfoot in the immersion than the raft runs. While Tfoot and Thand for NI were indeed the highest among all subjects in the immersion trials, the relative difference when compared with raft data was not distinguishable from I. Further, ΔTfoot and ΔThand were no greater for NI than for I. This suggests that there may be a threshold value for Tfoot and/or Thand at which metabolic responses are triggered. Such a threshold (i.e. 20°C) has been found for some low-temperature cold receptors (17), which closely corresponds to the Tfoot and Thand for NI during immersion. It may be that the lack of evidence of insidious hypothermia for NI is the result of inhibited thermoregulatory responses in the immersion runs.

Benzinger (2) and Davis (7) have suggested that metabolic output is a function of both peripheral and central thermal reception. According to Benzinger (2), increased metabolic responses should be expected when core temperatures go below 36.9°C when a steady state Tsk = 29°C exists. This did not occur in this study, despite Tre's reaching as low as 35°C. However, this may ultimately explain why subject B reached a stable Tre = 35.1°C at 145 min and then gradually increased his Tre over the subsequent 656 min.

The observed correlation between Tfore and Tre sup-

ports the previously discussed theory that insidious hypothermia is related to an impairment of the metabolic response to lowered body temperatures. Because of the large quantity of heat lost from the head in cold conditions (8), it should not be surprising that Tfore is correlated to Tre. If this were representative of greater overall heat losses across the skin surface, a correlation of Tsk and Tre would be expected. Since this was not observed, greater overall skin surface heat losses do not appear responsible for the observed insidious hypothermia, and a lower Tfore probably represents a lower metabolic rate.

One must also consider a possible role for Tfore in the control of metabolic response to cold in a manner similar to that proposed for the feet and hands by Keatinge *et al.* (12) and van Someren *et al.* (18). Assuming that the hypothalamic region of the brain is the major central thermosensitive structure, then hypothalamic temperature (Thy) should relate closely to the control of thermal homeostasis (3). This will be true even assuming integrated control of thermal homeostasis, with other afferent inputs originating from CNS sites (i.e. spinal cord and medulla) and cutaneous receptors (3). Tfore might then have some influence on Thy on the basis of localized cooling proposed by Cabanac (4). For this to be true, however, one would expect that the relatively higher Tfore observed in the NS runs would result in maintaining Thy. Increased metabolic output elicited by cutaneous receptor afferent signals therefore would be attenuated, as suggested by the null zero theory of Bligh (3), resulting in a lower Tre. This was not observed, thus supporting the findings of Shiraki *et al.* (18), who found a lack of evidence for facial cooling affecting central thermoregulation.

In this study, no relationship between insidious hypothermia and either body weight or percent body fat was observed. One might expect this since Hayward and Keatinge (9) observed that metabolic responses, and thus ultimate duration in the environment, could not be predicted by the body fat of a subject in 10°C water.

That insidious hypothermia could occur while wearing equipment designed to enhance survival is disconcerting. Though the experiences of the North Sea diving community should have suggested such a possibility (11), previous cases of insidious hypothermia were observed in much warmer conditions (5,11,12,19) than experienced in this study. Do these findings suggest a potential unexpected hazard with use of a raft for long periods? Perhaps, but a number of factors must be examined before reaching such a conclusion.

First, this study employed a garment which totally enclosed an individual, including the entire head and neck. Sensory inputs from the face, head, and neck were thus minimized. Most working environments do not employ such garments, with the result that some portion of the face, head, and neck would be exposed to the environment. Second, there is insufficient understanding of insidious hypothermia to predict its ultimate consequences. While insidious hypothermia has been demonstrated to result in diminished mental performance (6) and has been implicated in diving deaths in the North Sea, Keatinge (13) has suggested that a lower limit to cooling may exist beyond which the body responds. Finally, the use of a raft provides survivors with protection against drowning and a stable platform for deploying survival aids. Fatigue would increase the difficulties involved in being rescued as the time in a survival situation increases, thus making a raft increasingly valuable and desirable.

It is obvious that further study of the phenomena of insidious hypothermia is required in order to fully understand its implications. A much larger study needs to be performed examining each of the putative contributing factors to insidious hypothermia. As the demands for human survival in a cold environment for long periods increase, it has become essential that we understand the physiological responses to the microenvironments which we create.

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The opinions or assertions contained herein are the private views of the authors.

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TECHNICAL NOTE

Effectiveness of the Space Shuttle Anti-Exposure System in a Cold Water Environment

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BAGIAN JP, KAUFMAN JW. *Effectiveness of the Space Shuttle anti-exposure system in a cold water environment.* Aviat. Space Environ. Med. 1990; 61:753–7.

The purpose of this study was to evaluate the National Aeronautics and Space Administration (NASA) Space Shuttle Launch Entry Suit (LES) and raft for 24 h of protection against cold water immersion. Two configurations, the LES and the LES with raft (Navy LRU-18/U with NASA spray shield) were evaluated for anti-exposure protection. Conditions were selected to simulate worst case water and air temperatures along projected Space Shuttle ground tracks; i.e., water temperatures = 4.4°C (40°F), air temperature = 5.6°C (42°F), 1 foot waves (chop), and constant spray. Four males, 31–44 years of age, and one 32-year-old female, were studied once in each configuration. Trials with and without a raft were scheduled for up to 24 and 6 h, respectively. Mean LES trial durations were 150 ± 9 min and final rectal temperature (T_{re}, f) = 36.5 ± 0.3°C. Mean LES with raft (LES/r) trial durations were 398 ± 126 min and final rectal temperature (T_{re}, f) = 35.6 ± 0.4°C. LES and LES/r trials were terminated for reaching T_{re} = 35.0°C or subject-requested termination due to discomfort. The longest LES and LES/r trials were terminated due to subject discomfort. Although not achieving the desired durations, the LES and LES/r did prove capable of protecting individuals, respectively, for up to 3 and 13.5 h. Since the longest LES and LES/r runs were terminated due to subjective tolerance and not physical incapacitation, actual survival times greater than 3 and 13.5 h, respectively, could be expected for individuals in these ensembles.

INTRODUCTION

WITH THE ADVENT of an escape system for use on the Space Shuttle, it became apparent that some sort of anti-exposure system would be needed to maximize the survival potential of crewmembers after exiting the Shuttle. Such a system is necessary because the Shuttle's trajectory may place it over the surface of the Earth anywhere between 60° north or south latitude at any time of the year. The worst situation in which crewmembers might find themselves would be that involving water survival. The water environment is extremely hazardous due to the risks of hypothermia and drowning (1).

In order to attack the design of an anti-exposure system, it was necessary to first determine the amount of time and under what conditions survival would be required. The maximum time the complete system would be required to protect individuals was determined to be 24 h. This was based on the rescue community's estimate of how long it would take them to find and render assistance to a downed crewmember. Most importantly, any equipment used could not have a significant negative impact on normal operations. Furthermore, the anti-exposure system would also have to integrate with the partial pressure suit to be worn by crewmembers which will enable them to tolerate loss of cabin pressure. In view of these requirements, it was elected to combine a dry-type, anti-exposure suit with the partial pressure suit forming what is known as the Launch/Entry Suit (LES). To further enhance anti-exposure protection, each crewmember would be provided with a one-person life raft. The configuration of the LES, consisting of expedition-weight CAPILENE® underwear worn under a waterproof coverall offered the maximum insulating value in water compatible with allowable cockpit hyperthermic stress and functional operational requirements, as determined in previous testing (7). The purpose of this testing program was to characterize and compare the anti-exposure protection capabilities of the LES when used alone and with a raft (LES/r).

MATERIALS AND METHODS

Subjects: All subjects were volunteers and had been thoroughly informed as to the test protocols which had been previously approved by the Human Use Committee and the potential risks associated with participation

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in this study. Prior to being accepted as a test subject each individual had to pass a U.S. Navy Class II flight physical and each subject was also examined just prior to any test run. Four males (including one author, J.B.), ages 31 to 44, and one female, age 32, were studied once in each configuration (Table I).

Weight was recorded prior to each test run and the mean for each subject calculated. Body surface area (BSA) was calculated (3) from the mean weight and height of each subject. Percent body fat was determined from estimates of body density (2), which were computed from skinfold measurements obtained with Lange Skinfold Calipers (Cambridge Scientific Inc., Cambridge, MD) and the equations of Lohman (10), for the male subjects, and Jackson and Pollock (10), for the female subject.

Anti-exposure equipment: Each subject was equipped with a LES and a one-man raft. The LES is a partial pressure suit with a dome-type helmet, which by means of counter-pressure applied by bladders and restraint material can afford the crewmember protection from ambient pressures corresponding to altitudes in excess of 100,000 feet (Fig. 1). Integrated with the partial pressure suit in the LES is a dry-type, anti-exposure suit which extends the waterproof covering of the crewmember to include the feet and provides a waterproof barrier at the wrist. The air bladders of the suit cover the entire trunk and approximately 50% of the limbs with the remainder of the limbs covered by the polytetrafluoroethylene (PFTE) membrane of the dry suit. Additional insulation is provided by a pair of long underwear tops and bottoms made of expedition weight CAPILENE®, which were worn under the LES. Protection for the hands consisted of NOMEX® gloves with integral counter-pressure bladders and ⅛ inch neoprene over-mittens.

Launch/Entry Suit (LES) and ancillary equipment used in testing were as follows: a. parachute harness; b. life vest; c. parachute pack; d. life raft pack; e. LES helmet; f. LES gloves; g. survival mittens; h. CAPILENE® underwear; i. Polypropylene socks; j. urine collection device/DAC; k. boots; and l. flotation device.

A lightweight one-man life raft was provided to the subject for those runs requiring the use of a raft. The raft selected was a U.S. Navy LRU-18/U which was modified by the addition of a spray shield (Fig. 2).

Measurements: Each subject's baseline weight was obtained on a scale accurate to ±10 g (Scale-Tronix model 6006SP, Wheaton, IL) and ECG electrodes (3M Red Dot, Minneapolis, MN, along with Gould model 4600 series amplifiers, Cleveland, OH) were placed on the subject. Heat flux temperature transducers were attached to the following ten body sites: forehead (T_A), left anterior upper chest (T_B), left distal upper arm (T_C), dorsum of left hand (T_D), right anterior thigh (T_E), left posterior thigh (T_F), right shin (T_G), right foot (T_H), right proximal upper arm (T_J), and left lower back (T_K). These transducers consisted of a thermopile heat flux transducer with a thermistor located in the center (Hamburg Associates, Jupiter, FL). Analog signals from these transducers were transmitted via hardwire and were amplified (Bioinstrumentation Association model HF-12/Temp-14, San Diego, CA) and stored on the laboratory's data collection system. A rectal thermocouple (Sensortek model RET-1, Clifton, NJ) was inserted at least 8–10 cm anterior to the anal sphincter (later runs employed redundant thermocouples). All measurements were sampled at 10-s intervals and their 1-min means were stored.



Fig. 1. Launch Entry Suit (LES).

TABLE I. PHYSICAL CHARACTERISTICS OF SUBJECTS.

Subject	Age (years)	Height (m)	Weight (kg)	% Body Fat	Surface Area (m^2)
A	36	1.82	75.4	9.3	1.96
B	44	1.73	71.5	12.6	1.85
C	31	1.83	87.4	14.4	2.10
D	34	1.88	90.3	18.9	2.17
E	32	1.65	69.8	29.5	1.77



Fig. 2. Modified U.S. Navy LRU-18/U raft with spray shield. Spray shield is shown partially retracted during bailing operation.

Mean weighted skin temperature (T_{sk}) was calculated using the equation:

$$T_{sk} = \frac{\begin{array}{c}0.1T_A + 0.125(T_B + T_K) \\ + 0.07(T_J + T_C) + 0.06T_D \\ + 0.125T_E + 0.15T_G + 0.125(T_E + T_F)\end{array}}{2} + .05T_H$$

Where Ti's are the measured skin temperatures at locations i = A through K.

Environmental conditions: Conditions were selected to represent as closely as possible the worst expected water and air temperatures that might be encountered along the Space Shuttle ground track. These were T_{water} = 4.4°C and T_{air} = 5.6°C with 1-ft waves and constant 4.4°C spray and were achieved in the Environmental Physiology Laboratory of the U.S. Naval Air Development Center.

Test configurations: In order to gain a better understanding for the performance of the anti-exposure system, two test configurations were chosen. One consisted of the subject being immersed to approximately the neck while floating with the aid of a life preserver for 2 min. Following this the subject would board the raft, bail it out with a hand-bailer, and close the spray shield (time from boarding to canopy closure = 12.8 ± 3.7 min S.E.M.). The subject would remain that way for the rest of the trial, bailing as necessary with a small hand-pump (LES/r runs). The other run consisted of floating immersed approximately to the neck by means of the life preserver until the termination of the trial (LES runs).

Test design: Subjects employed each configuration at least once for a minimum of two total exposures per subject, performed in a random fashion. Repeated trials resulted from equipment problems; the additional runs provided more representative data for analysis. The minimum time interval between tests for a given subject was 2 d, to minimize acclimatization effects. LES runs and LES/r runs were planned to last for a maximum of 6 and 24 hours, respectively. A run could be terminated for any of the following reasons: a rectal temperature (T_{re}) of 35°C or less, a hand temperature of 10°C for 5 min, a foot temperature of 4.4°C for 5 min, subject request, loss of any of the data used for test termination criteria, principal investigator request, or medical monitor request.

Statistical analysis: Initial and final T_{re} and exposure duration data were analyzed using an analysis of variance (ANOVA). Paired-t tests were used to compare variations over time, and for comparing pooled data when applicable. Differences were considered significant at the level of $p < 0.05$.

RESULTS

None of the subjects proved able to endure the test conditions for the planned maximum exposure periods. The maximum LES trial duration was 177 min (T_{re} = 36.5°C), with termination resulting from discomfort due to development of suit leakage. In fact, all other LES trials were terminated due to subject request stemming from discomfort and had durations of between 68 and 154 min (Fig. 3). The maximum LES/r trial duration was 801 min and was terminated due to subject discomfort. Generalized severe discomfort resulting from cold exposure was the cause of trial terminations. It should be noted that in the 801-min exposure, the T_{re} had steadily increased from a low of 35.1°C at minute 150 to 35.9°C at trial termination. The second lengthiest run was also terminated for subject discomfort at minute 598. Two other LES/r trials were terminated for reaching a T_{re} = 35°C. The remaining LES/r trial, involving subject A, was terminated at T_{re} = 35.1 due to a loss of T_{re} monitoring capability. Overall LES/r runs ranged in length from 176 to 801 min (Fig. 4). At the termination of all trials, all subjects demonstrated the ability to effectively aid in their own rescue (e.g., by demonstrating the manual dexterity required to operate a PRC-90-2 survival radio).

While the change in mean weighted skin temperature ($T_{sk_{\Delta f}}$) was not significantly different between LES and LES/r, the mean weighted skin temperatures (T_{sk}) as a function of time for the LES/r trials were significantly greater than the LES trials ($p < 0.01$, Fig. 5 and 6, Table II). A highly significant difference ($p < 0.001$) was observed between the initial and final foot temperature ($T_{foot_{\Delta f}}$) for the LES and LES/r. Similarly, the $T_{hand_{\Delta f}}$ was

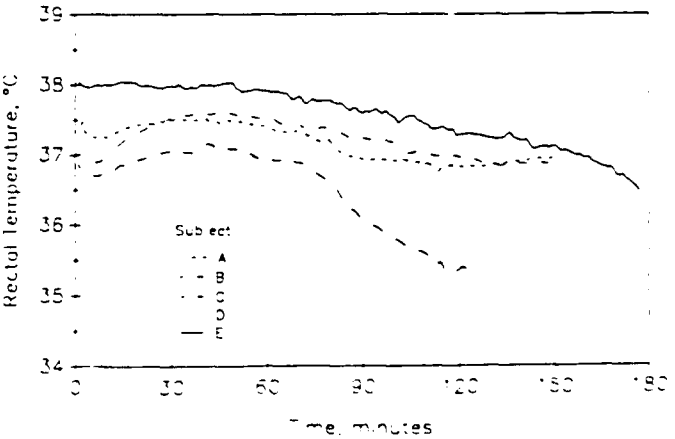


Fig. 3. T_{re} vs. time for the LES using only personal flotation, by subject. All trials terminated due to discomfort.

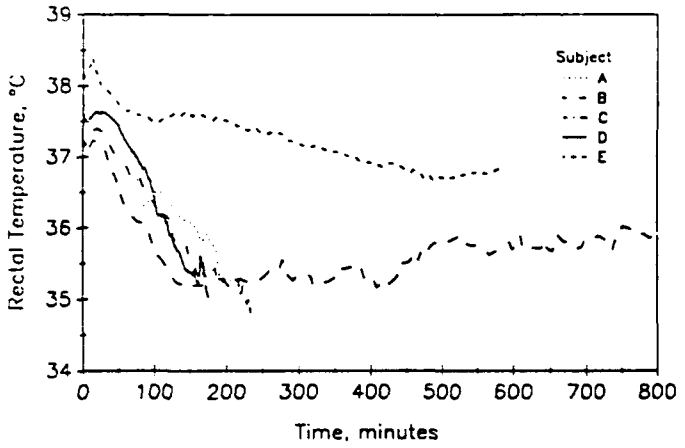


Fig. 4. T_{re} vs. time for the LES with the NASA raft. Trials for subjects C and D were terminated because T_{re} = 35°C. Sensor problems terminated subject A's trial with T_{re} approximately equal to 35°C. Subjects B and E had trial durations of 801 and 587 min, respectively, and their runs were terminated due to discomfort.

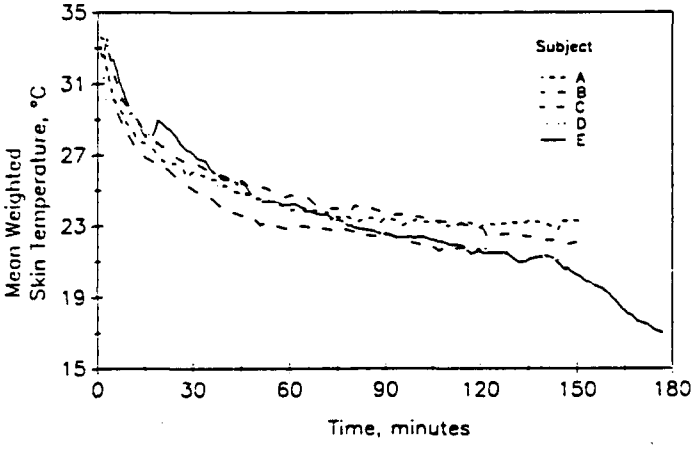


Fig. 5. T_{sk} vs. time for the LES using only personal flotation, by subject.

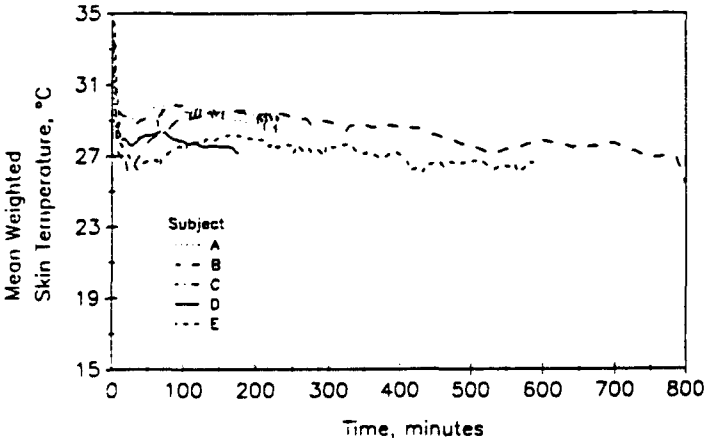


Fig. 6. T_{sk} vs. time for the LES with the NASA raft, by subject.

significantly different between the two configurations ($p < 0.05$, Table III). A comparison of change in T_{re} over the first 122 min (the maximum trial duration common to all trials in both configurations) showed that the mean difference for the LES runs was 0.9°C and for the LES/r

TABLE II. MEAN VALUES OF EXPOSURE DURATION AND MEAN WEIGHTED SKIN TEMPERATURE, BY CONFIGURATION, RESULTING FROM EXPOSURE TO EXPERIMENTAL CONDITIONS; i = INITIAL VALUE, f = FINAL VALUE.

Configuration		Exposure Duration (min)	Mean Weighted Skin Temperature (°C)		
			i	f	change
LES	Mean	150	32.7	22.2	−10.5
	S.E.M.	9	0.5	1.7	1.6
LES/r	Mean	398	33.3	27.6	−5.7
	S.E.M.	126	0.7	1.2	1.2

TABLE III. MEAN VALUES OF FOOT AND HAND TEMPERATURES, BY CONFIGURATION, RESULTING FROM EXPOSURE TO EXPERIMENTAL CONDITIONS; i = INITIAL VALUE, f = FINAL VALUE.

Configuration		Foot Temperature (°C)			Hand Temperature (°C)		
		i	f	change	i	f	change
LES	Mean	34.1	18.3	−15.9	33.6	17.7	−15.9
	S.E.M.	0.3	1.6	1.5	0.7	1.7	1.4
LES/r	Mean	33.9	26.9	−7.0	34.1	23.6	−10.5
	S.E.M.	0.4	0.9	1.1	0.6	0.5	1.1

was 1.88°C. This difference, though striking, was not statistically significant. The inter-individual variations for each configuration were significantly different ($p < 0.01$).

DISCUSSION

A comparison between LES and LES/r runs showed that the LES/r runs lasted significantly longer. It must be noted that the reasons for termination of the runs between the two configurations was different in a number of cases. Although in the case of the LES no runs were terminated for reaching a T_{re} = 35.0°C, two LES/r runs were terminated for reaching this temperature, as well as one terminated at 35.1°C for a monitoring equipment problem. The $T_{sk_{i/f}}$ did not significantly differ between the two configurations; however, the T_{sk} did significantly differ as a function of time as did the $T_{hand_{i/f}}$ and the $T_{foot_{i/f}}$. The larger rate of temperature decay of the subjects' hands and feet and T_{sk} together with the lower resulting absolute temperatures of these sites contributed in large part to the increased discomfort associated with the LES runs. While not unexpected, this resulted in the increased rate of trial termination due to discomfort in the LES trials when compared with the LES/r trials. T_{re} did not prove to be a good predictor of subject discomfort, as demonstrated by the fact that all subjects who were terminated for a T_{re} less than 35°C in the LES/r configuration had a substantially higher T_{re} during the LES trial where they voluntarily requested LES trial termination due to discomfort. The striking difference between T_{re} of the LES and LES/r trials as a function of time with the LES being generally greater than the T_{re} LES/r was unexpected and is counter-

intuitive. This observation appears to be worthy of further study, which we hope to report on in the future.

It is not uncommon to estimate survival times by linearly extrapolating the T_{re} data obtained prior to test termination (4,5,11,12). While this is often the only avenue which is available, it is obviously quite flawed in at least some instances, as demonstrated by two of the subjects in this study who not only showed their T_{re} had ceased declining but had, in fact, begun to increase (Fig. 4). It is not impossible that some of the other subjects in the study may have also demonstrated this trend reversal had their individual trials not been terminated for reaching the T_{re} = 35°C test cut-off point (5).

While the trial durations observed did not equal the objectives which had initially been defined (6 h for LES, and 24 h for LES/r) they still represent some of the longest trials ever reported from a laboratory study employing similar equipment and environmental conditions. How long an individual could endure in such conditions in an open water situation (9,11) is open to conjecture, but it is not unreasonable to believe that the trial durations which resulted from requested withdrawal would be longer in an actual survival situation where no such convenient way out would exist, all other things being equal (8). This is further supported by the observation that all subjects were able to properly operate their survival equipment and aid in their own rescue at the termination of all trials, thus demonstrating an existing functional reserve. The results observed here cast serious doubt on the practice of linear extrapolation for estimation of survival time (4,5,11,12) and open the question as to what the natural outcome of the runs which were terminated for T_{re} = 35°C would be. Additional investigation regarding this question appears to be warranted and will be reported separately.

Accordingly, although the durations demonstrated in this study did not exceed 177 min for LES, and 801 min for the LES/r, it is likely that individuals will demonstrate significantly longer durations in actual survival situations (6).

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ENHANCING CAPACITY FOR PERFORMANCE AT ALTITUDE

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I. Introduction

Successful performance at high altitude depends on a number of factors including:

1). Degree of acclimatization
2). Physical fitness
3). Pre-existing or developing medical problems - underlying cardiac or pulmonary disease, acute mountain sickness, frostbite, infection, trauma
4). Intrinsic physiological characteristics such as the hypoxic ventilatory response and hypoxic pulmonary vasoconstriction
5). Experience and skill with the particular activity
6). Environmental factors (absolute altitude achieved, temperature, weather, etc.), and the equipment to deal with these concerns such as clothing, footwear, technical gear, etc.
7). Diet - availability of food and water

In order to successfully complete a wilderness adventure, an individual must thus have the experience and skills to carry or find adequate food and water, shelter, and protection from a potentially hostile environment. The technical expertise and specialized equipment necessary to climb a mountain, run a river, or hike in the backcountry are also critical to a safe and enjoyable expedition.

After experience and external factors have been accounted for however, there remain important physiological variables that may determine work capacity and performance at altitude. The ability to modify these factors will be the focus of this presentation.

II. Physical Work Capacity

A. **Key Concept - maximal oxygen uptake (VO_2max)** is a key physiological parameter determining aerobic performance.

1 - is a function of both oxygen delivery and peripheral extraction
   a. at sea level is primarily limited by cardiac output
   b. at altitude may be further limited by diffusion in the lung
2 - has both genetic and environmental (training) components
3 - decreases with ascent to altitude approximately 1% for every 100 meters above 1500 meters
   a. may decrease to a greater extent in very well trained athletes (*but not to an absolute level less*

than an untrained individual!)

B. **Concept of "maximal steady state"** - that submaximal workload beyond which work cannot be sustained for prolonged periods of time.

1 - derived from relationship between VO_2 and ventilation, lactate, or heart rate
2 - usually occurs at a specific fraction of max

C. **Concept of submaximal endurance** - as long as work intensity is below the maximal steady state, endurance for long duration activities is determined primarily by local factors such as muscle strength and substrate availability

III. Altitude Acclimatization

A. If altitude exposure is prolonged, a complex series of physiological adjustments takes place which gradually improves the ability to perform work at altitude.

1 - increases in ventilation increase alveolar oxygen tension and improves systemic oxygenation
    a. importance of hypoxic ventilatory response
    b. may be blunted in trained athletes
2 - increases in hemoglobin and hematocrit increase oxygen delivery
3 - increased capillary density, mitochondrial number, and tissue myoglobin concentration facilitate peripheral oxygen uptake
4 - increased mobilization of free fatty acids and dependence on blood glucose spares muscle glycogen, thus improving substrate utilization.

B. Gradual acclimatization also reduces the incidence of high altitude illness

IV. Training for Altitude Performance

A. Endurance training improves VO_2max by approximately 15-40%, depending on duration and intensity
1 - remarkably similar to altitude acclimatization
2 - depends on baseline level of fitness
3 - **Key concept** - Training at either sea level or mild high altitude, improves maximal oxygen uptake **both** at sea level and at altitude by the same amount
    a. does not affect the hypoxic ventilatory response
    b. does not confer any protection from acute mountain sickness

B. Endurance training can improve submaximal work capacity and endurance by much more than it can VO2max, in the range of 100 - 200% or more.

1 - if training is done at the same *relative* intensity, endurance training at either sea level or altitude will increase endurance at both sea level and altitude.
2 - if training is done at *high* intensity at altitude, work capacity and endurance *at altitude* are increased more than can be obtained by sea level training
3 - concept of specificity of training

V. Conclusion

There is no substitute for appropriate training and experience in wilderness adventures. It is also obviously important to have the appropriate equipment, including adequate food and water to ensure a safe and successful experience. In addition, there is good evidence that basic endurance training at sea level can increase both maximal aerobic power and endurance for altitude activities. Such conditioning is critical to deal with the harsh environmental stress and physical demands of many wilderness ventures. However a high level of fitness does not protect against *high* altitude illness, and an appropriately graded ascent is the best insurance against acute mountain sickness.

Finally, for the athlete competing at altitude, it is beneficial to train for a period of time at altitude, primarily to obtain the advantages of acclimatization. If it is not possible to acclimatize substantially before competition, then training in a pressure chamber may improve altitude endurance and enhance performance.

Work and Training at Altitude

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Acute exposure to altitude results in a decrease in the amount of oxygen available to do physical work, and a reduction in maximal aerobic power of approximately 1% for every 100m above 1500m.[1] Chronic exposure to this hypoxic stress stimulates acclimatization, which includes several physiological adaptations that markedly improve the ability to perform submaximal work at altitude.[2] Increases in hemoglobin and hematocrit[3,4,5] improve the oxygen carrying capacity of the blood. Peripheral uptake of oxygen by exercising muscle is facilitated by increased capillary density[6,7] mitochondrial number[8] and tissue myoglobin concentration.[9] Substrate utilization is improved by increasing mobilization of free fatty acids and increasing dependence on blood glucose thus sparing muscle glycogen.[10,11] This is manifested by decreased accumulation of metabolites such as lactate[10] or ammonia[12] during submaximal exercise.

These changes of altitude acclimatization are remarkably similar to those induced by endurance training.[13] Increases in muscle mitochondria, capillary density, and oxidative enzyme capacity have all been well described after exercise training,[14] resulting in decreased lactate accumulation during submaximal exercise, and increased work performance. Greater reliance on fatty acids over glycogen as the primary fuel for exercising muscle also occurs with physical training.[14]

Elite athletes, in their constant search for ways to improve performance, have focused on these similarities between altitude acclimatization and endurance

training and many spend considerable resources to train at altitude.[15] The evidence supporting this practice however is controversial.[16] Most studies in this area are limited by four major problems: 1) small subject numbers, 2) lack of a clearly defined training program at altitude, 3) use of subjects of widely differing degrees of fitness, and 4) inadequate controls undergoing the same testing and training programs at sea level. How exercise training specifically interacts with altitude, whether by increasing the training stimulus either centrally or peripherally,[17] by superimposing acclimatization on endurance training,[18] by altering the perception of exercise intensity associated with ventilatory effort, or perhaps even by reducing training intensity, resulting in deconditioning,[19] thus remains unclear.

In the following review, we will first provide a historical perspective on the problems of work at altitude. We will then divide the modern research on the question of altitude training into two eras: up to and surrounding the Olympics in Mexico City, which took place at an altitude of 2250m in 1968; followed by subsequent research during the last two decades.

HISTORICAL PERSPECTIVE

That exertion at altitude was more taxing than at sea level was recognized in the eighth century, by Japan's greatest Buddhist priest, Kukai, describing the efforts of a monk named Shodo to climb a small mountain near Nikko. He wrote: "...when he had gone halfway up, his body was exhausted, his strength had left him..." Earlier Chinese traders, traveling to the Middle East across several high mountain ranges, were worried more about bandits and the headache of altitude sickness than about the labor of climbing. Mirza Haider, a Mongol Chieftain of the sixteenth century described the shortness of breath and palpitations of the heart which his men experienced while campaigning on the high Tibetan Plateau, and severe altitude sickness killed many of his men and horses.[20]

But only a few men ventured into high mountains and such effects went largely unnoted. Possibly the first scientist to describe the fatigue which limited work was De Saussure who wrote in 1787 of 3660m on Mont Blanc: "...these sturdy men for whom seven or eight hours of walking...were absolutely nothing, had hardly lifted five or six shovel fulls of snow when they found it absolutely impossible to continue."

When mountain climbing became fashionable a century later Whymper, the most celebrated climber of his time, wrote of his experience at 4880m on Chimborazo in the Andes: "...our incapacity was neither due to exhaustion or to deficiency of bodily strength...or to weakness from want of food, but it was caused by the whole of our attention being taken up in efforts to get air..."[21]

During the golden age of alpine climbing attention focussed on the symptoms caused by what Acosta in 1590 called "thinne aire"[22] and which Paul Bert proved were due to lack of oxygen.[23] Many dramatic accounts of weakness, often incapacitating, were written by mountaineers. The aggravating effect of exertion was shown by Kronecker who showed that nine subjects carried to the 2743m high Jungfraujoch were much less affected by the altitude than a comparable group who climbed. A Parisian astronomer compared the well-being of subjects hauled up in a sled with others who walked to altitude and confirmed Kronecker's findings.

Early in this century, expeditions went to Pike's Peak (4267m), Morococha (4572m) and Teneriffe (3352m), Cerro de Pasco (4360m), and, when the Nepal Himalaya had been opened to outsiders, on Cho Oyu, Makalu, and Everest (from 5790m to 7620m). Altitude illness was dissected into its different components, and the intricate relationships between ventilation, circulation and tissue utilization began to be understood. The "oxygen cascade" and the "oxygen transport system" were described; methods for calibrating effort expended were standardized and widely used.

Acclimatization

Centuries earlier both Haider and Acosta had seen that natives living at altitude were less affected there than were new arrivals. The benefits of acclimatization were recognized by Zuntz and Loewy[24] before 1895 although its importance was not fully appreciated until the expeditions to Everest in the 1920's, and the mechanisms are even now not fully grasped.

Haldane's expedition to Pike's Peak in 1911 produced the first hard data defining this process, though he mistakenly attributed it in part to the secretion of oxygen by adapted lungs.[25,26] A graphic description of work performance by acclimatized native miners at Cerro de Pasco (4328m high in the Andes), was written in 1925 by Barcroft: "Every few minutes like a bee out of some hive...someone would appear from the mouth of the mine. He would be much out of breath, he would take frequent pauses on they way up, but the weight on his back would be one hundred pounds".[27]

The outbreak of WWII brought aircraft which could climb higher than their pilots could, even if breathing pure oxygen. The armed forces of several nations found that the acclimatized flyer breathing oxygen had a higher "ceiling" than one who was not acclimatized. After the war the same principle was applied to climbers attempting Mt. Everest and other great peaks: Soviet, Japanese and German scientists tried to acclimatize mountaineers using different schedules in decompression chambers, but their data were challenged by many. For the men and women who would soon venture into space, Balke added a stay a decompression chamber on top of Pikes Peak![28]

When the 1968 Olympic Games were scheduled for Mexico City at 2250m many scientists expected that altitude would limit the competitors' capacity to exercise to the maximum at such an altitude, but the more dire predictions proved false.

The altitude proved to be surprisingly benign; several world records in speed events were surpassed (presumably secondary to the decreased wind resistance at altitude), and one of the official medical officers reported: n longer events times were slower than at sea level, but in many instances less prolonged than anticipated. The remarkable performance of the Kenyan team (who live at 1675m) suggests the possibility that prolonged training at higher elevations may confer special benefit. Other special populations include the Tarahumara Indians, for example, who demonstrate remarkable altitude endurance and run great distances at elevations of 1500 to 2500m.[29] However these altitude natives have never been successful at sea level competitions. Furthermore, no sea level records were set in endurance events in competitions which took place in the period immediately following these Olympics, raising the question of whether altitude training conferred any special advantage on performance at sea level.

TRAINING AT HIGH ALTITUDE FOR SEA LEVEL PERFORMANCE

The Mexico City Olympics Era

One of the first attempts at scientific study of whether training at altitude improves performance at sea level was the anecdotal but intriguing report by pioneer physiologist Bruno Balke. In 1965, Balke wrote in English summarizing a study done in the Austrian Alps in 1943.[30] He described three men who trained for one month at 600m, and then for one month at 3000m. Maximal aerobic power increased by about 11% after their altitude sojourn.

The 1968 Olympic Games held in Mexico City (2250m) were an important stimulus for a flurry of research in the late 1960's and early 1970's regarding training for performance **at** altitude, as well as altitude training for **sea level** performance. For this review, we will focus primarily on the latter and broader question.

Studies in Untrained Subjects

Balke, Nagle and Daniels reported in 1965 studies in five untrained men before and after ten days of training at 2800m.[31] VO_2Max increased in all subjects after altitude training, as were times in 400m and one mile time trials. Blood volume, hemoglobin, and hematocrit were all increased suggesting increased oxygen transport as a benefit of acclimatization. Klausen et al.[32] made similar observations; a 14% increase in maximal aerobic power at sea level in 5 subjects after a 5 week stay at 3800m. Unfortunately, no statistical analyses were reported and there were no controls. Their subjects ranged from a trained middle distance runner to a sedentary, heavy cigarette smoker, with similar responses in all subjects. In both these studies the use of untrained subjects without controls is an important limitation, and a simple training effect related to the increased physical activity at altitude, as well as the training and testing procedures can easily explain their observations. In the only study of this era to include a sea level control group, Hansen et al. studied 16 untrained subjects before and after 2 weeks at 4300m.[5] Half of these were randomly assigned to "increased physical activity" consisting of calisthenics (pull ups, sit ups, knee bends, and timed runs) or "reduced activity". A third group of 8 subjects lived at sea level and presumably performed similar exercises, though their activity is was not specified. There was no greater increase in VO_2Max in the altitude activity group compared to the sea level group. This well controlled study elegantly described the physiological responses to exercise at altitude, including invasive hemodynamic measurements, and suggested that activity at altitude does not improve fitness above similar activity at sea level. However, the training protocol was almost certainly inadequate to produce an optimal endurance training effect.

Studies in Athletes

To control for a bias due to the level of training, Faulkner, Daniels and Balke studied five well-conditioned runners and fifteen highly conditioned college-age swimmers.[33] Subjects began a uniform exercise regime six weeks before altitude training. The runners then spent 23 days at 2300m, and the swimmers 14 days at the same altitude. Post altitude training evaluation was done 3 and 21 days after the return from high altitude. VO_2Max (l/m) and maximum workload significantly increased in all runners after altitude training. The swimmers showed no benefit of altitude training. The limitation to pulmonary ventilation in prone swimming may complicate the interpretation of the results in the swimmers in this study. The authors were not rigorous about documenting that all of the runners had reached a training plateau before their altitude training. Attainment of a plateau in VO_2Max, or other indices of training, remains a clear prerequisite for informative studies in this field.

The authors made the interesting observation that some individuals had better race times after altitude training in spite of no changes in VO_2Max. They attributed the improved race times to enhanced aerobic work capacity. Similar uncontrolled studies on trained athletes were performed by Dill and Adams[34] and Buskirk et al.[35] with divergent results. Dill and Adams studied six male high school students who were all conference or district high school track champions. They lived at 3090m (530 torr) for 17 days. While they reduced the speed of their running, by training twice daily, they actually increased their total mileage compared to the 6 weeks immediately preceding the study. Sea level VO_2Max increased significantly by 4.2%. Buskirk on the other hand showed no improvement in performance either in the laboratory or on the track in 6 well-conditioned runners after two months in Peru at 4000m. These athletes followed no specific training

program at altitude and were additionally exposed to the stresses of third world travel.

In a well-designed study conducted at several different altitudes, Faulkner et al. used college track athletes "who were in a steady state of training at sea level when their study began".[36] Although the authors began the study with a uniform group, they did not prescribe uniform training intensity and duration at the different altitudes; the athletes trained at will. Three of five men that spent 34 days at 2300m had increased VO_2Max on return to sea level while none of the group that spent ten days at 4300m had improved VO_2Max. These subjects experienced mountain sickness during their initial stay at 4300m which largely prevented them from training. Saltin found that performance was unchanged or impaired in Swedish Olympic athletes after a stay at 2300m and hypothesized that for the most highly trained athletes, exercising at altitude forces a reduction in training workload which may minimize any benefit of altitude exposure.[37] Daniels and Oldridge attempted to address this question by interspersing periods of sea level training during a 10 week program of altitude training at 2300m in 6 world class runners.[38] Such a strategy would theoretically allow the runners to maintain the intense training workloads necessary for competing at a world class level. They observed a trend toward a 5% increase in VO_2Max after training and noted that 14 personal best times were run in post-altitude races. However, the change in VO_2Max did not reach statistical significance when tested by repeated measures ANOVA (statistics not reported in the original manuscript), and there was no control group performing similarly intense exercise entirely at sea level.

Chamber Studies

An important problem highlighted by the above studies was in determining the appropriate altitude at which to train. Roskamm et al.[39] addressed this question by studying 18 untrained male students randomly assigned to train for four

weeks in a hypobaric chamber, either at sea level, at 2250 meters or at 3450 m. The exercise program was completely individualized for each subject and standardized among groups. Each man trained for 30 minutes a day, 6 days a week in the altitude chamber for 4 weeks. The greatest increase in VO_2Max (17.5%) was seen in the group who trained at the **intermediate** altitude. This increase however was not statistically different from the 6.4% increase seen in the group training at sea level. The increase seen in the group training at the highest altitude (10%) was less than the intermediate group, but did reach statistical significance. This curious finding raises the question of very divergent responses among different subjects which cannot be answered without the individual data. Type II error of course is also possible in a study of this size. Although it is not specifically stated in the report, it appears that the subjects were only exposed to altitude during the training period. Thus this study addresses the question of graded hypoxic exercise, rather than altitude acclimatization, and suggests that moderate altitude might allow exercise at sufficient intensity to optimize training workload, but still derive the advantages of altitude exposure. No increase in hemoglobin was observed in any of the three groups, thus providing further evidence for the absence of acclimatization.

Chamber studies allow for more carefully controlled conditions and another ingenious approach to experimental design was used in a small study by Loeppky and Bynum[40] when they had nine fit subjects exercise at either a control altitude of 628 torr (1575m) or 523 torr (3050m) in a hypobaric chamber, with the subject **blinded** as to the altitude. Subjects exercised for one hour/day; treadmill running, cycle ergometry and calisthenics were done at progressively increasing altitudes from 3050m to 4270m. The altitude trained group (n=5) had a greater increase in VO_2Max than the control group (n=4), though this was not statistically significant. Total running time on the incremental test used to measure VO_2Max did improve to a significantly greater extent in the altitude trained group compared to control,

suggesting an increase in oxygen debt and possibly anaerobic capacity. Like Roskamm et al., these authors reported no changes in hemoglobin or hematocrit.

After the rush of study before, during and after the 1968 Olympic Games, several conclusions apparently rested in firm ground in 1970, although some would soon be challenged:

1. Maximal work capacity decreases roughly 1% for each 100m above 1500m but returns slightly toward sea level values during an extended stay at high altitude.

2. Long residence at high altitude does not enhance endurance performance at sea level.

3. In endurance competition at medium altitudes, athletes who have spent a lifetime at medium or high altitude appear to have a physiological advantage over those born and raised at low altitude.

4. In brief, intense exertion neither prior acclimatization nor altitude makes an appreciable difference.

5. No agreement existed about whether intermittent altitude exposure is adverse or beneficial in training for competition sports or for slow paced climbing.

6. Immense differences in speed and in work capacity between individuals had been demonstrated. Further differences were thought to arise from the type of work or exercise, level of training, and length of time spent at altitude. Many of these factors are unpredictable and probably explain the contradictory results found in different studies. They do help explain why some of these beliefs were soon to be altered.

The Post-Mexico City Era

In 1975, the most carefully controlled study up to that date was performed by Adams and colleagues, who reported an elaborate study of altitude training in well conditioned college middle-distance runners.[41] Matched groups trained at sea level, or 2300m for 3 weeks and then crossed over to the opposite training site.

Training intensity and duration were carefully controlled at the same *relative* workload at each altitude (6.5% slower pace at 2300m compared to sea level; i.e., lower *absolute* workload). They report no improvement in either maximal aerobic power, or 2 mile time trials, after altitude training upon return to sea level. They caution the reader that their results apply only to very well-conditioned athletes at this specific altitude. They suggest that the benefit observed by others may be due to an altitude threshold, time of testing after return to sea level, training intensity at altitude and the aerobic power of the athletes studied. It also re-emphasizes the question of whether the *absolute* training workload is the critical issue with altitude training. In 1978, Bannister and Woo reported on a complex training regimen consisting primarily of interval type, intensive training breathing a hypoxic gas mixture (12% O_2).[42] Both aerobic power and anaerobic capacity appeared to improve, though the lack of controls undergoing similarly intensive training make this study difficult to interpret.

Recent Training Studies

In the past few years, a number of well controlled training studies have been performed and have shed some light on the physiological effects and practical utility of altitude training. Terrados et al.[43] studied 4 competitive cyclists training daily in a hypobaric chamber at 2300m for 3-4 weeks, compared to 4 similarly matched cyclists training at sea level. They strictly controlled training intensity, and measured work capacity and skeletal muscle metabolic activity after training. They found that work capacity at sea level was improved by the same degree regardless of training altitude. Work capacity *at altitude* however was increased more by altitude training (33% vs 14%). Muscle capillarity was increased and glycolytic capacity was reduced, which paralleled a reduction in lactate concentration at submaximal workloads at altitude. Levine et al. addressed the issue of training intensity by randomly dividing 21 fit but untrained subjects into 3 matched groups: one group

trained at sea level at 70% of measured sea level VO_2Max; a second group trained in a hypobaric chamber at 2500m at the same *relative* workload (i.e., 70% of measured altitude VO_2Max); and the third group trained at altitude at the *absolute* workload that would require 70% VO_2Max at sea level, i e., same absolute but greater relative workload.[44] Endurance time, both at sea level and altitude was also measured at 85% of altitude specific VO_2Max. They observed that both sea level and altitude VO_2Max improved by approximately 14% regardless of training altitude or intensity. Endurance however was improved most *at the training altitude.* Mizuno et al. added an important wrinkle by studying a group of 10 elite cross-country skiers after 2 weeks of training at 2700m.[45] They ensured that their athletes had reached a training plateau by repeatedly measuring VO_2Max over a number of months prior to the study to document stability of aerobic power and anaerobic capacity. They observed no change in maximal oxygen uptake, but noted an increase in maximal O_2 deficit and running time to exhaustion after the training period. By examining muscle biopsies in both the upper and lower extremities, they were able to sort out the relative effects of altitude exposure (changes in both gastrocnemius and triceps) from muscle specific training with cross-country skiing. The triceps, which were used relatively more intensively at altitude (skiing > running) demonstrated increases in capillarity, while the gastrocnemius, used less with skiing than with running demonstrated decreases in mitochondrial enzyme activities, consistent with relative detraining. However, both muscle groups showed increases in buffer capacity, which the authors hypothesized was the primary altitude specific effect that could explain the observed increase in anaerobic capacity and running time. Finally, Levine et al. have recently described a unique approach of studying two groups of trained runners (n = 10). One group lived at altitude (2500m) and the other lived near sea level (1280m) with both groups training together near sea level.[18] They hypothesized that the athletes living at

altitude but training near sea level would develop the salutary changes of acclimatization, but still train at a high enough aerobic power to maintain their high level of fitness. Training intensity was carefully controlled and monitored by testing of heart rate, lactate and VO_2 in the field during training sessions. The athletes who lived at altitude improved their VO_2Max by approximately 5%, associated with almost 30 seconds faster 5000m performance on the track. They also identified a 500 ml increase in blood volume and hypothesized that acclimatization rather than hypoxic exercise is the key to altitude training and serves to increase oxygen transport (a natural form of "blood doping"). No changes were seen in the athletes who lived near sea level.

Basic Mechanisms

Several recent studies have provided some insight into the nature of the interaction between hypoxia and exercise. Connett has reviewed the current understanding of the way in which hypoxia acts at the cellular level to alter intracellular processes.[46] While the details of this concept are beyond the scope of the current review, this paper emphasizes the importance of viewing hypoxia as an interaction between the cellular subsystems of oxidative phoshphorylation, the Krebs cycle, glycolysis, substrate supply, and cell energetics, to match ATP demand and aerobic ATP production. Under most atmospheric conditions under which altitude training might occur, true skeletal muscle O_2 limited cytochrome turnover, termed "dysoxia" is unlikely to exist. In fact, local metabolism is probably less affected during hypobaric hypoxic exercise than expected from the decrease in arterial PO_2.[47] Rowell and colleagues have demonstrated that hypoxemia is a potent stimulus to skeletal muscle blood flow during exercise at any given oxygen consumption, even at very high workloads.[48] They observed relatively high femoral venous O2 content with maximal quadriceps exercise during hypoxia, suggesting that muscle is overperfused relative to its oxygen uptake. Furthermore,

O2 delivery to muscle was essentially constant at any VO_2 under either normoxic or hypoxic conditions, providing evidence that O2 delivery to muscle may somehow be a regulated variable. These concepts are important in understanding the recent observations of Terrados et al.,[17] who performed an elegant study examining ten subjects who trained one leg under normobaric conditions, and the other under hypobaric conditions (2300m). Each leg was trained at the same absolute work load and therefore the same degree of oxygen turnover and substrate flux through mitochondrial aerobic enzyme systems. They observed a striking increase in citrate synthase activity (a marker of the carboxylic acid cycle) in the leg trained under hypobaric conditions, along with an increase in myoglobin content. This study provides evidence for a specific synergistic interaction between exercise and altitude which increases the magnitude of the training stimulus. Because the substrate flux and metabolic demands were the same for each leg, the authors hypothesized that lowered blood oxygen content (along with increased blood flow) might be the primary stimulating factor. An alternative explanation could also be that the same *absolute* workload under hypobaric conditions, was a greater *relative* workload, the latter being the most important factor determining the cardiovascular response to exercise.[49]

Finally, two other unique studies deserve a brief mention. The first was a study by Withey and colleagues who demonstrated both sodium and water retention, with an increase in plasma volume of 0.76 liters when altitude exposure (up to 3629m) was combined with prolonged, low grade exercise (hill walking for 7hr/day).[50] This increase in plasma volume with an increase in extracellular fluid, was at the expense of the intracellular space which decreased by approximately 1 liters. The mechanism was thought secondary to an observed rise in plasma aldosterone concentration and renin activity, in contrast to the changes usually seen with altitude exposure without exercise. The second study was by Mairburl et al.

who observed a decrease in hemoglobin oxygen affinity (shift of oxyhemoglobin dissociation curve to the right), manifested by an increase in the P-50 value (oxygen pressure at 50% saturation of hemoglobin) and an increase in 2,3 DPG that was significantly greater in untrained subjects who exercised at altitude (2300m) compared to subjects who remained sedentary.[51] These changes were associated with an increase in exercise capacity at altitude, presumably facilitating an increase in A-V O_2 difference.

Conclusions

In summary, after more than 25 years of specific investigation, the effect of altitude training is becoming clearer, though by no means fully settled. Both altitude acclimatization and hypoxic exercise appear to play important roles in improving oxygen carrying capacity of the blood, and altering the training stimulus at the skeletal muscle level. It is reasonable to conclude that for performance at altitude, acclimatization and training at altitude carry important benefits. The situation for performance at sea level is probably divergent, depending on the level of fitness and training, the training altitude, and the specifics of the training program. For untrained individuals, there is probably minimal benefit to training at altitude compared to equivalent training at sea level with regard to improving maximal aerobic power. Endurance and work capacity however are likely to be most improved at the training altitude. For elite athletes, training at altitudes above 3000m probably limits absolute training intensity and precludes any favorable impact of altitude acclimatization. Training at lower altitudes, if sufficiently intense may carry added advantages, though this remains to be proved. The most efficient technique may well be living at altitude and training at sea level, though logistical difficulties may limit the utility of this approach in practice.

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CAN YOU ENHANCE CAPACITY FOR PERFORMANCE IN THE COLD?

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I. INTRODUCTION AND LEARNING OBJECTIVES

- A. UNDERSTAND HUMAN THERMOREGULATORY RESPONSES TO COLD, AND PATTERNS OF ADAPTATION
- B. UNDERSTAND HOW ACCLIMATIZATION, PHYSICAL FITNESS, AGING, HYDRATION AND NUTRITION STATUS AFFECT RESPONSE TO COLD

II. ACUTE COLD STRESS

- A. REVIEW OF HUMAN THERMOREGULATORY RESPONSES TO COLD
- B. EFFECTS OF AGING ON THERMOREGULATORY RESPONSE TO COLD
- C. EFFECTS OF PHYSICAL TRAINING ON THERMOREGULATORY RESPONSE TO COLD
- D. EFFECTS OF HYDRATION AND DIET ON THERMOREGULATORY RESPONSE TO COLD

III. HUMAN PHYSIOLOGICAL ADAPTATIONS TO REPEATED AND/OR CHRONIC COLD STRESS

- A. DEFINITIONS
  1. COLD ACCLIMATIZATION
  2. COLD ACCLIMATION
  3. COLD HABITUATION
- B. DIFFERENT TYPES/PATTERNS OF HUMAN COLD ADAPTATION
  1. THERMOREGULATORY RESPONSES TO WHOLE BODY COLD EXPOSURE
     - a. METABOLIC ADAPTATION
     - b. HYPOTHERMIC ADAPTATION
     - c. INSULATIVE ADAPTATION
  2. REGIONAL (HANDS AND FEET) RESPONSES TO REPEATED LOCALIZED COLD EXPOSURE
  3. CLASSICAL CONDITIONING FOR INDIVIDUALS WITH RAYNAUDS SYNDROME OR PREVIOUS COLD INJURY

C. RELATIVE IMPORTANCE OF PHYSIOLOGICAL ADAPTATIONS COMPARED TO BEHAVIORAL ADAPTATIONS FOR DEFENSE OF BODY TEMPERATURE AND PREVENTION OF INJURY DURING EXPOSURE TO COLD

1. WHOLE-BODY COLD EXPOSURE
2. LOCALIZED COLD EXPOSURE

a. HANDS
b. FEET

IV. SUMMARY AND CONCLUSION

CAN YOU ENHANCE CAPACITY FOR PERFORMANCE IN THE HEAT?

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I. LEARNING OBJECTIVES
   - A. UNDERSTAND HOW HEAT ACCLIMATION STATE, LEVEL OF AEROBIC FITNESS, HYDRATION LEVEL AND SKIN DISORDERS AFFECT THERMOREGULATORY RESPONSES AND EXERCISE PERFORMANCE IN THE HEAT
   - B. UNDERSTAND WHETHER GENDER AND AGING AFFECT EXERCISE-HEAT TOLERANCE

II. INTRODUCTION
   - A. FACTORS INCREASING HEAT PRODUCTION AND HEAT LOSS
   - B. HEAT ILLNESSES AND THEIR INFLUENCE ON HUMAN PERFORMANCE

III. EXERCISE-HEAT ACCLIMATION
   - A. CLASSIC PHYSIOLOGICAL ADJUSTMENTS DURING ACCLIMATION
     1. REDUCED HEART RATE
     2. LOWERED SKIN AND CORE TEMPERATURES
     3. POTENTIATED SWEATING RESPONSES
     4. GREATLY IMPROVED EXERCISE-HEAT TOLERANCE
   - B. PERCEPTION OF EFFORT AND THERMAL SENSATION GREATLY IMPROVED DURING ACCLIMATION
   - C. COMPLETE ACCLIMATION OCCURS AFTER 10-14 DAYS EXPOSURE
   - D. 75% OF PHYSIOLOGICAL ACCLIMATION RESPONSES DEVELOPED BY END OF FIRST WEEK
   - E. REGULAR HEAVY EXERCISE IN THE HEAT MOST EFFECTIVE METHOD IN DEVELOPING ACCLIMATION
   - F. DAILY 100 MIN EXPOSURES OPTIMAL TO INDUCE ACCLIMATION PROCESS

IV. AEROBIC FITNESS
   - A. HIGH AEROBIC FITNESS ACHIEVED THROUGH ENDURANCE TRAINING REDUCES PHYSIOLOGICAL STRAIN DURING EXERCISE IN THE HEAT
   - B. HIGH AEROBIC FITNESS IS MAJOR FACTOR IN RETENTION OF HEAT ACCLIMATION BENEFITS AND RAPID REACCLIMATION
   - C. IMPROVED AEROBIC FITNESS BY ENDURANCE TRAINING MUST BE RELATED TO SIGNIFICANT ELEVATIONS IN CORE TEMPERATURE DURING TRAINING TO IMPROVE EXERCISE-HEAT TOLERANCE

V. HYDRATION LEVEL
- A. DEHYDRATION DEGRADES ENDURANCE PERFORMANCE IN THE HEAT
- B. PERFORMANCE DEGRADED WHEN BODY WEIGHT LOSS EXCEEDS 2%
- C. DEHYDRATION LEVELS GREATER THAN 2% ASSOCIATED WITH ELEVATIONS IN CORE TEMPERATURE AND HEART RATE WHILE SWEATING RESPONSE IS REDUCED

VI. SKIN DISORDERS
- A. HEAT RASH (MILIARIA RUBRA) AND SUNBURN RESULT IN EXERCISE-HEAT INTOLERANCE
  1. HEAT RASH
     - (a) HEAT RASH CAUSES REDUCTIONS IN PERFORMANCE TIME DURING EXERCISE IN THE HEAT
     - (b) ELEVATES MEAN BODY TEMPERATURE AS WELL AS HEAT STORAGE WHEN COMPARED TO RESPONSES IN A NON-RASH STATE
     - (c) EXERCISE-HEAT INTOLERANCE DEMONSTRATED WITH 20% OF BODY SURFACE RASHED
     - (d) INTOLERANCE PERSISTS FOR UP TO THREE WEEKS
  2. SUNBURN
     - (a) RESPONSIVENESS AND CAPACITY OF THE SWEAT GLAND TO DELIVER SWEAT TO THE CUTANEOUS SURFACE IMPAIRED
     - (b) PERCEPTION OF EFFORT AND THERMAL SENSATION ELEVATED AFTER SUNBURN SUGGESTING ENHANCED SUGGESTIVE DISTRESS
     - (c) PREVENTION OF THE EGRESS OF SWEAT FROM THE GLAND AFTER SUNBURN PROBABLE CAUSE

VII. GENDER CONSIDERATIONS
- A. IN GENERAL, WOMEN THERMOREGULATE LESS EFFECTIVELY THAN MEN WHEN EXPOSED TO ACUTE HEAT STRESS
- B. MEN AND WOMEN SIMILAR IN AEROBIC FITNESS AND SELECTED MORPHOLOGICAL FACTORS DO NOT DIFFER DRAMATICALLY IN EXERCISE-HEAT TOLERANCE

VIII. EFFECTS OF AGING
- A. IN GENERAL, MIDDLE-AGED AND OLDER MEN AND WOMEN DISPLAY GREATER EXERCISE-HEAT INTOLERANCE THAN THOSE YOUNGER
- B. OLDER AND YOUNGER MEN AND WOMEN MATCHED FOR AEROBIC FITNESS AND SELECTED MORPHOLOGICAL FACTORS DO NOT DIFFER IN EXERCISE-HEAT TOLERANCE

Assessment of Performance Capacity for Wilderness Adventure

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Presentation Outline

I. Background

A. *Factors limiting physical performance*
1. Biological capacity - functional capacity (physical fitness)

2. Biomechanical - skill

3. Psychological - motivation/concentration

B. *Biological Capacity*
1. Heredity - who you selected as parent can be very important

2. Environmental - physical training
nutrition
ergogenic aids

C. *Limitations of Performance*

1. High speed/power up to 15 seconds	high energy compounds available in muscle muscle fiber composition heredity > environment
2. Sustained Speed 10 seconds - 4 minutes	lactic acid accumulation in skeletal muscle fiber type, glycogen content heredity > environment
3. Long Distance 4 minutes - 60 minutes	0_2 transport by CV system to muscle stroke volume of heart oxidative capacity of skeletal muscle environment > heredity
4. Sustained Activity 60 minutes - hours Sustained activity on consecutive days	oxygen content of skeletal muscle fiber type & training & nutrition environment > heredity

II. Clinical Exercise Testing to Assess Functional Capacity

A. Factors to consider in making decision to test

1. clinical status of participant - risk factors, medical history
2. age
3. nature of physical & psychological demands & exposure or adventure

B. Objectives of testing

1. To determine functional capacity (workload, METS, $V0_2$, etc.)
2. To elicit abnormal response (ischemia, BP, arrhythmia, claudication, etc.)
3. To provide individualized exercise training or participation guidelines

C. *Exercise Testing Protocol Considerations*

1. Pre-test medical evaluation - exclusion criteria
2. Exercise test device - treadmill > cycle > steps
3. Selection of workload profile - continuous, multistage
4. Test termination criteria - submaximal vs. maximal
5. Measurements recorded - ECG, BP, RPE, V_E, $V0_2$, other

D. *Test Interpretation and Recommendations*

1. Measurement of functional capacity - age and gender specific values
2. Abnormal test results - what advice should be given regarding participation in wilderness adventure?
   (a) myocardiac ischemia
   (b) left ventricular dysfunction
   (c) significant dysrhythmias
   (d) exercise hypertension
3. Translation of results to wilderness adventure activities
   (a) Physical demands
   (b) Environmental demands
   (c) Psychological/risk demands

Guidelines for Testing Functional Capacity Using Treadmill Exercise Testing

A. Medical Indication for Exercise Testing
1. Evaluate functional capacity
2. Diagnose etiology of chest pain
3. Determine prognosis and severity of coronary artery and valvular disease
4. Evaluate treatment, both surgical and medical (drug effects)
5. Evaluate dysrhythmias
6. Evaluate peripheral vascular and pulmonary disease
7. Screen for asymptomatic coronary artery disease
8. Determine an exercise prescription

B. Medical Screening Prior to Exercise Testing
1. Medical History Questionnaire

2. Physical Examination
   (a) 12-lead resting EKG
   (b) Blood pressure: supine, sitting and standing
   (c) Auscultation of lungs
   (d) Auscultation of heart for murmurs, gallops, etc.
   (e) Orthopedic problems
   (f) Carotid, abdominal or femoral bruits
   (g) Palpation of carotid, femoral and pedal pulse

C. Medical Contraindications to Exercise Testing
1. Absolute:
   (a) Resting EKG suggesting acute myocardial infarction
   (b) Unstable angina
   (c) Serious dysrhythmias, i.e. V-tach, 3rd-degree A-V block, uncontrolled atrial fibrillation
   (d) Severe aortic or mitral stenosis

2. Relative:
   Relative contraindications in this context suggest that it is up to the physician's discretion to proceed with the test in light of the following existing conditions in their patient.
   (a) Anemia or high fever
   (b) Atrial ventricular conduction defects
   (c) Untreated sustained hypertension
   (d) Electrolyte abnormalities, i.e., low potassium/calcium levels
   Additional absolute and relative contraindication can be found in the ACSM Guidelines for Exercise Testing and Prescription (see reading list)

D. Need to Sign Informed Consent

Individuals should sign an informed consent form prior to stress testing. The purpose of this form is to make certain the patient if aware of the small, but real risk of stress testing. The risk is approximately 1 death and 3 cardiac events per 10,000 tests.

E. Test Protocol Selection
   1. Bruce: ostensibly healthy, asymptomatic individuals
   2. Mod-Balke: symptomatic and asymptomatic individuals
   3. Mod-Bruce/Naughton: high risk and/or symptomatic individuals

   It is apparent that the population being evaluated and time will dictate what protocol is suitable.

F. Instruction to Patient
   1. Patients should abstain from food, tobacco, alcohol, and caffeine for at least 3 hours prior to testing.
   2. Women should bring loose fitting blouse that buttons down the front with short sleeves and should avoid restrictive undergarments such as panty hose or a girdle. It is best to wear a snug fitting bra made of a material other than nylon.
   3. Clothing should permit freedom of movement and include a comfortable walking/ running shoe.
   4. Correct walking technique on treadmill with objective, if possible, of no gripping of bars with hands.
   5. Patient should be instructed to indicate any type of discomfort including shortness of breath, leg pain, arm pain, jaw pain, etc.

G. EKG Records, Prior, During and Following Stress Test
   1. 12-lead EKG
      (a) Supine
      (b) Standing
      (c) Standing/sitting hyperventilation: for purpose of inducing ST segment depression owing to a variation in autonomic nervous system
      (d) Minimum of 1 record per stage of exercise
      (e) Peak/immediate post-exercise record
      (f) One record for first 3 minutes of recovery followed by 2 minute intervals until the patient returns to pretest baselines (approximately 6-10 minutes).

H. Blood Pressures
   1. Same as EKG recordings

I. Criteria for Test Termination
   1. Patient requests to stop
   2. Pre-determined endpoint is reached (Borg Scale 17-20)
   3. Progressive angina (on scale of 104 stop at 3+)
   4. 1.0 mm horizontal or downsloping ST-depression or elevation
   5. Sustained supraventricular tachycardia
   6. Ventricular tachycardia
   7. Significant drop (10 mmHg) or failure of the systolic blood pressure to rise with increase in exercise
   8. Systolic > 250 mmHg, or diastolic > 120 mmHg
   9. R on T PVC
   10. Multifocal PVC's
   11. Patient is confused, dizzy, losing coordination on treadmill, etc.

Suggested Readings for Exercise Testing to Evaluate Functional Capacity

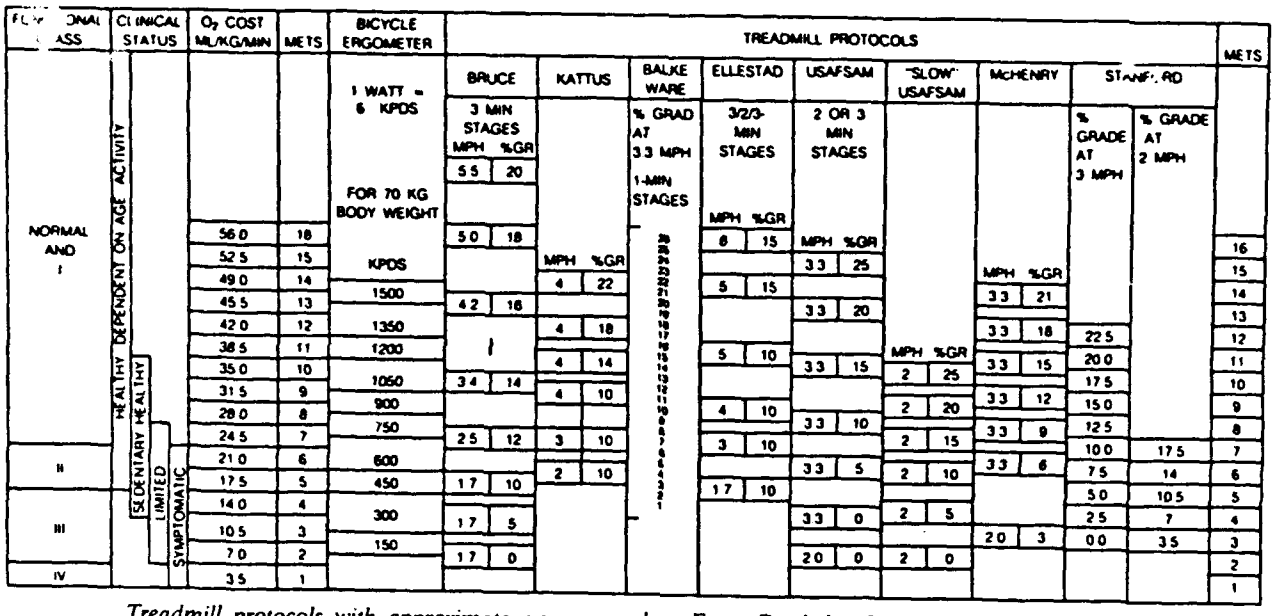
1. American College of Sports Medicine. Guidelines for Exercise Testing and Prescription (3rd Edition) Lea and Febiger, Philadelphia, PA, 1986.

2. Froelicher, VF. Exercise and the Heart: Clinical Concepts. Book Medical Publishers, Chicago, Ill, 1989.

3. Exercise Standards: A Statement for Health Professionals from The American Heart Association. American Heart Association, Dallas, TX, 1991.

4. American College of Sports Medicine. Resource Manual for Guidelines for Exercise Testing and Prescription. Lea and Febiger, Philadelphia, PA, 1988.

5. Skinner J. Exercise Testing and Exercise Prescription for Special Cases. Lea and Febiger, Philadelphia, PA, 1987.



Treadmill protocols with approximate oxygen uptakes. From Froelicher VF: Exercise and the Heart: Clinical Concepts. *Chicago, Year Book Medical Publishers, Inc, 1987, p 15. Reproduced with permission.*

Risk of Sudden Cardiac Arrest During Exercise Training

Study	Status	Activity	Monitoring	Supervision	Sudden cardiac arrests events/hr
In the general population					
Vuori et al[57]	normal	cross-country skiing	none	none	1/600,000
Gibbons et al[58]	normal	jogging swimming tennis	none	none	1/375,000
Thompson et al[59]	normal	jogging	none	none	1/396,000
Vander[60]	normal	jogging court games	none	none	1/888,000
Average for general population					1/565,000
Individuals with known heart disease					
Fletcher and Cantwell[61]	cardiac	jogging	intermittent	present	1/6,000
Leach et al[62]	cardiac	jogging	intermittent		1/12,000
Mead et al[63]	cardiac	jogging	intermittent	present	1/6,000
Hartley[64]	cardiac	jogging	intermittent	present	1/6,000
Hossack and Hartwig[65]	cardiac	jogging	none	present	1/65,185
Haskell[66]	cardiac	mixed	intermittent	present	1/22,028
Van Camp and Peterson[67]	cardiac	mixed	continuous	present	1/117,333
Hartley*	cardiac	mixed	continuous	present	1/98,717
Van Camp*	cardiac	mixed	intermittent	present	1/121,955
Hartley*	cardiac	bicycling walking	intermittent	none	1/70,000
Fletcher*	cardiac	mixed	intermittent	present	0/70,200
Average all cardiac					1/59,142

*Unpublished data.

Approximate Energy Requirements in METs For Horizontal and Grade Walking

% Grade	mi·h⁻¹	1.7	2.0	2.5	3.0	3.4	3.75
	m·min⁻¹	45.6	53.7	67.0	80.5	91.2	100.5
0		2.3	2.5	2.9	3.3	3.6	3.9
2.5		2.9	3.2	3.8	4.3	4.8	5.2
5.0		3.5	3.9	4.6	5.4	5.9	6.5
7.5		4.1	4.6	5.5	6.4	7.1	7.8
10.0		4.6	5.3	6.3	7.4	8.3	9.1
12.5		5.2	6.0	7.2	8.5	9.5	10.4
15.0		5.8	6.6	8.1	9.5	10.6	11.7
17.5		6.4	7.3	8.9	10.5	11.8	12.9
20.0		7.0	8.0	9.8	11.6	13.0	14.2
22.5		7.6	8.7	10.6	12.6	14.2	15.5
25.0		8.2	9.4	11.5	13.6	15.3	16.8

Approximate Energy Requirements in METS for Horizontal and Uphill Jogging/Running

a. Outdoors on solid surface

% Grade	m·h⁻¹	5	6	7	7.5	8	9	10
	m·min⁻¹	134	161	188	201	215	241	268
0		8.6	10.2	11.7	12.5	13.3	14.8	16.3
2.5		10.3	12.3	14.1	15.1	16.1	17.9	19.7
5.0		12.0	14.3	16.5	17.7	18.8		
7.5		13.8		18.9				
10.0		15.5	18.5					

b. On the treadmill

% Grade	m·h⁻¹	5	6	7	7.5	8	9	10
	m·min⁻¹	134	161	188	201	215	241	268
0		8.6	10.2	11.7	12.5	13.3	14.8	16.3
2.5		9.5	11.2	12.9	13.8	14.7	16.3	18.0
5.0		10.3	12.3	14.1	15.1	16.1	17.9	19.7
7.5		11.2	13.3	15.3	16.4	17.4	19.4	
10.0		12.0	14.3	16.5	17.7	18.8		
12.5		12.9	15.4	17.7	19.0			
15.0		13.8	16.4	18.9				

Table D–4. Approximate Energy Expenditure in METs During Bicycle Ergometry

Body Weight		Exercise Rate (kg·m·min⁻¹ and Watts)						
kg	lbs	300 50	450 75	600 100	750 125	900 150	1050 175	1200 (kg·m·min⁻¹), 200 (Watts)
50	110	5.1	6.9	8.6	10.3	12.0	13.7	15.4
60	132	4.3	5.7	7.1	8.6	10.0	11.4	12.9
70	154	3.7	4.9	6.1	7.3	8.6	9.8	11.0
80	176	3.2	4.3	5.4	6.4	7.5	8.6	9.6
90	198	2.9	3.8	4.8	5.7	6.7	7.6	8.6
100	220	2.6	3.4	4.3	5.1	6.0	6.9	7.7

NOTE: VO_2 for zero load pedaling is approximately 550 ml·min⁻¹ for 70 to 80 kg subjects.

Leisure Activities in METS: Sports, Exercise Classes, Games, Dancing

	Mean	*Range*
Archery	3.9	3–4
Back Packing	—	5–11
Badminton	5.8	4–9+
Basketball		
Gameplay	8.3	7–12+
Non-game	—	3–9
Billiards	2.5	–
Bowling	—	2–4
Boxing		
In-ring	13.3	–
Sparring	8.3	–
Canoeing, Rowing and Kayaking	—	3–8
Conditioning Exercise	—	3–8+
Climbing Hills	7.2	5–10+
Cricket	5.2	4.6–7.4
Croquet	3.5	–
Cycling		
Pleasure or to work	—	3–8+
10 mph	7.0	–
Dancing (Social, Square, Tap)	—	3.7–7.4
Dancing (Aerobic)	—	6–9
Fencing	—	6–10+
Field Hockey	8.0	–
Fishing		
from bank	3.7	2–4
wading in stream	—	5–6
Football (Touch)	7.9	6–10
Golf		
Power cart	—	2–3
Walking (carrying bag or pulling cart)	5.1	4–7
Handball	—	8–12+
Hiking (Cross-country)	—	3–7
Horseback Riding		
Galloping	8.2	–
Trotting	6.6	–
Walking	2.4	–

Leisure Activities in METS: Sports, Exercise Classes, Games, Dancing *Continued*

	Mean	*Range*
Horseshoe Pitching	—	2–3
Hunting (Bow or Gun)		
Small game (walking, carrying light load)	—	3–7
Big game (dragging carcass, walking)	—	3–14
Judo	13.5	–
Mounting Climbing	—	5–10+
Music Playing	—	2–3
Paddleball, Racquetball	9	8–12
Rope Jumping	11	–
60–80 skips/min	9	–
120–140 skips/min	—	11–12
Running		
12 min per mile	8.7	–
11 min per mile	9.4	–
10 min per mile	10.2	–
9 min per mile	11.2	–
8 min per mile	12.5	–
7 min per mile	14.1	–
6 min per mile	16.3	–
Sailing	—	2–5
Scubadiving	—	5–10
Shuffleboard	—	2–3
Skating, Ice and Roller	—	5–8
Skiing, Snow		
Downhill	—	5–8
Crosscountry	—	6–12+
Skiing, Water	—	5–7
Sledding, Tobogganing	—	4–8
Snowshoeing	9.9	7–14
Squash	—	8–12+
Soccer	—	5–12+
Stairclimbing	—	4–8
Swimming	—	4–8+
Table Tennis	4.1	3–5
Tennis	6.5	4–9+
Volleyball	—	3–6

CORE CURRICULUM: HAZARDOUS MARINE LIFE

WILDERNESS MEDICAL SOCIETY
PAUL S. AUERBACH, M.D.

The following comprises the content of the teaching slide set entitled "Hazardous Marine Life" to be copyrighted to and distributed by the Wilderness Medical Society. No portion of this material should be reproduced without the express permission of the Wilderness Medical Society

DANGEROUS MARINE LIFE

TRAUMATOGENIC
STINGING
POISONOUS ON INGESTION
SHOCKING

BIOTOXICITY: POISONS, VENOMS AND DEFENSE

ORAL TOXINS: poisonous to eat

PARENTERAL TOXINS: venoms produced in specialized glands
injected mechanically with spines, teeth and fins

CRINOTOXINS: venoms produced in specialized glands
administered <u>without</u> the aid of "devices"
administered with slimes, gastric secretions, etc.

MARINE VENOMS

Denature membranes
Catabolize cyclic 3',5'-AMP
Degranulate mast cells
Provoke histamine release
Initiate arachidonate metabolism
Accelerate coagulopathies
Disrupt cellular transport
Impede neuronal transmission
Induce anaphylaxis and shock

GENERAL FIRST AID PRINCIPLES

Control the <u>Airway</u>
Support <u>Breathing</u>

Maintain the Circulation
Treat Anaphylaxis
Debride and Irrigate Wounds

THE MARINE ENVIRONMENT

Ocean water: a saline milieu for microbes

Sodium chloride
Sulphates
Magnesium
Potassium
Bicarbonate
Bromine
Boric acid
Strontium
Fluorine

MARINE BACTERIA

Generally halophilic
heterotrophic
motile
Gram negative rods
facultative anaerobes
highly proteolytic

BACTERIA ISOLATED FROM MARINE WATER, SEDIMENTS, MARINE ANIMALS AND MARINE-ACQUIRED WOUNDS

More than 65 species, including genera:

Acinetobacter
Actinomyces
Aeromonas
Alcaligenes
Alteromonas
Bacillus
Bacteroides
Chromobacterium
Clostridium
Deleya
Enterobacter
Erysipelothrix
Legionella
Mycobacterium
Pasteurella
Proteus
Pseudomonas

Salmonella
Staphylococcus
Streptococcus
Vibrio

MARINE BACTERIA: GROWTH IN CULTURE

Most marine bacteria which are pathogenic to man can be readily recovered on standard media

Thiosulfate-citrate-bile salts-sucrose agar is recommended for detection of marine *Vibrio* species

Antibiotic susceptibility testing requires addition of NaCl 2.3% to the broth or agar used for disk diffusion

MARINE BACTERIA: ANTIBIOTIC THERAPY I

MINOR ABRASIONS OR LACERATIONS

1. Prophylactic antibiotics not required in normal host

2. Persons who are chronically ill (e.g., diabetes, hemophilia, thalassemia), immunosuppressed (e.g., leukemia, AIDS, chemotherapy, corticosteroids) or with serious liver disease (e.g., hepatitis, cirrhosis, hemochromatosis require prophylactic antibiotics

3. Use ciprofloxacin, trimethoprim-sulfamethoxazole or tetracycline/doxycycline

MARINE BACTERIA: ANTIBIOTIC THERAPY II

INJURIES WITH HIGH INFECTION POTENTIAL

Large lacerations
Extensive burns
Deep puncture wounds, particularly hand/foot
Retained organic foreign material

1. Use prophylactic antibiotics.

2. If surgery necessary, parenteral antibiotic choices include trimethoprim-sulfamethoxazole, tobramycin/amikacin/gentamicin, or cefoperazone/cefotaxime/ceftazidime

3. If outpatient, use ciprofloxacin, trimethoprim-sulfamethoxazole or tetracycline/doxycycline

MARINE BACTERIA: ANTIBIOTIC THERAPY III

INFECTED WOUNDS: ANTICIPATE *VIBRIO* INFECTION

Rapidly progressive cellulitis and/or myositis indicates *Vibrio vulnificus* or *V. parahaemolyticus* infection

1. Culture for aerobes and anaerobes

2. Pending culture results, parenteral choices include:

    Imipenem-cilastatin
    Tobramycin/amikacin/gentamicin plus cefoperazone or piperacillin
    Chloramphenicol plus trimethoprim-sulfamethoxazole

***ERYSIPELOTHRIX RHUSOPATHIAE* INFECTION**

Classic erysipeloid appearance
Commonly follows seafood handling

Antibiotics: erythromycin, penicillin or cephalexin

MARINE ANTIVENINS

Chironex fleckeri (box-jellyfish) antivenin, from Commonwealth Serum Laboratories (CSL), Melbourne, Australia: versus *Chironex fleckeri* and *Chiropsalmus quadrigatus*

Enhydrina schistosa (beaked sea snake) and *Notechis scutatus* (terrestrial tiger snake) polyvalent antivenin, from CSL; versus most sea snakes

Notechis scutatus antivenin, from CSL: versus most sea snakes

Enhydrina schistosa monovalent antivenin, from the Haffkine Institute in Bombay, India: versus most sea snakes

Synanceja trachynis (stonefish) antivenin, from CSL: versus stonefish and some scorpionfishes

TRAUMATOGENIC MARINE HAZARDS

Sharks
Barracuda
Moray Eels
Sea Lions

HAZARDOUS MARINE LIFE SLIDE SET - WMS (AUERBACH, 1991)

SHARK LIFE AND HABITS

Found in oceans, tropical rivers and lakes
Range in size from 10 cm to over 15 m and 18,000 kg
Approximately 32 out of 350 species implicated in human attacks
50-100 annual attacks worldwide; less than 10% fatal
Exquisite ocular perception of motion and olfactory detection of blood

Most dangerous sharks:

Great white
Mako
Bull
Dusky
Hammerhead
Grey reef

GREAT WHITE SHARK: *CARCHARODON CARCHARIAS*

The most dangerous of all sharks
May attain a length of 30 feet and weight of 7000 pounds
Biting force of up to 18 tons/square inch

Commonly found in waters of southern Australia, east coast of South Africa, middle Atlantic coast of North America, and the American Pacific coast north of Point Conception, California

SHARK FEEDING AND ATTACK

Two feeding patterns: subdued (solitary) or frenzied (mob)

Some sharks can achieve 20-40 mph underwater

More than 70% of victims are bitten only once or twice

Lower teeth used first in feeding

Aggression seems to be directed at frightened or wounded victim

SHARK ATTACK

Odds of attack along North American coastline: 1/5,000,000

Danger greatest: during summer months
in recreational areas

during late afternoon and evening (feeding)
in murky/warm water
in deep channels or drop-offs
upon surface swimmers

CLINICAL ASPECTS OF SHARK BITES

Massive tissue loss
Hemorrhage
Shock
Death

Extremity bites most common
High propensity for infection because of contamination with sea water, sand, plant debris, shark teeth and mouth flora

SHARK BITE TREATMENT

Manage bleeding
Administer prophylactic antibiotics
Explore wounds in the operating room
Irrigate extensively
Close loosely around drains or pack open for delayed primary closure drains

SHARK ATTACK PREVENTION

1. Avoid shark infested water, particularly at dusk and night
2. Do not swim with domestic animals in shark waters
3. Swim in groups. Pay attention to your companions
4. Avoid turbid water, dropoffs, deep channels and sanitation waste outlets
5. Do not bleed in the water
6. Avoid shiny metal adornments and brightly colored swimwear
7. Do not carry tethered (dead or injured) fish
8. Be alert for sharks when fish act erratically
9. Do not tease or corner a shark
10. Do not panic or splash at surface

BARRACUDA LIFE AND HABITS

Distributed from Brazil north to Florida, and in the Indo-Pacific from the Red Sea to the Hawaiian Islands

Only the great barracuda *Sphyraena barracuda* has been implicated in human attacks

Grows to 10 feet and 100 pounds

HAZARDOUS MARINE LIFE SLIDE SET - WMS (AUERBACH, 1991)

Solitary swimmer with canine mouth and large, knifelike teeth

Attracted to motion and shiny objects (e.g., metal anklet)

MORAY EEL LIFE AND HABITS

Found in tropical, subtropical and temperate waters

Muscular, powerful bottom dwellers up to 3 m in length

Reside in holes, ledges and crevices

Attack when cornered or provoked

MORAY EEL BITE CLINICAL ASPECTS

Multiple puncture wounds from long, sharp, retrorse and fanglike teeth

Tenacious bite; to release the animal, it may be necessary to perform decapitation

High propensity to infection, particularly of hand

MORAY EEL BITE TREATMENT

1. Remove the animal
2. Locate all puncture wounds
3. Perform neurovascular exam
4. Debride/irrigate wounds
5. Explore wounds for retained teeth
6. DO NOT SUTURE PUNCTURE WOUNDS; large defects may be approximated loosely
7. Administer prophylactic antibiotics

SEA LION BITES

Generally mild-mannered animals, except during breeding season

Males are more aggressive, except when females defend pups

Biting and butting injuries

Propensity for post-traumatic wound infections

STINGING ANIMALS - INVERTEBRATES

Sponges
Coelenterates
Mollusks
Annelid Worms
Echinoderms
Corals

SPONGES LIFE AND HABITS

Approximately 4,000 species
Horny but elastic skeletons
Embedded spicules of silica or calcium carbonate
Can be colonized by hydrozoans, algae and coelenterates

Secondary coelenterates cause "Sponge Diver's Disease"

DERMATITIS FROM SPONGES

PRURITIC DERMATITIS (e.g., fire sponge *Tedania ignis*)

Similar to allergic (plant) dermatitis
Characterized by burning, itching, erythema, edema
Severe reaction may cause erythema multiforme, nausea, fever, chills, muscle cramps
Commonly desquamates in 10 days to 2 months

SPICULE DERMATITIS

Follows skin penetration by spicules
May introduce associated crinotoxins
Difficult to distinguish from allergic-type reaction

SPONGE ENVENOMATION TREATMENT

1. Dry the skin
2. Remove spicules using adhesive tape or facial peel
3. Apply acetic acid (2.5-5%) soaks for 10-30 minutes qid
4. Do not use corticosteroids as primary decontaminant
5. After decontamination, if inflammation is mild, use topical corticosteroids
6. If inflammation is moderate/severe, administer systemic corticosteroids
7. Administer antibiotics for cellulitis or purulent vesicles

COELENTERATE CLASSIFICATIONS

APPROXIMATELY 9,000 SPECIES, OF WHICH AT LEAST 100 ARE DANGEROUS TO HUMANS

Cnidaria: possess stinging cells (nematocysts)
Acnidaria: do not possess nematocysts

CNIDARIA: Hydrozoans (e.g., Portuguese man-of-war)
Scyphozoans (e.g., true jellyfish)
Anthozoans (e.g., anemones, soft corals)

COELENTERATE VENOM APPARATUS

Nematocyst is a stinging cell produced by cnidocyte
Millions of cnidocytes on tentacles or near the mouth
Stinging cell is venom-filled and contains a coiled thread tube
Thread tube may attain length of 200-400 microns
Exocytosis of tube stimulated by contact and hypotonic state
Velocity of ejection estimated at 2 m/sec, acceleration 40,000 g
Skin is struck by "injectors" at 2-5 lb/sq in
Upper dermis penetrated; venom diffuses into circulation

NEMATOCYST VENOM COMPONENTS

Bradykinin
Hemolysin
Serotonin
Histamine
Prostaglandins
Adenosine triphosphatase
Nucleotidases
Hyaluronidase
Alkaline protease
Acid protease
Phosphodiesterase
Acid phosphatase
Alkaline phosphatase
Fibrinolysin
Leucine aminopeptidase
RNAase
DNAase

CLINICAL ASPECTS OF HYDROID ENVENOMATION

Instantaneous mild burning, itching and urticaria

Erythematous miliary skin irritation
Rare delayed (12-14 hrs) papular, hemorrhagic or zosteriform reaction
Burning sensation in conjunctivae, oral mucous membranes

THERAPY FOR HYDROID ENVENOMATION

1. Rinse the skin with sea water or 3N saline (not with fresh water)
2. Apply brief soak of acetic acid 5% or isopropyl alcohol 40-70%
3. If inflammation is residual, apply topical corticosteroid

FIRE CORAL CLINICAL ASPECTS

Millepora species are not true corals
They are the most ubiquitous stingers in the marine environment
Numerous configurations, so adverse encounters are frequent

Immediate intense burning, with occasional central radiation
Painful pruritus, with large wheals (worsened by rubbing)
Maximum discomfort in 30-60 minutes
Untreated, rash flattens over 12-24 hours
Total resolution in 3-7 days, with residual hyperpigmentation
Regional lymphadenopathy in severe cases

THERAPY FOR FIRE CORAL STING

1. Rinse the skin with sea water or 3N saline (not with fresh water)
2. Apply 15 min soak of acetic acid 5% or isopropyl alcohol 40-70%
3. If inflammation is residual, apply topical corticosteroid
4. If rash is indolent or lymph nodes are inflamed, administer 2 week tapering course of oral corticosteroids or parenteral corticosteroid injection

PORTUGUESE MAN-OF-WAR CLINICAL SYNDROME

1. Immediate pain, itching, erythema, "tentacle prints"
2. Rare anaphylaxis
3. Weakness, dizziness, nausea, vomiting, diarrhea, abdominal pain
4. With large surface area involvement, may develop headache, mononeuritis, delirium, seizures, hemolysis, hypotension, arrhythmias, bronchospasm, pulmonary edema, erythema multiforme, renal failure, parasympathetic dysautonomia, coma
5. Persistent or recurrent skin lesions with minimal contact suggest immune sensitization

SCYPHOZOAN MEDUSAE AND JELLYFISH

Armed with some of the most venoms in existence
Most highly dangerous species are located in Indo-Pacific waters

Examples include:

Chironex fleckeri
Cyanea capillata
Chiropsalmus quadrigatus
Pelagia noctiluca
Carukia barnesi
Tamoya haplonema
Chrysaora quinquecirrha

CLINICAL ASPECTS OF SCYPHOZOAN ENVENOMATION

Similar to Portuguese man-of-war, with markedly increased severity

Multiple stings from the box-jellyfish (*Chironex fleckeri*) with tentacle contact in excess of 6-7 m can cause death from cardiorespiratory arrest in 60 seconds

Tentacle prints may have frosted, cross-hatched appearance with prominent blistering

Skin wounds frequently become necrotic despite topical therapy

CLINICAL ASPECTS OF ANEMONE ENVENOMATION

Similar to Portuguese man-of-war, but with more pronounced skin urticaria or ischemic appearance

Hemorrhagic perimeter to sting sites common, followed by ulcerations, intense local vesiculation with anemones *Sagartia, Actinia, Anemonia, Actinodendron, Triactis*

Systemic effects are rare, and include fever, chills, somnolence, malaise, weakness, nausea, vomiting

DERMATOLOGICAL THERAPY FOR COELENTERATE ENVENOMATION - I

1. Rinse the wound with sea water or 3N saline. <u>DO NOT RINSE WITH FRESHWATER</u>. DO NOT RUB THE WOUND. Wear protective gloves (double glove).
2. Remove gross tentacle fragments with forceps.
3. Apply acetic acid 5% as topical decontaminant (ABSOLUTE with *Chironex fleckeri*) for at least 30 min. Alternatives:

isopropyl alcohol 40-70%, dilute ammonium hydroxide, papaya latex (juice). Do not use organic solvents.
4. Do not immerse in hot water.

DERMATOLOGICAL THERAPY FOR COELENTERATE ENVENOMATION - II

5. After decontamination, remove nematocysts. Apply shaving cream or paste of baking soda and shave with sharpest edge available.
6. Apply topical corticosteroids. Use local anesthetics sparingly.
7. If inflammation is severe, use oral or parenteral corticosteroids.
8. Administer standard anti-tetanus prophylaxis.
9. Watch closely for development of wound infection.
10. If sting is from *Chironex fleckeri* and skin reaction is severe, administer antivenin.

SYSTEMIC THERAPY FOR COELENTERATE ENVENOMATION

1. Anticipate and manage anaphylaxis.
2. Manage hypotension with crystalloid infusions.
3. Manage hypertension: consider alpha-adrenergic blockers.
4. Observe for and treat arrhythmias. Obtain an EKG.
5. Identify and manage hemolysis and renal failure with urine alkalinization and mannitol infusion.
6. If sting is from *Chironex fleckeri* or *Chiropsalmus*, administer antivenin for any reaction that is more severe than minor, particularly if decompensation is rapid.
7. Control pain with narcotics or NSAIDs.

MOLLUSKS HAZARDOUS TO MAN

Phylum Mollusca: 45,000 species
Unsegmented, soft-bodied invertebrates, often with calcareous shell
Hazardous to humans:
Pelecypods (e.g., scallops, oysters, clams, mussels)
Gastropods (e.g., snails, slugs)
Cephalopods (e.g., squids, octopuses, cuttlefish)

CONE SHELLS VENOM APPARATUS AND DANGER

At least 18 species implicated in human envenomations

Conus geographus, C. aulicus, C. gloria-maris, C. omaria, C.

striatus, C. textile, C. tulipa are worst offenders

Inject venom with detachable, dartlike radular tooth used as a "harpoon"
Proboscis which launches dart can extend all the way back to the thick crown of the shell

CONE SHELL ENVENOMATION

Mild stings resemble wasp sting

Initial symptoms: localized wound ischemia and numbness
burning and stinging
paresthesias
dysphagia
weakness
syncope

Severe reaction: generalized muscular paralysis
respiratory distress
cardiac failure
coma
cerebral edema
disseminated intravascular coagulopathy

THERAPY FOR CONE SHELL ENVENOMATION

1. Apply pressure immobilization technique
2. Observe for 2-4 hours
3. Manage hypotension with crystalloid infusion
4. Administer oxygen
5. Be prepared to manage respiratory distress
6. Anticipate coagulopathy

PRESSURE IMMOBILIZATION TECHNIQUE FOR VENOM SEQUESTRATION

1. Place a cloth or gauze pad of approximate dimensions 6-8 cm x 6-8 cm x 2-3 cm (thickness) directly over bite site (optional)
2. Wrap the extremity with an elasticized bandage at lymphatic-venous occlusive tension
3. Distal fingertips and toes should be pink; arterial pulsations should be present (proper capillary refill)
4. Keep bandages in place until the victim is brought to medical attention
5. Anticipate swelling and loosen bandages if arterial circulation is compromised

HAZARDOUS MARINE LIFE SLIDE SET - WMS (AUERBACH, 1991)

BLUE-RINGED OCTOPUS

Found throughout the Indo-Pacific, with greatest concentration off northern Australia

Toxin contains tetrodotoxin, a potent neuromuscular blocking agent

Bite is painless, usually on an extremity
Site is initially numb, followed by discomfort after 5-10 minutes
Variable local urticaria
After 30 minutes, site is red, warm, swollen and pruritic
Rarely hemorrhagic at bite site
Within 10-15 minutes, onset of progressive paralysis
Other symptoms include:
paresthesias, diplopia, blurred vision, aphonia, dysphagia, ataxia, myoclonus, weakness, nausea, vomiting, peripheral neuropathy

THERAPY FOR BLUE-RINGED OCTOPUS ENVENOMATION

REMEMBER, THE VICTIM MAY BE AWAKE AND MENTATING IF OXYGENATION IS ADEQUATE, EVEN IN THE FACE OF FLACCID PARALYSIS

1. If practicable by virtue of location, apply pressure-immobilization technique
2. Provide prompt respiratory assistance
3. Administer oxygen
4. Establish intravenous access
5. Transport the victim rapidly to a critical care facility
6. Do not utilize wound excision

BRISTLEWORM ENVENOMATION

Chitinous bristles are attached to parapodia used for locomotion
When worm is stimulated, bristles are erected
Bristles enter skin like mini cactus spines

CLINICAL PRESENTATION

Burning sensation
Red, geographical rash with flat or raised borders
Urticaria is common; necrosis is rare
Inflammation can last for up to 10 days with severe itching

THERAPY FOR BRISTLEWORM ENVENOMATION

1. Remove all large visible bristles with forceps
2. Dry the skin
3. Remove embedded bristles with adhesive tape or facial peel
4. Apply acetic acid 2.5-5% or isopropyl alcohol 40-70%
5. Apply topical corticosteroids
6. If inflammation and discomfort are severe, administer oral or parenteral corticosteroids

ECHINODERMS

Starfish
Sea urchins
Sea cucumbers

CROWN-OF-THORNS STARFISH LIFE AND HABITS

Found in the coral reef communities of the Great Barrier reef, throughout the Pacific and Indian Ocean, in the Red Sea, and in the eastern Pacific from the Galapagos to the Gulf of California

Slimy venom produced in glands within or under the epidermis
Attains 25-35 cm in diameter
7-23 arms, with spines of up to 6 cm
Magnesium calcite spine can easily penetrate a diving glove or neoprene suit

CLINICAL ASPECTS OF STARFISH ENVENOMATION

Immediate intense pain, bleeding and edema
Wound may be dusky or discolored

Multiple punctures lead to systemic effects:

- Paresthesias
- Nausea, vomiting
- Lymphadenopathy
- Local muscle paralysis
- Delayed granuloma (foreign body)

THERAPY FOR STARFISH ENVENOMATION

1. Immerse the wound(s) into nonscalding hot water to tolerance (113° F or 45° C) for 30-90 minutes or until there is significant pain relief
2. Use local anesthetic for additional pain control

3. Debride and explore to remove foreign material
4. Obtain X-ray to visualize radiopaque foreign body
5. Remove any residual foreign body
6. Manage dermatitis with topical antipruritics
7. If puncture wound is more than superficial, consider prophylactic antibiotics

SEA URCHIN VENOM AND VENOM APPARATUS

Free-living echinoderms with egg-shaped, globular or flattened bodies

Covered by regularly arranged spines and triple-jawed pedicellariae (seizing and envenoming organs)

Spines can be brittle, hollow, sharp and venom-filled

Most persons envenomed when they step upon or brush against urchin

SEA URCHIN ENVENOMATION CLINICAL ASPECTS

Intense local pain, radiating deep into muscle
Rapid erythema and swelling
Purple discoloration may reflect spine dye or retained fragment
Entry into joint induces severe synovitis
Metacarpal or MCP proximity can cause distal fusiform digit swelling

Multiple punctures or pedicellarial envenomation can cause: nausea, vomiting, paresthesias, local paralysis, syncope, hypotension and respiratory distress

THERAPY FOR SEA URCHIN ENVENOMATION

1. Extract any obvious spine fragments. DO NOT CRUSH THE SPINES WITHIN THE SOFT TISSUES.
2. If pedicellariae are attached, remove with a razor edge using shaving cream and hot water.
3. Immerse the wound(s) into nonscalding hot water to tolerance (113° F or 45° C) for 30-90 minutes or until there is significant pain relief.
4. Use local anesthetic for additional pain control. To locate additional puncture sites, infiltrate with lidocaine without epinephrine and observe egress of fluid.
5. Debride and explore to remove foreign material.
6. Obtain X-ray to visualize radiopaque foreign bodies.

HAZARDOUS MARINE LIFE SLIDE SET - WMS (AUERBACH, 1991)

7. Operate to remove all spines near joints or significant neurovascular structures.
8. Manage dermatitis with topical antipruritics.
9. If puncture wound is more than superficial, consider prophylactic antibiotics.

CLINICAL ASPECTS OF SEA CUCUMBER ENVENOMATION

Tentacular organs of Cuvier produce "holothurin", a cantharidin-like substance

Contact with tentacles or fragments produces dermatitis
Corneal inflammation if fragments enter the eyes
Severe reaction may lead to blindness

Sea cucumbers may eat nematocysts and excrete coelenterate venom

Holothurin is a potent cardiac glycoside

THERAPY FOR SEA CUCUMBER ENVENOMATION

1. If skin is involved, decontaminate with acetic acid 5% or isopropyl alcohol 40-70%
2. If eye is involved, anesthetize with proparacaine HCl 0.5%, then irrigate with 250 ml normal saline
3. Stain the cornea and inspect for corneal abrasion/ulceration
4. Treat inflammatory keratitis with cycloplegic and mydriatic agents. Topical corticosteroid ophthalmic solution may be helpful

CORAL CUTS AND POISONING CLINICAL ASPECTS

Initial reaction is stinging pain, erythema and pruritus

"CORAL POISONING":

indolent wound associated with poor healing, persistent exudate, regional lymphadenopathy, reactive bursitis, malaise, low grade fever

THERAPY FOR CORAL CUTS

1. Vigorously irrigate the wound with fresh or disinfected water or normal saline
2. Scrub the wound with nontoxic soap or antiseptic solution
3. Repeat wound irrigation
4. Debride macerated edges and explore to remove foreign material
5. If the wound is painful in a manner that suggests coelenterate envenomation, consider decontamination with acetic acid 2.5% or isopropyl alcohol 20% in an extremely brief application, followed by irrigation
6. DO NOT SUTURE WOUNDS CLOSED IF AT ALL POSSIBLE. If edges must be approximated, use surgical adhesive strips
7. Debride daily, utilizing wet-to-dry dressings or topical antiseptics (nonadherent dressings)
8. Administer antibiotics at earliest signs of infection

STINGING ANIMALS - VERTEBRATES

Stingrays
Scorpionfishes

STINGRAYS LIFE AND HABITS

Most commonly incriminated group of fishes involved in human envenomations

11 species found in U.S. coastal waters

One to four venomous stings on the dorsum of caudal appendage
Spines are retroserrate vasodentine with associated venom glands

Injuries are commonly to lower extremity with both a laceration and envenomation

Integumentary sheath is ruptured, releasing venom into wound

STINGRAY ENVENOMATION CLINICAL ASPECTS

Immediate intense pain as puncture occurs, with central radiation
Spine tip(s) may break off and remain in the wound(s)
Thorax or abdomen may be penetrated
Wound is edematous and hemorrhagic, occasionally dusky or mottled
Rapid fat and muscle hemorrhage and necrosis
Systemic effects include:
weakness, nausea, vomiting, diarrhea, diaphoresis, vertigo, headache, seizures, syncope, fasciculations, paralysis,

hypotension, arrhythmias and death

THERAPY FOR STINGRAY ENVENOMATION

1. Extract any obvious spines, spine fragments, or pieces of integumentary sheath.
2. Immerse the wound(s) into nonscalding hot water to tolerance (113° F or 45° C) for 30-90 minutes or until there is significant pain relief.
4. Use local anesthetic infiltration (without epinephrine) for additional pain control. If necessary, perform a regional nerve block.
5. Debride and explore to remove foreign material. Irrigate with warmed saline.
6. Obtain X-ray to visualize radiopaque foreign bodies.
7. Operate to remove all retained foreign matter.
8. Do not suture wounds closed if at all possible. Leave open for delayed primary closure.
9. Administer prophylactic antibiotics.
10. Observe all victims for at least 4 hours for onset of systemic effects.

CATEGORIES OF SCORPIONFISHES

Genus *Pterois*:	zebrafish, lionfish, tigerfish, turkeyfish, scorpionfish, firefish
Genus *Scorpaena*:	scorpionfish, bullrout, sculpin
Genus *Synanceja*:	stonefish, stargazer, warty ghoul

SCORPIONFISHES LIFE AND HABITS

Venom organs: 12-13 (of 18) dorsal, 2 pelvic and 3 anal spines

Pectoral spines are not venomous

Each spine is covered with integumentary sheath and carries associated venom glands

Animals are often extremely well camouflaged and difficult to locate, even for experienced divers

SCORPIONFISH ENVENOMATION CLINICAL ASPECTS

HAZARDOUS MARINE LIFE SLIDE SET - WMS (AUERBACH, 1991)

Immediate pain from puncture wounds
Pain peaks at 60-90 minutes and persists for up to 12 hours
Wound initially ischemic, then cyanotic, then red, warm and swollen
Vesicles may form
With severe sting, may have tissue necrosis and sloughing
Indolent wounds require months to heal

Systemic effects include:
headache, tremors, maculopapular skin rash, nausea, vomiting, diarrhea, abdominal pain, diaphoresis, pallor, delirium, seizures, paralysis, lymphangitis, arthritis, fever, hypertension, respiratory distress, arrhythmias, congestive heart failure, pericarditis, hypotension, syncope and death

THERAPY FOR SCORPIONFISH ENVENOMATION

1. Extract any obvious spines, spine fragments, or pieces of integumentary sheath.
2. Immerse the wound(s) into nonscalding hot water to tolerance (113° F or 45° C) for 30-90 minutes or until there is significant pain relief.
4. Use local anesthetic infiltration (without epinephrine) for additional pain control. If necessary, perform a regional nerve block. Localize all puncture wounds with anesthetic infiltration, observing for egress of fluid.
5. Debride and explore to remove foreign material. Irrigate with warmed saline.
6. Obtain X-ray to visualize radiopaque foreign bodies.
7. Operate to remove all retained foreign matter.
8. Do not suture wounds closed if at all possible. Leave open for delayed primary closure.
9. Administer prophylactic antibiotics.
10. Observe all victims for at least 4 hours for onset of systemic effects.

OTHER MARINE VERTEBRATES THAT STING AND CAUSE INJURIES SIMILAR TO SCORPIONFISHES

Weeverfishes
Catfishes
Dragonfishes
Venomous (spined) sharks
Ratfishes
Toadfishes
Rabbitfishes
Stargazers
Squirrelfishes
Sea robins

HAZARDOUS MARINE LIFE SLIDE SET - WMS (AUERBACH, 1991)

Flying gurnards
Goosefishes

SEA SNAKES LIFE AND HABITS

The most abundant reptiles on Earth
At least 52 species, all venomous

Distributed in tropical and warm temperate Pacific and Indian Oceans

There are no indigenous sea snakes in the Atlantic Ocean or Caribbean Sea

Carry 2-4 hollow maxillary fangs with associated venom glands

Venom is a neurotoxin, with additional hemolysins and myotoxic compounds (phospholipase A)

CLINICAL ASPECTS OF SEA SNAKE BITES

DIAGNOSIS OF SEA SNAKE BITE IS BASED UPON:

1. Location of incident. One must be in the water or handling a sea snake to have been bitten.
2. Absence of pain. Most bites are initially painless.
3. Fang marks. These are multiple, hypodermic-like and number up to 20.
4. Identification of the snake.
5. Characteristic symptoms: euphoria, malaise, anxiety, slurred speech, facial paralysis, parotid gland swelling, myalgia, paralysis, arthralgia, trismus, blurred vision, dysphagia, drowsiness, vomiting, ptosis. Symptoms occur within 6-8 hours.

6. With significant envenomation, demise is rapid within first 2-3 hours. Symptoms and signs include myoglobinuria, paralysis, respiratory distress, aspiration, hyperkalemia, acute renal failure, myonecrosis, hypotension.

THERAPY FOR SEA SNAKE BITES

1. Apply the Extractor device if within the first 3 minutes post bite.
2. Apply the pressure immobilization technique for venom sequestration.
3. Immobilize the affected limb.
4. Obtain intravenous access in 2 sites.
5. With any evidence of envenomation, administer antivenin.

6. Monitor for hemoglobinuria or myoglobinuria. If evident, promote diuresis with mannitol infusion and alkalinize urine with bicarbonate.
7. Administer oxygen. Observe closely for signs of respiratory failure. Be prepared to provide mechanical ventilatory assistance.

ESSENTIALS OF DIVING MEDICINE

by
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Synopsis: The aquatic environment presents numerous hazards for the underwater worker or sportsman. This presentation will familiarize practitioners with the environmental hazards confronting scuba divers, focusing especially on those conditions due to increased atmospheric pressure. After attending this lecture one should have an increased understanding of diving, in general, and a better understanding of the pathophysiology, clinical manifestations and treatment of barotrauma, dysbaric air embolism and decompression sickness, in particular.

LECTURE OUTLINE

I. A Brief Overview of Diving

II. Basic Physics and Physiology of Diving

- A. Pressure and its measurement
- B. Gas laws
- C. Cardiorespiratory physiology of submersion and hyperbaria

III. Primary Diving Medicine Problems

- A. Environmental exposures
- B. Problems related to the breathing medium
- C. Dysbarism
- D. Hazardous marine life
- E. Miscellaneous

IV. Barotrauma - local injury resulting from inadequate pressure equalization between air-containing cavities and the ambient atmosphere; by far the most common dysbaric problem of divers

A. Otolaryngologic

B. Gastrointestinal

C. Pulmonary

D. Other

V. Dysbaric Air Embolism

A. Pathophysiology

B. Clinical manifestations

C. Treatment

VI. Decompression Sickness - a multisystem disorder resulting from the liberation of inert gas from solution and the resultant formation of gas bubbles in tissue

A. Pathophysiology

B. Clinical manifestations

C. Treatment

VII. Differential Diagnosis of Diving Accidents

A. Diving accident history

B. Sources of consultation

1. Diving Alert Network (DAN) - 919:684-8111

2. U.S. Navy Experimental Diving Unit - 904:234-4351

3. Naval Medical Research Institute - 202:295-1839

2. Local and regional sources

Diving Medicine

*Kenneth W. Kizer, M.D., M.P.H.**

The oceans are the last frontier on earth, and in recent years there has been a virtual explosion in the number of divers exploring their depths. It is estimated that there are about three million recreational scuba divers in the United States, and more than 200,000 new sport divers are trained each year.[45] In addition, there are many thousands of commercial, scientific and military divers.

The medical problems of diving are primarily due to the intrinsic hazards of the aquatic environment and the breathing of compressed gas, that is, air or other gas mixtures at more than one atmosphere of pressure. A useful way of categorizing the many disparate medical problems related to diving is shown in Table 1. Since it is beyond the scope of this article to review all of these problems, this discussion will focus on the several pressure-related syndromes that are peculiar to diving and collectively known as dysbarism. Emergency physicians should be familiar with the management of these problems because the ever-increasing number of divers and the burgeoning diving vacation industry, which accounts for many injured divers presenting to emergency departments far removed from the diving site, make these problems increasingly common.

PHYSICAL PRINCIPLES

Human beings did not evolve for a marine existence, and, indeed, they are not well adapted for functioning in the aquatic environment. Consequently, divers encounter many adverse physical conditions, such as cold, wetness, changes in light and sound conduction, increased density of the surrounding environment, and increased atmospheric pressure. Of these, pressure is by far the single most important environmental factor in

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Portions of this article were taken from Kizer KW: Management of dysbaric diving casualties. Emerg Med Clin North Am. 1:659–670, 1983.

Table 1. *Medical Problems of Divers**

Environmental Exposure Problems
Motion sickness
Near drowning and other immersion syndromes
Hypothermia and heat illness
Sunburn
Phototoxic and photoallergic reactions
Irritant and other dermatitides
Infectious diseases
Physical trauma
Dysbarism
Barotrauma
Dysbaric air embolism
Decompression sickness
Breathing Gas-Related Problems
Inert gas narcosis
Hypoxia
Oxygen toxicity
Hypercapnia
Carbon monoxide poisoning
Nitrogen oxide toxicity
High pressure nervous syndrome
Cutaneous isobaric counterdiffusion
Lipoid pneumonitis
Hazardous Marine Life
Miscellaneous
Hyperventilation
Dysbaric osteonecrosis
Compression arthralgia
Hyperbaric cephalgia
Hearing loss
Carotogenic blackout
Panic and other psychological problems

*From Kizer KW: Management of dysbaric diving casualties. Emerg Med Clin North Am, *1*:659–670, 1983.

diving, with its effects either directly or indirectly accounting for the majority of serious diving medical problems. Therefore, having an understanding of basic pressure physics and physiology is essential to understanding diving diseases.

Pressure

Pressure is defined as force per unit area and is measured in a number of different units (Table 2). Since we are adapted to standard terrestrial atmospheric conditions, we usually do not think about the density and pressure of the air we breath. Actually, though, the weight of air at sea level is equal to 14.7 pounds per square inch (psi) or one atmosphere absolute (ATA).

When a diver goes under water, the pressure increases because of the weight of the water, and since water is much more dense than air, large changes in pressure will accompany small fluctuations in depth. Thus, as shown in Figure 1, at a depth of 33 feet of sea water (fsw) the pressure is 2 ATA and at 165 fsw it is 6 ATA. In addition, it should be clear from the Figure that the proportionate change in pressure per unit depth change is

Table 2. *Units of Pressure Measurement**

1 atmosphere absolute	= 33 feet salt water (fsw)†
	= 34 feet fresh water (ffw)
	= 5.5 fathoms of sea water
	= 14.7 pounds per square inch (psi)
	= 760 mm Hg
	= 29.9 in Hg
	= 1.033 kg/cm^2
	= 1.013 bar
	= 10.06 meters
	= 0 atmosphere gauge

*From Kizer KW: Management of dysbaric diving casualties. Emerg Med Clin North Am, *1*:659–670, 1983.

†1 fsw = 0.445 psi = 0.0303 atmospheres

greatest near the surface. Most scuba diving is done at pressures less than 6 ATA.

Since the tissues of the body are composed mostly of water, which is not compressible, they are not significantly affected by pressure changes. However, gases are compressible and, consequently, the gas-filled organs of the body are directly affected by changes in ambient pressure.

Gas Laws

Three gas laws are fundamental to diving medicine and must be understood by persons treating dysbaric diving casualties.

The first of these gas laws is Boyle's law, which states that the volume of a gas is inversely proportional to its pressure at a constant temperature. This is depicted by the following equation:

$$PV = k,$$

where P is pressure, V is volume, and k is a constant. As shown in Figure

	Depth (fsw)	Gauge Pressure (atmos)	Absolute Pressure (atmos)	Gas Volume	Bubble Diameter
air	0	0	1	100%	100%
sea water	33	1	2	50%	79%
	66	2	3	33%	69%
	99	3	4	25%	63%
	132	4	5	20%	58%
	165	5	6	17%	54%

Figure 1. Pressure-volume relationships according to Boyle's law. Gauge pressure is always one atmosphere less than absolute pressure. Bubble diameter is probably a more important consideration than volume when assessing the ability of recompression to restore circulation to a gas embolized blood vessel. (*From* Kizer KW: Management of dysbaric diving casualties. Emerg Med Clin North Am, *1*:661, 1983.)

1, when the pressure is doubled, the volume of a unit of gas is halved and the converse is also true. Boyle's law explains the basic mechanism of all types of barotrauma.

The second key gas law is Dalton's law. According to this law, the pressure exerted by each gas in a mixture of gases is the same as each gas would exert if it alone occupied the same volume, or, alternatively, the total pressure of a mixture of gases is equal to the sum of the partial pressures of the component gases. This can be mathematically stated as follows:

$$P(t) = P(O_2) + P(N_2) + P(x)$$

where P(t) is the total pressure, $P(O_2)$ is the partial pressure of oxygen, $P(N_2)$ is the partial pressure of nitrogen and P(x) is the partial pressure of the remaining gases in the mixture. Since the biologic effects of a gas depend on partial pressure, which changes in proportion to ambient pressure even though concentration remains constant, this law is fundamental to the understanding of decompression sickness and other breathing gas–related problems.

Henry's law is the third basic gas law of diving physiology. It states that the amount of gas dissolved in a given volume of fluid is proportional to the pressure of the gas with which it is in equilibrium. This can be depicted mathematically as follows:

$$\%Y = [P(Y)/P(t)] \times 100$$

where %Y is the amount of gas dissolved in a liquid, P(Y) is the partial pressure of gas Y, and P(t) is the total atmospheric pressure. This law explains why, for example, more inert gas, nitrogen, dissolves in the diver's body as ambient pressure is increased with descent and, conversely, is released from tissue with ascent.

The pressure-related diving syndromes can be divided into problems caused by the mechanical effects of pressure (that is, barotrauma) and problems caused by breathing gases at elevated partial pressures (that is, gas toxicities and decompression sickness). Fundamentally, these can be viewed as the direct and indirect effects of pressure, respectively.

DIRECT EFFECTS OF PRESSURE

The gas pressure in the air-filled spaces of the body is normally in equilibrium with the environment; however, if something should obstruct the various portals of gas exchange for these spaces, a pressure disequilibrium will develop. If the air-filled space is not collapsible, the pressure imbalance will cause tissue distortion, vascular engorgement and mucosal edema, hemorrhage, and other tissue damage that is collectively known as barotrauma. Overall, barotrauma is the most common affliction of divers, and for the purposes of discussion it can be divided according to whether it occurs during descent or ascent.

Barotrauma of Descent

Barotrauma of descent, or "squeeze" as it is known in common diving parlance, results from the compression of gas in enclosed spaces as ambient pressure increases with descent underwater.

Otolaryngologic Barotrauma. Aural barotrauma is the most common type of barotrauma and is a major cause of morbidity among divers, being experienced by essentially all divers at one time or another.[12] Three types of aural barotrauma may occur, depending on which part of the ear is affected, either singly or in combination.

Ear Canal Squeeze. The first type of aural barotrauma involves the external auditory canal and is generally referred to as external ear squeeze. The external ear canal normally communicates with the environment; consequently, the air in the canal is replaced by water when submerged. However, if the external ear canal is occluded by cerumen, ear plugs, exostoses, or other material, water entry is prevented, and compression of the enclosed air with descent will be compensated for by outward bulging of the tympanic membrane and swelling or hemorrhage of the soft tissues of the canal. This is typically manifested by pain or bloody otorrhea, or both.

Examination of the ear canal may reveal petechiae, blood-filled blebs, and erythema or rupture of the tympanic membrane. As with other types of ear canal injury, treatment for this type of squeeze involves keeping the canal dry, prohibition of swimming or diving until healed, and, sometimes, antibiotics and analgesics.

Middle Ear Squeeze. The overall most common type of aural barotrauma is middle ear squeeze or "barotitis media." This results from a failure to equalize the middle ear and ambient environmental pressures because of occlusion or dysfunction of the eustachian tube.

The eustachian tubes normally open and allow equalization of middle ear pressure when the pressure differential between the middle ear and the phyarynx reaches about 20 mm Hg. This can be facilitated by yawning, swallowing, or utilizing various autoinflation techniques (for example, the Valsalva or Frenzel maneuver). If middle ear pressure equalization is not achieved, the diver will notice discomfort or pain when the pressure differential across the tympanic membrane reaches 100 to 150 mm Hg or, roughly, when there has been a 20 per cent reduction in middle ear gas volume. As the pressure differential further increases, mucosal engorgement and edema, hemorrhage, and inward bulging of the tympanic membrane develop. If descent is not halted, these things will not be able to compensate for the gas volume contraction, and the tympanic membrane will rupture. Fortunately, this degree of injury is not common.

A number of things may impair eustachian tube function (for example, mucosal congestion secondary to upper respiratory infection, allergies, or smoking, mucosal polyps; excessively vigorous autoinflation maneuvers; and previous maxillofacial trauma), and divers with such conditions are at increased risk of aural barotrauma.

As would be expected from the way that pressure changes with depth (see Fig. 1), most middle ear squeezes occur near the surface. Divers

having this problem usually complain of ear fullness or pain, which is typically severe enough that it causes the diver to abort the dive. If not, it will continue to worsen until the ear drum ruptures, at which time the diver may feel bubbles escaping from the ear and experience disorientation, nausea, and vertigo secondary to the caloric stimulation of cold water entering the middle ear. This sequence of events has accounted for numerous cases of panic and near drowning.

The otoscopic appearance of the tympanic membrane in cases of middle ear squeeze varies according to the severity of the injury and can be graded according to the amount of hemorrhage in the tympanic membrane,[12] running from grade 0 (symptoms only) to grade 5 (gross hemorrhage and tympanic membrane rupture). Utilizing this grading scheme (Fig. 2) facilitates communication when describing these injuries. Physical examination may also disclose blood around the nose or mouth and a mild conductive hearing loss.

Treatment of middle ear squeeze requires abstinence from diving until the condition has resolved, along with the use of decongestants and, if there is an allergic component to the eustachian tube dysfunction, antihistamines. Combining an oral decongestant preparation with a long-acting topical nasal spray is usually most effective, at least for the first two or three days. Antibiotics should be used when there is a tympanic membrane rupture, a pre-existing infection, or the dive was in polluted waters. Clearly, no diving should be done until a perforated ear drum has healed. Oral analgesics or topical aural anesthetics (for example, Auralgan otic solution) may be needed initially, although ear drops should not be used when there is a tympanic membrane perforation. Ideally, an audiogram should be obtained in anyone complaining of decreased hearing, and serial audiograms should be obtained in patients having hearing loss.

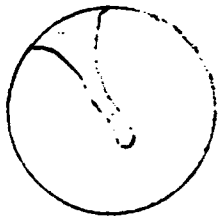
Most middle ear squeezes will resolve without complication in three to seven days; however, prevention of the problem is clearly preferable. This usually can be achieved if the individual refrains from diving when unable to easily equalize pressure in the ears and heeds warning signs of ear pain.

Inner Ear Squeeze. The third type of aural barotrauma of descent affects the inner ear and involves rupture of the round or oval windows, resulting in the development of a perilymph fistula (PLF). This essentially always involves the round window. Although more than one set of circumstances may result in a perilymph fistula, the basic requisite condition is the sudden development of markedly different pressures between the middle ear and the inner ear.[12, 17-19, 23, 37] This can occur after a rapid descent without adequately equilibrating the middle ear, resulting in excessive inward movement of the ear drum and stapes so that the oval window is imploded. Conversely, a forceful Valsalva maneuver may abruptly raise the cerebrospinal fluid pressure (which is transmitted to the perilymph) so much so that it causes an explosive outward rupture of either the round or the oval window. Perilymph fistula may also develop during ascent. Whatever the specific circumstances, though, development of a labyrinthine fistula and consequent leakage of perilymph fluid will result in irreversible

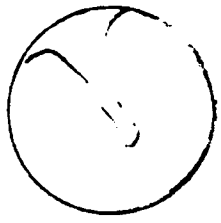
Grade 0. Symptoms only



Grade 1. Erythema over malleus



Grade 2. Erythema of malleus plus mild hemorrhage of tympanic membrane



Grade 3. Gross hemorrhage throughout tympanic membrane



Grade 4. Free blood in middle ear



Grade 5. Free blood in the middle ear plus perforation of the tympanic membrane



Figure 2. Grading scheme for middle ear barotrauma is shown. (Adapted from Edmonds et al.)

cochlear damage if the condition is not recognized and treated without delay.

Patients with inner ear barotrauma may complain of "blockage" in the affected ear, tinnitus, vertigo, disorientation, ataxia, or hearing loss, but the classic triad of symptoms is tinnitus, vertigo, and deafness. Examination typically reveals findings of middle ear barotrauma, sensorineural hearing loss, and vestibular dysfunction.

Management of perilymph fistula is somewhat controversial. Some authorities prefer a trial of bed rest and symptomatic measures for vertigo before attempting surgical correction, whereas others advocate prompt exploration and repair of the fistula. Whichever route is taken, though, there is unanimity that an individual should not ever attempt further diving after suffering a perilymph fistula.

Sinus Squeeze. As with the ears, the paranasal sinuses may fail to equalize pressure during descent, resulting in a sensation of fullness or pressure in the affected sinus, pain, or hemorrhage. Again, predisposing conditions for barosinusitis include upper respiratory infections, sinusitis, nasal polyps, or anything else that impairs the free flow of air from sinus cavity to nose. The maxillary and frontal sinuses are most often affected.

Sinus squeeze is treated much the same as middle ear squeeze, although antibiotics are probably indicated when the frontal sinuses are involved.

Lung Squeeze. When breath-holding divers descend below the depth at which their total lung volume is reduced to less than residual volume, they may develop lung squeeze. This usually occurs at depths greater than 120 to 130 fsw. As occurs in other types of barotrauma of descent, the underventilated lung air spaces fill with tissue fluids and blood in an attempt to relieve the negative pressure. Clinical manifestations include chest pain, cough, hemoptysis, dyspnea, and other signs of acute pulmonary edema. Treatment includes administration of 100 per cent oxygen, fluid replacement, and other supportive measures as clinically indicated. Owing to the intrinsic lung injury and consequent potential for gas embolism, positive-pressure breathing, for example, positive end-expiratory pressure (PEEP) or continuous positive airway pressure (CPAP) should be avoided if at all possible. Overall, though, very few divers attempt to free dive to depths likely to cause lung squeeze, and its occurrence is rare.

Other Types of Squeeze. Barotrauma of descent can also affect any other gas space that does not equilibrate with ambient pressure. For example, conjunctival and scleral hemorrhage may result from wearing goggles when diving. The same thing can happen if the diver fails to exhale into his mask during descent, resulting in telltale erythema, ecchymosis, and petechiae of the part of the face enclosed by the facemask, that is "facemask squeeze." Similarly, if an area of skin is tightly enclosed by a dry diving suit, a "suit squeeze" may occur. Although the appearance of these injuries may be spectacular, no special treatment is usually required.

Barotrauma of Ascent

If there has been adequate equilibration of the pressure in the body's air-filled spaces during descent, the gas in those spaces will expand

according to Boyle's law as ambient pressure decreases with ascent. The resulting excess gas is normally vented to the atmosphere. However, if this is prevented by obstruction of the air passages, the expanding gases will distend the tissues surrounding them; the resulting damage is known as barotrauma of ascent and is the reverse process of squeeze.

Otolaryngologic Barotrauma of Ascent. It is unusual for the ears or sinuses to be affected by barotrauma of ascent, since impediment of air egress is unlikely if pressure equalization can be achieved with descent. However, middle ear barotrauma of ascent, or "reverse squeeze," as it is usually called, can occur in divers having upper respiratory congestion that is treated with a short-acting topical nasal spray, the vasoconstrictive effect of which wears off while the diver is submerged. Similarly, "alternobaric vertigo" (ABV) resulting from asymmetric vestibular stimulation due to unequal middle ear pressure may occur during ascent.[7, 16, 34, 36] Although usually only transient, alternobaric vertigo may be severe enough to cause panic, and, occasionally, symptoms may last for several hours or days after a dive.

Gastrointestinal Barotrauma of Ascent. This type of barotrauma, which is also known as aerogastralgia or "gas in the gut,"[26, 35] is caused by expansion of intraluminal bowel gas as ambient pressure is decreased during ascent. It occurs most commonly in novice scuba divers, who seem more prone to aerophagia; other predisposing conditions include repeated performance of the Valsalva maneuver in the head down position (which forces air into the stomach), drinking carbonated beverages or eating a heavy meal before diving (expecially one containing legumes or other substances causing flatulence), or chewing gum while diving.

Symptoms of gastrointestinal barotrauma include abdominal fullness, colicky abdominal pain, belching, and flatulence. It is rarely severe because most divers will readily vent any excess bowel gas during ascent; however, it has been known to cause syncopal and shock-like states.[13] Only one case of actual gastric rupture from gastrointestinal barotrauma is known to have occurred, and this was a very unusual case of a scuba diver who swallowed a large volume of water secondary to near drowning.[10]

Pulmonary Barotrauma of Ascent. The most serious type of barotrauma of ascent is pulmonary barotrauma (PBT), which may result in several different injuries. These are collectively referred to as the pulmonary overpressurization syndrome (POPS) or "burst lung."

Breathing compressed air underwater puts the diver at risk of POPS because the compressed gas will expand during ascent according to Boyle's law. If the diver does not allow the expanding gas to escape from his lungs, it will rupture the alveolae and allow air to escape into extra-alveolar locations, resulting in pneumomediastinum, subcutaneous emphysema, pneumopericardium, pneumothorax, pneumoperitoneum, pulmonary interstitial emphysema, or systemic air embolism. Clinically, the result of such injury will depend on the location and amount of escaped gas. Symptoms may develop immediately upon surfacing or be delayed for several hours.

Mediastinal and subcutaneous emphysema are the most common forms of the POPS and usually present with gradually increasing hoarseness, neck fullness, and substernal chest pain several hours after diving. Dyspnea,

dysphagia, syncope, and other symptoms may be present as well. The history is usually diagnostic, although radiographs should be obtained to verify the location of gas and exclude a pneumothorax.

Although quite unusual, the occurrence of a pneumothorax while diving is an especially serious problem, for intrapleural gas cannot be vented to the environment and will progress to a tension pneumothorax during ascent. This possibility must always be considered when treating cases of suspected air embolism, and no patient should ever be recompressed with an untreated pneumothorax.

Except for pneumothorax, which may require needle aspiration or tube thoracostomy, treatment of uncomplicated pulmonary overpressurization typically requires only observation, rest and, if severe, supplemental oxygen. Recompression of these patients is potentially dangerous and necessary only in extremely severe cases, for example, if mediastinal or subcutaneous emphysema is so severe that it compromises the airway. Conversely, though, patients with air embolism require rapid recompression, and all patients with pulmonary barotrauma should be carefully examined for evidence of this potentially serious condition.

Air Embolism

The most feared complication of pulmonary overpressurization is systemic air embolism. Indeed, dysbaric air embolism (DAE) is one of the most dramatic and serious injuries associated with diving and is a major cause of death and disability among sport divers.

Dysbaric air embolism results from the entry of gas bubbles into pulmonary veins via ruptured alveolae. After passing through the heart these bubbles are disseminated more or less randomly through the systemic circulation and lodge in small arteries, occluding the more distal circulation. The resulting manifestations will depend on the location of the occlusion, and, depending on the site, even minute quantities of gas can have disastrous consequences (for example, if in the coronary arteries).

Dysbaric air embolism usually presents immediately after the individual surfaces from a dive, at which time the high intrapulmonic pressure resulting from lung overexpansion is relieved and allows bubble-laden blood to return to the heart; it is axiomatic that symptoms of dysbaric air embolism always develop within 10 minutes after surfacing from a dive. Although the classic history is that the diver ascends rapidly because of running out of air, panic, or a similar situation, this is not always the case, and localized overinflation may also result from focally increased elastic recoil of the lungs in some divers.[9]

The manifestations of dysbaric air embolism are usually dramatic. Coronary occlusion and cardiac arrest may occur,[5] although the brain is by far most often affected.[2 5 13 20 30 35 44 46] Neurologic manifestations are typical of an acute stroke, for example, monoplegia or multiplegia, focal paralysis, sensory disturbance, blindness or other visual aberration, deafness, vertigo, dizziness, confusion, convulsions, or aphasia. Sudden loss of consciousness upon surfacing should always be assumed to be due to gas embolism until proved otherwise. Although hemoptysis is often mentioned in standard references as a presenting sign of dysbaric air embolism, this

was noted in only 2 of 42 cases reviewed by me.[30] Likewise, hemiplegia and other purely unilateral brain syndromes that are often taught as being characteristic of dysbaric air embolism are infrequent, with asymmetric multiplegias being the most common presentation. Not surprisingly, the differentiation of dysbaric air embolism from severe neurologic decompression sickness is often impossible because of the multiplicity of deficits.[30, 39] Other reported clinical findings such as visualization of bubbles in the retinal arteries or Liebermeister's sign (a sharply circumscribed area of glossal pallor) are exceedingly rare.

Although the majority of patients with dysbaric air embolism will have signs of neurologic injury when evaluated, some patients who were initially very symptomatic may be much improved by the time they arrive at the emergency department or hyperbaric treatment facility.[30] The mechanism of spontaneous recovery is not clear, but it is consistent with clinical experience with iatrogenic air embolism. Nonetheless, these patients should still be referred for recompression because it is impossible to totally exclude neurologic impairment in the acute care setting, and waiting to do definitive diagnostic studies may allow subtle dysbaric injuries to become irreversible.

INDIRECT EFFECTS OF PRESSURE

Several diving-related problems may develop as a result of breathing gases at higher than normal atmospheric pressure. Chief among these are nitrogen narcosis and decompression sickness.

Nitrogen Narcosis

Nitrogen has an anesthetic effect at elevated partial pressures. Consequently, nitrogen narcosis, or "rapture of the deep" as it has been dubbed by Jacques Cousteau, is an inescapable concomitant of breathing compressed air when diving. The narcotic effects are similar to those of alcohol intoxication (that is, giddiness, impaired judgment, poor concentration, and so forth) and become evident in most divers between 80 and 100 fsw. Most divers are so impaired at 200 fsw that they can do no useful work, and many will lose consciousness at depths deeper than 300 fsw. Although these narcotic effects are entirely reversed as the P_{N_2} decreases with ascent, nitrogen narcosis is not an uncommon precipitating factor in diving accidents and may impair a diver's memory of the circumstances leading up to the accident. This should always be considered when taking the history of a diving accident.

Decompression Sickness

Decompression sickness (DCS) was first reported in the mid-1800s among caisson workers and tunnelers. Subsequently, the similarity of "caisson disease" and "diver's paralysis" became evident, but it has been only in the last few years that a real understanding of decompression sickness has evolved.

In essence, decompression sickness is a multisystem disorder resulting from the liberation of inert gas from solution and the resultant formation of

gas bubbles in blood and body tissues when ambient pressure is decreased. The critical factor in its pathogenesis is increased tissue absorption of inert gas, which in most diving situations is nitrogen.

As an air-breathing diver descends underwater, the ambient pressure increases, the partial pressure of respirable nitrogen rises, and a positive gradient for nitrogen flow from alveoli to blood to tissue develops. After a period of time at depth, the tissues absorb more nitrogen and this gradient diminishes, eventually becoming zero as a new equilibrium is reached. The time that it takes for the new equilibrium to be achieved will depend on the gradient of alveolar to tissue inert gas, the tissue blood flow, and the ratio of blood to tissue inert gas solubility. Consequently, the rate at which a diver reaches a new inert gas equilibrium will be an exponential function of the diffusion and perfusion characteristics of the different tissues.

Although tissue uptake of increased inert gas is the primary condition required for the occurrence of decompression sickness, the absorption of increased gas by itself does not cause decompression sickness; it is only when ambient pressure is decreased too rapidly to remove the excess body burden of inert gas that decompression sickness occurs. Indeed, this is the basis for the observation of early caisson workers that "one pays only on leaving."

Although decompression sickness is due to the development of gas bubbles in tissue and blood, the occurrence of bubbles alone cannot explain the varied manifestations of decompression sickness. In fact, it has now been well demonstrated by ultrasonic techniques that many divers develop intravascular bubbles without experiencing any apparent illness; these have been called "silent bubbles."[3, 21, 40, 42, 43] Therefore, the pathophysiology of decompression sickness should be viewed as a dual phenomenon involving both mechanical and biophysical effects of bubbles (Fig. 3).[1, 6, 13–15, 44]

The major mechanical effect of bubbles in decompression sickness is vascular occlusion; however, the bubbles in decompression sickness form primarily in the venous circulation and thus impair venous return. In addition, the bubbles in decompression sickness can form in the lymphatics, intracellularly, and essentially anywhere else, resulting in lymphedema, cellular distention and rupture, and similar mechanical effects. Also, venous gas emboli may cause paradoxical arterial embolization via intrapulmonic and intracardiac shunts.

Bubbles also exert a variety of biophysical effects due to blood-bubble surface interaction. Bubbles are viewed by the immune system as foreign matter and, consequently, incite an inflammatory reaction.[6] The key step in the process is activation of Hageman factor, which, in turn, activates the intrinsic clotting, kinin, and complement systems, resulting in platelet activation, cellular clumping, lipid embolization, increased vascular permeability, interstital edema, and microvascular sludging.[1, 6, 13, 14, 24] The net effect of these processes is decreased tissue perfusion and ischemia.

The clinical manifestations of decompression sickness are protean, with several different forms being recognized (Table 3). Overall, the joints and spinal cord are most often affected. Technically, the term "bends" refers only to the musculoskeletal form of decompression sickness, but it is commonly used in the generic sense to mean any type of decompression

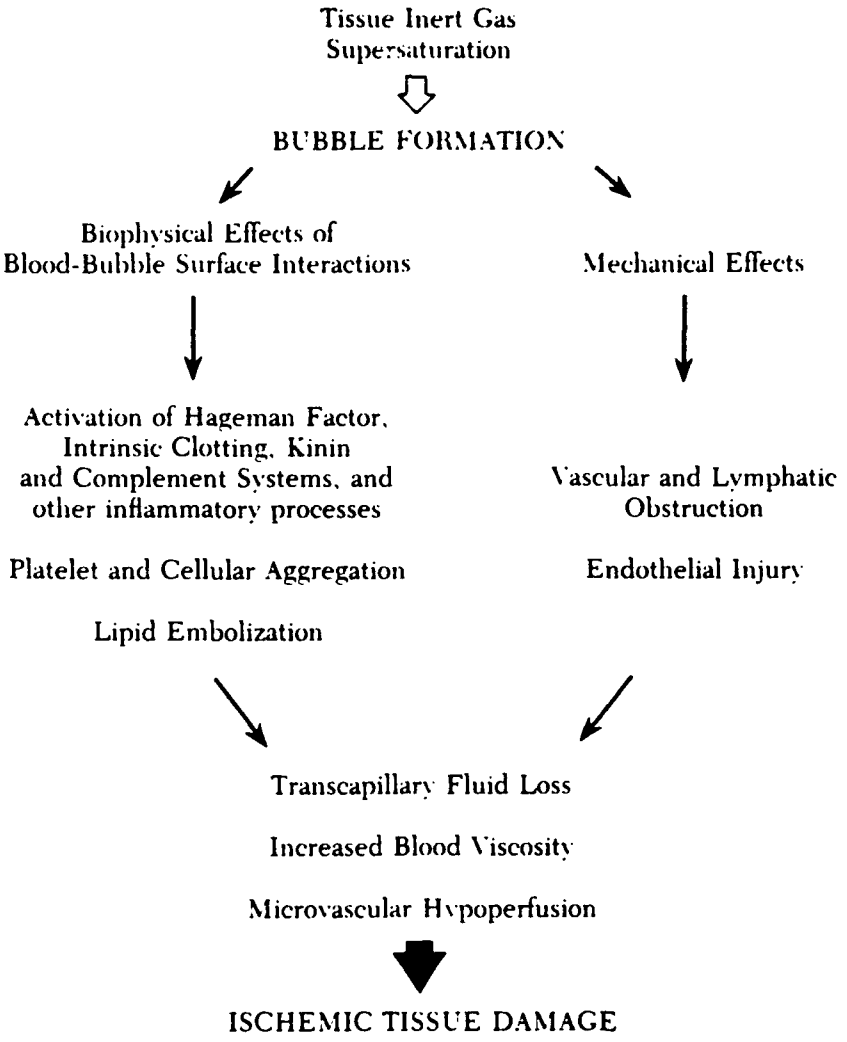


Figure 3. The pathogenesis of decompression sickness is schematically represented. (*From* Kizer KW: Management of dysbaric diving casualties. Emerg Med Clin North Am, *1*:665, 1983.)

Table 3. *Types of Decompression Sickness*

Cutaneous ("skin bends")
Lymphatic
Musculoskeletal (the "bends" or "pain only bends")
Neurologic
spinal cord
cerebral
cerebellar (the "staggers")
inner ear (the "staggers")
peripheral nerves
Pulmonary (the "chokes")
Cardiovascular ("decompression shock")
Visceral

sickness. The various forms of decompression sickness have also been arbitrarily categorized as either types I or II,[22] with type I referring to the mild forms of decompression sickness (skin, lymphatic, and musculoskeletal) and type II including the neurologic and other serious types. Although the latter categorization is widely used, it is clinically more meaningful to refer to the systems affected when discussing patients with decompression sickness.

More than half of all patients with decompression sickness will become symptomatic in the first hour after diving, with most of the rest becoming symptomatic within six hours.[13, 25, 26, 29, 41, 44] A few patients (1 to 2 per cent) may note their first symptoms 24 to 48 hours after diving, and these patients often present a diagnostic dilemma.

Cutaneous manifestations of decompression sickness include pruritus; formication; subcutaneous emphysema; and scarlatiniform, erysipeloid, or mottled rashes. Similarly, localized swelling or peau d'orange may result from lymphatic obstruction.

"Joint bends" or "pain only bends" are what most people think as decompression sickness, and, indeed, periarticular joint pain is the single most common symptom of decompression sickness. The pain is typically described as a deep dull ache, although it may be throbbing or sharp. There may be a vague area of numbness or dysesthesia around the affected joint, which should not be confused with neurologic decompression sickness. Movement of the affected extremity usually aggravates the pain, but inflation of a blood pressure cuff around the involved joint may relieve the pain for as long as the cuff is inflated. The shoulders and elbows are most often affected in scuba divers, although essentially any joint may be involved.[13, 25, 26, 29, 41, 44]

Neurologic decompression sickness may be manifested by a vast array of symptoms and signs because of the random nature in which the neurologic injury occurs, and essentially any symptom is compatible with neurologic decompression sickness. Classically, however, neurologic decompression sickness most often involves the lower thoracic, lumbar, and sacral portions of the spinal cord and produces paraplegia or paraparesis, lower extremity paresthesias, and bladder dysfunction. Historically, urinary retention was such a frequent manifestation of spinal cord decompression sickness that a urethral catheter used to be part of the diver's standard equipment.[4] Recently, Hallenbeck et al. have demonstrated that spinal cord decompression sickness results from infarction of the cord due to obstruction of venous drainage in the epidural vertebral venous plexus.[24]

Pulmonary decompression sickness, or the "chokes," results from diffuse venous air embolization and usually becomes symptomatic when 10 per cent or more of the pulmonary vascular bed is obstructed. Overt manifestations include tachypnea, chest pain, cough, dyspnea, and signs of circulatory compromise; radiographs may show pulmonary edema.[33] These patients often have a fulminant downhill course, as do victims of decompression shock, which results from the generalized liberation of gas bubbles in the circulation. Fortunately, both of these conditions are rare.

Although a variety of laboratory abnormalities may be demonstrated in decompression sickness most of them have little clinical relevance to the

acute care of these patients. However, two tests that are useful are the urine specific gravity and hematocrit, since intravascular volume depletion and hemoconcentration are common in serious decompression sickness. Other diagnostic tests are typically not necessary in the initial management of uncomplicated decompression sickness. However, the patient should always be carefully evaluated for life-threatening nondysbaric injuries as well. This is one of the most important things that the emergency physician can do before sending a patient to the hyperbaric chamber.

TREATMENT OF SERIOUS DYSBARIC INJURIES

Dysbaric air embolism and decompression sickness are true medical emergencies that should be treated in a hyperbaric chamber as soon as possible. However, since relatively few recompression chambers are available and they are often not located near popular diving areas, most victims will be first treated via the emergency medical services system. Thus, emergency personnel should be familiar with the basic management of these casualties, especially insofar as being able to determine when recompression is needed. This has been discussed previously,[32] and, in general, it is wise to consider all symptoms that begin after diving with compressed air as being due to decompression sickness or dysbaric air embolism unless they can be readily explained otherwise.

The resuscitation of the victim of a dysbaric diving casualty should begin as soon as a problem is suspected, with the specific measures depending on the circumstances. Obviously, the victim should be rescued from the water and basic life support measures (that is, establishing an airway, cardiopulmonary resuscitation, and so forth) commenced, as needed. Thermal balance should be maintained as for any aquatic accident victim.

If dysbaric air embolism is suspected, the victim should be placed in either the Trendelenburg or Durant position. The head down position is believed to decrease the chance of additional gas emboli traveling to the brain, and it may cause cerebral vasodilatation by increasing venous pressure, which should facilitate passage of bubbles through to the venous circulation. Whether this really happens or whether addition of the left lateral decubitus position (the Durant position) is of true benefit has not been well demonstrated, although theoretical reasoning and anecdotal data suggest that these positions are beneficial. In any case, unless transport to a hyperbaric chamber will take longer than 30 to 60 minutes, these maneuvers should cause no harm. However, the head down position does increase intracranial pressure, and if the patient is left in this position for very long, it may cause or worsen cerebral edema.

Supplemental high flow 100 per cent oxygen should always be given to suspected victims of dysbaric casualties as soon as possible. This facilitates off-gassing of the nitrogen bubbles and improves oxygenation of damaged tissues.

Depending on local circumstances, patients with suspected dysbaric air embolism or decompression sickness may be taken directly to the

recompression chamber or may need to go via the emergency department. Whichever the case, transportation should be as rapid as is safe. In cases of air transportaiton, the patient should be subjected to the least possible pressure reduction so as not to cause any further gas expansion. Either a low-flying helicopter or light airplane (that is, one capable of flight at 1000 feet or less) or aircraft that can be pressurized to 1 ATA (for example, a Lear jet or Hercules C-130) can be used.

Advanced life support drugs should be administered according to the victim's condition and standard protocols. In general, most decompression sickness victims will be at least mildly volume depleted, so parenteral and oral (if the patient is alert) fluids should be given at a brisk rate unless contraindicated for other reasons. High dose parenteral corticosteroids are probably beneficial[6, 11, 27] and should be given as soon as possible.

If the physician is uncertain about the need for recompression or the location of the nearest hyperbaric treatment facility, assistance is available 24 hours a day through the National Diving Alert Network at Duke University (919-684-8111).

Space does not allow for a discussion of recompression treatment here. Suffice it to say that hyperbaric oxygen is the keystone of treatment for both decompression sickness and dysbaric air embolism and that it is administered according to well-established protocols. Various types of hyperbaric chambers may be utilized for treatment, and the relative merits of one type or another are beyond the scope of this discussion.

The outcome of recompression treatment will depend on the severity of the disease, the delay in commencing hyperbaric treatment, and the victim's health prior to the accident. Overall, 80 to 90 per cent success rates have been reported,[11, 13, 25, 26, 29, 44] and even though recompression is more likely to be beneficial the sooner that it commences after the onset of symptoms, dramatic recoveries have been reported after treatment delays of seven to nine days.[11, 31]

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ORIGINAL CONTRIBUTION
embolism, air, dysbaric

Dysbaric Cerebral Air Embolism in Hawaii

Cerebral air embolism is a major cause of death and disability among sport scuba divers. To better define the epidemiologic and clinical manifestations of this infrequently encountered disorder, the records of all recompression treatments in Hawaii from 1976 through 1979 were reviewed. Forty-two cases of dysbaric air embolism (DAE) were identified on the basis of clinical criteria, accounting for 18% of the patients undergoing recompression treatment for diving-related disorders during this four-year period. In 22 patients (52%), DAE was part of a dysbarism syndrome that involved one or more forms of decompression sickness and/or in which DAE could not be differentiated from neurologic decompression sickness. The presenting signs and symptoms varied, with asymmetric multiplegia being the most common finding. Two patients died, giving a case fatality rate of 5% for those who survived until reaching the recompression chamber. Overall, 78% of the cases manifested either complete (61%) or substantial (17%) recovery with recompression and adjunctive medical measures. Traditional concepts of dysbaric cerebral air embolism are not adequate to explain the spectrum of clinical manifestations encountered in this condition. [Kizer KW: Dysbaric cerebral air embolism in Hawaii. Ann Emerg Med May 1987;16:535-541.]

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INTRODUCTION

Scuba diving has become increasingly popular in recent years. There are now about 3 million recreational scuba divers in the US, and more than 300,000 new sport divers are trained each year. In addition, compressed air diving has become an integral part of many occupations, accounting for many thousands of commercial, scientific, and military divers. Illustrative of the rise in popularity of diving is the fact that nearly 100,000 people are employed in the scuba diving industry in this country, with the industry generating revenues of $600 million in 1983.

Cerebral air embolism is the most dramatic and serious medical emergency associated with compressed air diving, and is a major cause of death and disability among recreational scuba divers. Most literature on dysbaric air embolism (DAE) has focused on fatal cases or has been based on US Navy experience with submarine escape training. This is not entirely analogous to the diving situation, in which there may be problems associated with inert gas loading and other diving-related factors.

This study was undertaken to define the characteristics of DAE in Hawaii, where dysbaric diving accidents are unusually frequent and where, until 1980, essentially all cases were treated by the US Navy Undersea Medicine Service at Pearl Harbor. The epidemiologic and clinical aspects of dysbaric cerebral air embolism in Hawaii have not been reported previously.

MATERIALS AND METHODS

The records of all recompression treatments in Hawaii from 1976 through 1979 were reviewed. Forty-two cases meeting clinical criteria for DAE were identified. These criteria included a compatible diving history (most often a rapid or uncontrolled ascent to the surface), sudden onset of symptoms within five minutes of surfacing from a compressed air dive, and characteristic symptoms and signs (typically, those of a sudden brain injury). These cases accounted for 18% of the patients who underwent recompression treatment for diving-related disorders during this four-year period.

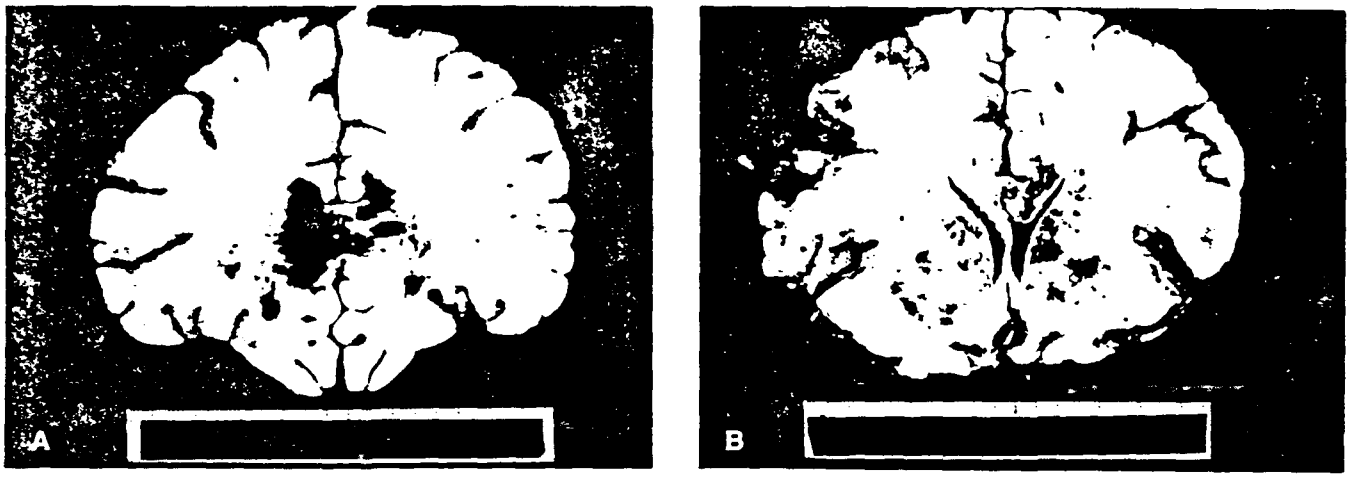


FIGURE. *Cross-sections of the brain of a 31-year-old male sport diving instructor who died four days after suffering an apparent cerebral air embolism (although he also omitted 38 minutes of required decompression). Bubble-induced infarcts were found in the distribution of both the carotid and vertebral arteries. The immediate cause of death was hemorrhage from a perforated gastroduodenal ulcer and Gram-negative sepsis. A) The effect of bubbles embolizing the lenticulate striate arteries. These are end arteries, and multiple discrete hemorrhagic infarcts can be seen corresponding to the area of tissue supplied by individual embolized vessels. B) A large infarct of the corpus callosum and scattered other infarcts. C) Infarcts adjacent to both lateral ventricles, but more severe on the right side. D) Diffuse infarction of the brainstem.*

Not included were several other presumed cases of DAE that occurred during this period but that resulted in death before recompression treatment could be commenced because of scant information. Thirty-nine of the patients (93%) were scuba divers, while three cases (7%) occurred in nondiving US Navy personnel undergoing submarine escape training.

The patients were divided into two groups. Group 1, with 20 patients (48%), had classical cerebral arterial air embolism. Group 2, with 22 patients (52%), had clinically apparent cerebral arterial air embolism, but in these DAE occurred as part of a dysbarism syndrome in which either the patient had omitted substantial amounts of required decompression (six cases, average omitted decompression [OD] of 35 minutes) or in which the patient manifested signs and symptoms of decompression sickness (DCS) and/or in which DAE could not be differentiated from neurologic DCS (16 cases, average OD of 85 minutes). The cases were analyzed according to host characteristics, dive factors, clinical presentation, and treatment methods and outcome.

Treatment outcome was divided into four levels of recovery according to the patient's assessment of improvement (eg, how much sensation or strength had returned) and the findings observed on physical examination. *Complete* recovery was defined as essentially complete resolution of all symptoms and signs; *substantial* recovery was defined as some residual problem but less than 30% of the pretreatment impairment. If 30% to 70% of the original manifestations persisted after treatment, the results were described as *moderate* recovery. If no demonstrable improvement occurred or if more than 70% of the original symptoms remained after treatment, then *minimal* recovery was said to have occurred. Although this categorization was arbitrary it corresponded well with the degree of improvement judged by clinical assessment, and has been used previously.[1]

RESULTS

Thirty-seven patients (88%) were men. The ages of the afflicted divers ranged from 18 to 57 years, with a mean of 30. Except for a relative preponderence of cases during the three months of September through November (40%), the cases were evenly distributed throughout the year.

Thirty-eight (97%) of the scuba divers had been diving around the four main Hawaiian Islands (Oahu, 58%; Maui, 18%; Hawaii, 18%; Kauai, 6%), while one case occurred in Tonga but was not treated until the patient arrived in Hawaii two days later.

The majority of divers (54%) were recreational scuba divers, although 46% were involved in some sort of commercial activity: spearfishing or underwater gill net fishing in 14 cases, gathering black coral in three, and cleaning a ship's hull in one. The three nondiving US Navy personnel suffered their accident as part of formal submarine escape training.

Information on the depth and bottom time of the dives was available for all the scuba divers, although the accuracy of these parameters was uncertain in some instances (Table 1). The greatest depths reported for Group 1 patients ranged from 15 to 180 feet of sea water (fsw), with a mean of 111 fsw. The depths reported for Group 2 patients ranged from 80 to more than 250 fsw (mean, 140 fsw).

Information on the diver's training and experience was available on 36 of the 39 scuba divers (92%). Twenty-six (72%) were certified sport divers; four also had graduated from a military or commercial diver training program. Eight divers had never received any formal training. Two cases (6%) occurred in neophyte sport divers who were participating in ocean training sessions. Of the 34 divers not part of a training program, six (17%) had been diving for less than one year since certification, and five (14%) had one to four years of experience. Nine patients (25%) reported diving experience ranging from five to nine years, while the remaining 14 divers (39%) each re-

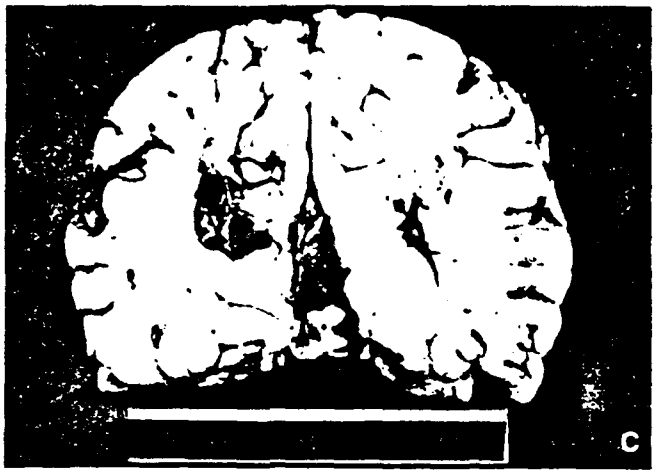




TABLE 1. *Depth of dives*

Depth (fsw)	Group 1 (%)	Group 2 (%)	Total (%)
15 - 40	4 (24)	--	4 (10)
60 - 90	4 (35)	2 (9)	8 (21)
110 - 180	7 (41)	18 (82)	25 (64)
> 200	—	2 (9)	2 (5)

ported more than ten years of experience. Most of these latter patients were diving fishermen.

All of the Group 1 divers had made only one dive, while 64% of the Group 2 patients had made multiple dives (as many as six) in the 24 hours preceding their accident. The amount of required decompression that had been omitted, according to the Standard US Navy Decompression Tables[2] for the reported dive profiles ranged from 0 to 300 minutes. Almost all of the Group 1 divers (17 of 20) omitted no required decompression, and the mean omitted decompression for these cases was less than one minute. Conversely, all of the Group 2 divers had omitted some required decompression, with the mean omitted decompression for Group 2 being 72 minutes.

In 14 of the 25 cases (56%) in which the information was available, the affected diver reported running out of air at depth and making an emergency free ascent. This was more common in Group 1 than in Group 2 (62% versus 50%). Two cases involved buddy breathing, in one the diver with the air supply remaining panicked and "shot" to the surface. No cases were reported as being due to equipment malfunction.

The symptomatic status of the patients on arrival at the recompression chamber was reported in 36 patients. All three of the submarine escape trainees were symptomatic on surfacing from their practice ascent and, consequently, were immediately placed into the on-site recompression chamber. Twenty of the 36 divers (56%) had either partial (36%) or complete (20%) resolution of their initial symptoms by the time they arrived at the recompression chamber (Table 2). Nonetheless, all patients were recompressed.

The primary presenting symptoms and signs varied (Table 3), although the recorded symptoms surely understate the actual array of presenting manifestations because the need for rapid initiation of recompression treatment, the spontaneous resolution of some symptoms, the stoic nature of many of the affected persons, and language barriers precluded obtaining a detailed history in a number of cases.

Paresis or paralysis of one or more extremities was the most common finding in this series, occurring in nearly two-thirds of the cases. Asymmetric diplegia of the lower extremities was present in nine patients (21%). Hemiplegia and monoplegia were present in eight (19%) and five (12%) patients, respectively. Varying degrees of quadriplegia or triplegia were present in the other five patients (12%). Four patients (10%) demonstrated a unilateral facial nerve palsy in combination with motor deficits in the extremities. Some degree of sensory disturbance was usually present in the same general anatomic distribution as the motor dysfunction, although some patients manifested seemingly pure motor or sensory findings. The sensory disturbance was usually described as "tingling" or "numbness."

Although 26% of patients complained of chest pain or dyspnea, only two had hemoptysis (one was a scuba diver; the other patient was undergoing submarine escape training), and only one had radiographic evidence of pulmonary over-pressurization (pneumomediastinum). There were no cases of pneumothorax. Most patients did not have chest radiographs prior to recompression because this capability was not available at the hyperbaric treatment facility, and the majority of patients presented directly to the recompression chamber instead of first being seen at a hospital where such diagnostic evaluation might have been performed.

The most common visual disturbance was "blurry vision," occurring in seven of the 11 patients having a visual aberration. Two patients initially were totally blind.

Treatment for the two groups was not notably different and consisted primarily of recompression (ie, hyper-

baric oxygen) and hydration. Steroids, mannitol, and/or diazepam were used in some cases. Several patients had medicated themselves with aspirin, codeine, and alcoholic beverages prior to seeking medical care. When the three submarine escape trainees (who were recompressed immediately after manifesting symptoms) and two divers who were first seen more than 24 hours after the onset of symptoms were excluded, the average delay in commencing recompression was three hours.

Two patients arrived at the recompression chamber in extremis and subsequently died, giving a case fatality rate of 5% for patients arriving at the chamber alive (Figure).

The majority of patients (60%) who were still symptomatic on arrival at the recompression chamber were recompressed to six atmospheres absolute (ATA), in accordance with US Navy Treatment Tables 5A, 6A, or 4, while the remainder were recompressed to 2.8 ATA, in accordance with Treatment Table 6.[2] The choice of treatment table was at the discretion of the treating physician. (Six different physicians supervised the care of these 42 cases.) The recompression schedule was extended in one-third of these latter cases.

Overall, 78% of the patients manifested either complete (61%) or substantial (17%) recovery (Table 4). When the recovery rates were compared according to whether steroids were used (Table 5) and whether the patients were recompressed to 6 ATA or 2.8 ATA (Table 6) no definite differences could be discerned. Unfortunately, when treatment outcome was evaluated according to all of the relevant factors (eg, symptomatic status on arrival at the recompression chamber, recompression schedule, use of steroids or other drugs, delay in beginning recompression, and severity of the patient's condition), the small number of patients in each group of conditions precluded any statistical analysis of the specific treatment regimens.

TABLE 2. *Symptoms present on arrival at the recompression chamber*

Symptoms*	Group 1 (%)	Group 2 (%)	Total (%)
Resolved	5 (31)	2 (10)	7 (20)
Decreased	5 (31)	8 (40)†	13 (36)
Unchanged	6 (38)	7 (35)	13 (36)
Worsened	0	3 (15)	3 (8)

*Does not include the three submarine escape trainees or three divers, for whom the information was not available
†One of these patients had complete resolution of all neurologic symptoms but continued to have hip pain typical of decompression sickness

TABLE 3. *Primary manifestations of dysbaric air embolism*

Clinical Findings*	Frequency (%)
Paresis/paralysis	27 (64)
Sensory disturbance	26 (62)
Dizziness or vertigo	18 (43)
Altered mental status†	17 (40)
Generalized weakness or fatigue	11 (26)
Visual disturbances	11 (26)
Nausea or vomiting	11 (26)
Chest pain or "tightness"	11 (26)
Joint pain	10 (24)
Headache	8 (19)
Loss of consciousness	7 (17)
Slurred speech	4 (10)
Shortness of breath	4 (10)
Back pain	4 (10)
Abdominal pain	3 (7)
Apnea	3 (7)
Nystagmus	2 (5)
Anisocoria	2 (5)
Hemoptysis	2 (5)
Neck pain	2 (5)

*Because most patients had multiple symptoms the percentages add up to more than 100% and indicate the proportion of all cases having that particular manifestation
†This was variously described as confusion, disorientation, decreased cognition, impaired memory, agitation, and drowsiness

DISCUSSION

Next to drowning, cerebral air embolism is the leading cause of death in sport scuba divers, accounting for about 30% of recreational scuba diving fatalities. It is generally regarded as the most serious of the dysbaric diving diseases. Nonetheless, it remains a rather rare and poorly understood disorder.

The epidemiologic and clinical characteristics of DAE in scuba divers have not been well defined. Among Navy submarine escape trainees, DAE has been reported to occur with a frequency of about one per 10,000 ascents, 5% to 15% of those accidents are fatal.[3-6] The incidence of DAE among sport scuba divers is not known because little information is available on the number of dives made per year by recreational divers. However, at active diving accident treatment centers in the US, DAE typically constitutes about 40% of the case load, ranging from 12% to 55%.[7-12] Despite the relatively large number of cases treated during this four-year period, the fact that air embolism contributed a relatively small

TABLE 4. *Treatment outcome for symptomatic cases*

Recovery	Group 1 (%) (n = 15)	Group 2 (%) (n = 20)	Total (%) (n = 35)
Complete	10 (67)	11 (55)	21 (61)
Substantial	4 (27)	2 (10)	6 (17)
Moderate	1 (6)	2 (10)	3 (8)
Minimal	0	5 (25)	5 (14)

TABLE 5. *Treatment outcome according to use of steroids*

Recovery	Steroids Used (%) (n = 17)	Steroids Not Used (%) (n = 17)
Complete	8 (46)	13 (76)
Substantial	3 (18)	2 (12)
Moderate	3 (18)	0
Minimal	3 (18)	2 (12)

TABLE 6. *Treatment outcome according to recompression regimen*

Recovery	6 ATA (%) (n = 14)	2.8 ATA (%) (n = 19)
Complete	9 (64)	12 (64)
Substantial	1 (8)	5 (26)
Moderate	2 (14)	1 (5)
Minimal	2 (14)	1 (5)

porportion (18%) of the total case load probably reflects the frequency with which decompression sickness occurs in Hawaii.[13-16]

DAE is the most feared complication of pulmonary barotrauma of ascent or, as it is also known, the pulmonary over-pressurization syndrome (POPS).[17] POPS, which is a graphic clinical manifestation of Boyle's law, occurs when compressed gas breathed underwater expands during ascent to the surface at a faster rate than it can be vented from the lung, resulting in overinflation, lung rupture and, consequently, spread of gas into extra-alveolar locations. This may produce pneumomediastinum, subcutaneous emphysema, pneumopericardium, pneumoperitoneum and/or arterial air embolism.

In contrast, decompression sickness is a multisystem disorder that occurs when ambient pressure is decreased, resulting in liberation of inert gas from solution and formation of gas bubbles in blood and body tissues. In scuba divers, DCS most often affects the musculoskeletal and central nervous systems. The critical factor in the pathogenesis of DCS is increased tissue absorption of inert gas, which is a time- and pressure- (ie, depth) related phenomenon dictated by Dalton's law of partial pressures and Henry's law of gas solubility. In most diving situations the inert gas is nitrogen because compressed air is breathed.

Classically, DAE occurs when a diver ascends while breathholding and traps the expanding intrapulmonic gas until it ruptures the lung. However, DAE also occurs in divers who have exhaled properly during ascent.[12,18,19] Such cases, which may be more numerous than previously believed, probably result from focal overinflation of the lung due to localized increased elastic recoil.[18] Whatever the exact precipitating cause, however, the end result is the same; that is, the sequential passage of air bubbles from the ruptured alveoli into the pulmonary veins, left atrium, left ventricle, and systemic circulation, where they cause arterial occlusion and resultant anoxic injury.

These data support previous observations that diving air embolism is usually associated with violations of basic principles of safe diving (eg, diving too deep or not monitoring one's air supply) and that DAE should be a preventable problem. The relatively increased frequency of cases found among experienced divers is interesting, but is probably atypical of scuba divers in general and is more likely attributable to the capricious diving practices of Hawaii's diving fishermen.[1,14-16] Conversely, the relative paucity of cases among sport diving trainees, which are especially numerous in Hawaii, attests to the safety consciousness of the local scuba diving instructors.

DAE typically presents immediately on, or soon after, surfacing from a dive, at which time the diver exhales and relieves the high intrapulmonic pressure buildup resulting from lung overexpansion, allowing bubble-laden pulmonary venous blood to return to the heart. Symptoms of DAE occur within ten minutes of surfacing from a dive. Much more common, however, is the onset of symptoms of DAE less than five minutes after surfacing, which was the case with all patients in this series.

Manifestations of DAE are myriad, depending on the exact location of the arterial occlusion, the volume of gas disseminated, and the rate at which gas gains access to the left ventricle and systemic circulation. The presenting symptoms are typically quite dramatic, with the brain being most often affected. Loss of consciousness, convulsions, blindness or other visual disturbances, aphasia, confusion, dizziness, vertigo, headache, focal weakness or hemiparesis, and various sensory disturbances have been the most commonly reported manifestations.[3,6,12,20-24] Sudden loss of consciousness in a scuba diver immediately on surfacing should always be considered due to cerebral air embolism until proved otherwise. Less frequent manifestations of DAE include

cardiac arrest due to coronary artery occlusion and abdominal pain due to visceral injury.[12,25]

The frequency of asymmetric multiplegia, the wide array of neurologic symptoms, and the high proportion of cases in which DAE could not be clearly differentiated from DCS in this series support the view that traditional clinical concepts about diving air embolism are too limited.[12,26] Indeed, the seemingly random and diffuse distribution of air bubbles in the cerebral circulation was well demonstrated by one of the two fatalities in this series (Figure). Whether these DAE-DCS syndromes resulted from pulmonary barotrauma and resultant systemic air embolization, arterialization of venous gas emboli, or a combination of both processes could not be discerned clinically.

Arterialization of venous gas bubbles may result from transpulmonary migration of air emboli through the pulmonary capillary bed, migration through arteriovenous shunts in the pulmonary or bronchial circulation, and/or migration from right to left atria through a patent or potentially patent foramen ovale.[12,27] All of these processes are more likely to occur in the presence of pulmonary hypertension, which is generally believed to occur in decompression sickness as a result of diffuse pulmonary venous embolization.

One particular finding that is often mentioned as a cardinal sign of DAE is hemoptysis; however, this was unusual in this series, occurring in only two (5%) of the patients.

Although the majority of DAE patients will have definite signs and symptoms of neurologic injury when evaluated, at least temporary spontaneous recoveries may occur. This has been rarely described in the past, probably because most of the literature is based on submarine escape tank trainees who are typically recompressed before this might occur. However, it is clear from this series that this phenomenon is not rare among sport scuba divers suffering cerebral arterial air embolism. This is consistent with what often has been observed after iatrogenic arterial air embolism.

Even though the initial overt manifestations of DAE may spontaneously resolve, many patients will later deteriorate, often to a worse condition than first experienced. This may occur even after apparently successful recompression treatment and is probably due to cerebral edema.[12,28,29] When these observations are considered along with the difficulty in uncovering subtle neurologic or neuropsychologic deficits in the acute care setting and the possible deleterious effects that may result from delaying recompression treatment, as well as the rarity of untoward effects associated with hyperbaric treatment, it seems clear that anyone suspected of suffering from a dysbaric air embolism should be referred immediately for recompression treatment.

Although computerized tomography may have a role in the post-recompression management of DAE,[30] the initial diagnosis of DAE and DCS are made solely on the basis of history and clinical findings. As demonstrated by many patients in this series, however, the distinction between these entities may not be possible. This can make management problematic because the traditional hyperbaric treatment advocated for these two conditions is different.[2,12,31,33]

Recent evidence, however, suggests that recompression to 2.8 ATA with hyperbaric oxygen may be as effective in treating DAE as the conventional recompression to 6 ATA on air.[33-36] The treatment outcomes achieved in this series also suggest that recompression to only 2.8 ATA may be adequate for treating DAE patients who are seen after delays of several hours, as well as those patients having combined dysbarism syndromes, but no definitive conclusions can be drawn from these data.

Interestingly, in Waite's classical work,[24] which is often cited as the basis for recommending recompression to 6 ATA for cerebral air embolism, maximal clearing of intravascular bubbles during recompression occurred before reaching 4 ATA. It may be that recompression to 6 ATA is optimal for submarine escape trainees, who will be recompressed within a very few minutes of suffering a DAE and, therefore, have primarily a simple mechanical bubble problem. However, in patients presenting with a several-hour history of anoxic brain injury, with its associated edema, microvascular sludging, etc, there may be more benefit in administering pure oxygen at 2.8 ATA than in reducing the diameter of any residual intravascular bubbles from 70%, which is achieved at 2.8 ATA, to 54%, which is achieved at 6 ATA.

Given the current proliferation of monoplace (ie, single lock) hyperbaric chambers in this country, and the continued paucity of multiplace chambers (ie, hyperbaric chambers having two or more locks that can be independently pressurized), this issue does have important implications for where and after what delay air embolism patients will be treated.[37,38]

Unfortunately, no conclusions could be drawn from this series about the effectiveness of various first aid and field care or of adjunctive medical measures for treating DAE. Placement of suspected DAE patients in the Trendelenburg or Durant positions continues to be a recommended immediate post-accident intervention, but its true efficacy remains unclear. Also, its ability to cause or worsen cerebral edema if maintained longer than 30 to 60 minutes has become recognized in recent years, as well as the increased difficulty in ventilating patients in this position.[12,17] Although statistical analysis was not possible, it seemed that the sooner patients were administered oxygen in the field, the better was their outcome. Whether this was artifactual, due to spontaneous resolution or some other cause, could not be determined, although this has been observed previously.[19]

The importance of hydrating dysbaric casualties seems to be well established,[3,12,14,32,39-43] although there is the same concern about overhydrating DAE victims as there is with other brain-injured patients. The usefulness of corticosteroids and various other pharmacologic agents in the treatment of DAE remains anecdotal, although steroids may be beneficial.[32,43-45] Overall, however, there are no adequate replacements for recompression and hyperbaric oxygen in the treatment of dysbaric air embolism.

CONCLUSION

Emergency physicians should be familiar with the variable clinical presentation of dysbaric air embolism and its management because of the increasing frequency of this problem due to the growing number of sport scuba divers and the burgeoning diving vacation industry, which accounts for many injured divers presenting to emergency departments far removed

from the diving site. All patients suspected of suffering from this disorder must be referred for recompression as rapidly as possible.

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Delayed Treatment of Dysbarism

A Retrospective Review of 50 Cases

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• Fifty cases of decompression sickness are reviewed in which recompression treatment was delayed for 12 hours or longer after the onset of dysbaric symptoms. Twenty-four patients (48%) had pain only. The other 26 patients (52%) had more serious decompression sickness; two patients may have had air embolisms. Ninety percent of the patients recovered either completely (66%) or substantially (24%) with recompression and associated treatment, although standard recompression protocols had to be lengthened in many of the cases.

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IT IS estimated that there are about 3 million recreational scuba divers in the United States now, and about 200,000 new sport divers are certified each year. In addition, there are thousands of commercial, scientific, and military divers. Concomitant with the increased participation in underwater activities in recent years there has been an increased occurrence of dysbaric diving diseases.

Recompression and hyperbaric oxygen therapy are the primary treatment for decompression sickness (DCS) and diving air embolism (DAE), and this treatment is more likely to be effective the sooner that it commences after the onset of dysbaric symptoms. Illustrative of this is the 100% success rate that Bayne[1] recently reported among a group of Navy divers suffering from decompression sickness who were aggressively treated after minimal delays. Typically, however, civilian diving is much less disciplined than military diving, and it also differs in other ways. In particular, many recreational divers do not appreciate the seriousness and consequences of DCS, or they are victims of diving accidents far removed from a hyperbaric treatment facility. Consequently, treatment of such cases is often delayed.

The efficacy of delayed recompression treatment of DCS and DAE has not been well established. Some case reports[2-3] and limited data from larger series[4-11] have indicated that it may be effective after long delays, but, in general, there are scant published data on this subject. The present study was undertaken to assess the effectiveness of delayed treatment of these maladies in Hawaii, where dysbaric diving accidents are unusually frequent.

MATERIALS AND METHODS

The records of all recompression treatments in Hawaii from 1976 through 1979 were reviewed. Delayed treatment was defined as treatment that commenced 12 or more hours after the onset of dysbaric symptoms. Fifty cases meeting this criterion were identified. These cases were divided into two groups according to type of dysbarism: group 1, mild or pain-only DCS (the "bends"); and group 2, serious DCS, a DAE, or both. These two groups were then subdivided according to the length of delay in beginning treatment: groups 1A and 2A, 12 to 24 hours; and groups 1B and 2B, more than 24 hours. The longest delays for groups 1B and 2B were 168 and 103 hours, respectively. The number of cases and mean delay for each group are listed in Table 1. The cases were then analyzed according to host characteristics, dive factors, clinical presentation, and treatment methods and outcome.

Ninety-two percent of the cases involved divers who had been diving around the main Hawaiian Islands (Oahu, 60%; Maui, 14%; Kauai, 14%; and Hawaii, 4%); the remaining four cases (8%) came from other islands in the western Pacific basin.

The outcome of treatment was divided into four levels of recovery according to the patient's assessment of his improvement (eg, how much of the pain had resolved) and the findings observed on physical examination. Complete recovery was defined as essentially complete resolution of all symptoms, while substantial recovery was defined as some residual but less than 30% of the pretreatment symptoms. If 30% to 70% of the original manifestations persisted after treatment, the results were described as moderate recovery. If no demonstrable improvement occurred or if more than 70% of the original symptoms remained after treatment, then minimal recovery was said to have occurred. Although this categorization was arbitrary it corresponded well with the degree of improvement judged by clinical assessment.

RESULTS

Ninety-four percent (47) of the patients were men. The ages of the afflicted divers ranged from 19 to 52 years, with a mean of 30 years. The mean age for each of the four groups is listed in Table 1.

The patients had been sport diving in 52% of the cases. Forty-six percent of the cases involved various sorts of commercial diving: spearfishing or gill-net fishing in 18 cases, harvesting black coral in three cases, gathering valuable shells in one case, and collecting tropical fish in one case. The one case involving a military diver has been discussed elsewhere.[4]

Information on length of diving experience was available in 49 cases. In general, the divers were experienced, with the reported diving experience being less than one year in 14%, between one and four years in

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Table 1 —Mean Age, Omitted Decompression, and Delay

Group	Cases, No.	Age, yr	Omitted Decompression, min	Delay, hr
1A	13	28	103	15 8
1B	11	33	85	55 2
2A	13	29	80	16 2
2B	13	31	38	44 6

Table 2 —Time Interval Between Surfacing and Patient's Awareness of Dysbaric Symptoms

	%				
	≤10 min	10-60 min	1-6 hr	6-12 hr	>12 hr
Group 1, n=24	29	21	33		17
Group 2, n=24	46	21	12	17	4
Total, n=48	38	21	23	8	10

37%, between five and nine years in 12%, and more than ten years in 37%. Twenty percent of the divers reported diving for more than 15 years, with one diver reporting 28 years of experience.

Information on the depth and bottom time of the dives was available on all cases, although the accuracy of this information was uncertain in some instances. The deepest depth reported ranged from 40 to 260 feet of seawater (fsw); the average deepest depth for the whole group was 106 fsw.

In 78% of the cases the diver had made multiple dives, sometimes exhausting more than six scuba tanks of air in only a few hours. The amount of required decompression omitted for the reported dive profiles, according to the Standard US Navy Decompression Tables,[7] ranged from 0 to 416 minutes, with a mean of 70 minutes. The average amount of omitted decompression for the four groups is listed in Table 1.

Forty-eight (96%) of the patients had diving histories and symptoms characteristic of DCS; the other two patients probably had cerebral air embolisms, although neurological decompression sickness as either the primary or a concomitant process could not be excluded in either case, since both patients had omitted decompression (three and 14 minutes). Both of these cases were included in group 2B.

Information on the time of symptom onset after surfacing from diving was available in 48 cases. More than half (59%) of these patients noted their first symptoms within one hour of surfacing. Four patients (8%) said they first became aware that something was wrong between 24 and 48 hours after the last dive, although it appeared that these divers experienced some symptoms earlier but ignored them. In a few cases dives were made after the onset of symptoms; in all such cases the diver was considerably more symptomatic after doing so. In these cases the delay in commencing treatment was calculated from the time the diver surfaced from the last dive, since the interval between the onset of first symptoms and surfacing from the last dive was usually not known. The distribution of symptom onset according to time after surfacing is shown in Table 2.

Thirty-seven (74%) of the patients reported having musculoskeletal pain typical of the "bends," but only 24 (48%) reported this as their only symptom. Multiple joints were involved in several patients. The anatomic distribution of the 53 affected joints is listed in Table 3.

The primary presenting symptoms for the 26 cases of neurological dysbarism are listed in Table 4. The spinal cord was most often affected, and the most common symptoms were "numbness" or "tingling" in one or more extremities. The frequency of symptoms referable to the brain is noteworthy.

Twelve patients (24%) reported having had DCS one or more times before; these were all commercial divers. In addition, several of the patients had other medical problems such as hypertension, asthma, and obesity. Both of the patients with presumed air embolism had a history of asthma.

Table 3 —Joints Affected in Cases of Pain-Only Decompression Sickness*

	%		
	Left	Right	Total
Shoulder	37	23	60
Elbow	15	4	19
Wrist	4		4
Hip	7	3	10
Knee	2	5	7

*Since many patients had multiple symptoms the percentages indicate the proportion of group 2 patients having that particular manifestation of decompression sickness

Table 4 —Primary Symptoms in the Serious Decompression Sickness Cases*

Symptoms	%
Headache	41
Nausea	37
Dizziness	33
Malaise or lethargy	28
Impaired cognition	28
Vertigo	17
Impaired vision or hearing	12
Loss of consciousness	4
Monoplegia	20
Diplegia	17
Generalized weakness	17
Facial paresis	4
Ataxia	25
Sensory disturbance of one or more extremities	61
Facial paresthesias	8
Truncal paresthesias	4
Periarticular joint pain	52
Chest pain	29
Neck pain	17
Abdominal pain	12
Back pain	12
Bladder dysfunction, bowel dysfunction, or both	20

*Since many patients had multiple symptoms the percentages indicate the proportion of group 2 patients having that particular manifestation of decompression sickness

The treatment for groups 1A and 1B was not substantially different. Recompression and oral hydration were the principal modalities employed, although two patients were given a single parenteral dose of steroids, and six patients were given aspirin, 650 or 975 mg, at the beginning of recompression. Some of the patients had taken unspecified quantities of aspirin, codeine, or other analgesics, as well as substantial quantities of alcoholic beverages, before arriving at the recompression chamber. There was no notable difference among these patients in the

Table 5 —Percent Overall Recovery Rates

Group	Complete	Substantial	Moderate	Minimal
1A, n=13	62	15	8	15
1B, n=11	70	30		
2A, n=13	84	8		8
2B, n=13	54	38	8	
Total, n=50	66	24	4	6

amount of recovery experienced by those who had only recompression and hydration and those who had recompression, hydration, and steroids or aspirin.

Three group 1 patients, all of whom had only minimal symptoms when recompression commenced, achieved complete relief of their pain with US Navy Treatment Table 5.[7] One other patient demonstrated no improvement with Treatment Table 5 and, for unclear reasons, was not given the benefit of additional recompression. All of the remaining patients required longer recompression according to US Navy Treatment Table 6,[7] which was often extended by several oxygen breathing periods, to achieve maximal pain relief. Overall, 90% of group 1 patients recovered completely or substantially.

Detailed treatment records were available on 25 of the 26 neurological cases. As with group 1, treatment was not substantially different for groups 2A and 2B. Slightly less than half (48%) of these patients were treated with parenteral steroids in addition to recompression and hydration. Although no definite pattern could be discerned as to the rationale for prescribing steroids (seeming to relate to individual physician habits more than anything else), there was a tendency to give steroids to the more severely affected divers. Dexamethasone was used in all cases in which corticosteroids were prescribed, usually as a 10- or 12-mg intravenous bolus before recompression and followed by 4 mg every six hours for up to three days. In a few cases high-dose hydrocortisone sodium hemisuccinate (1,000 mg) was given along with dexamethasone as an initial bolus; when this was done the initial dose of dexamethasone was usually decreased to 4 or 6 mg. In no instance was there any adverse effect that could be associated with the use of steroids.

Except for there being relatively more severely affected patients in the steroid-treated group, there were no gross differences between the steroid-treated and non-steroid-treated groups. It seems notable, then, that more of the steroid-treated patients recovered fully. Ten (84%) of these patients recovered completely, while one recovered substantially and the other moderately. Conversely, only seven (54%) of the non-steroid-treated group recovered completely, and five (38%) recovered substantially. The one other patient in this group recovered minimally.

With one exception, the group 2 patients were treated according to US Navy Treatment Table 6,[7] although more than half of these patients (52%) required extension of the recompression schedule to achieve a stable level of recovery. (The two possible cases of air embolism were also treated with the Table 6 protocol, presumably because the treating physician saw no benefit to pressurization to 6 atmospheres absolute on air, as required by Navy Treatment Table 6A[7] after such long delays, as well as there being some question about whether these were cases of neurological DCS or air embolism.) In general, the more severe cases required lengthening of the treatment table, while the less serious cases responded to an unmodified Table 6. This seems to be reflected in the recovery rates as well, since 63% of the patients treated with an extended Table 6 recovered completely, while 80% of the patients treated with a straight Table 6 fully recovered.

Interestingly, when the recovery rates of the patients treated with an extended Treatment Table 6 are compared according to whether steroids were used or not, there appeared to be a benefit associated with the use of corticosteroids. Six (75%) of eight steroid-treated patients recovered completely, while only two (40%) of five non-steroid-treated patients recovered fully.

The treatment outcome for all 50 cases is shown in Table 5. In 46 cases (92%), the amount of recovery was that which was achieved with the initial recompression and adjunctive measures. The remaining four cases (8%) were treated with additional hyperbaric oxygen therapy (HBOT) on a daily basis for as long as the patient demonstrated improvement that could be attributed to the HBOT. The number of such treatments ranged from two to eight. Both of the patients with presumed air embolism underwent HBOT, but neither fully recovered. (One recovered substantially, and the other improved moderately.) Overall, two thirds of the patients recovered completely, and 90% demonstrated at least substantial improvement. Unfortunately, long-term follow-up on the patients with residual impairment was not available. Not surprisingly, the least beneficial results were achieved in the neurological cases that were most delayed.

COMMENT

General epidemiologic and clinical characteristics of DCS and DAE in Hawaii have been presented elsewhere.[10-13] The 50 cases reported here constituted 19% of the cases treated during this four-year period, and they did not substantially differ from the larger group in age, sex, dive factors, presenting symptoms, treatment methods, or overall recovery rate.[13] The capricious diving practices of Hawaii's diving fishermen and the high proportion of serious DCS treated in Hawaii have been noted before.[10-13]

The reasons for delayed treatment in this series were not always clear. The few cases that occurred outside of the Hawaiian Islands usually involved complicated and lengthy transportation arrangements. More often, though, the delay was due to the diver failing to recognize the importance of postdive symptoms or appreciate the need for recompression treatment. A number of the diving fishermen first attempted self-treatment by in-water recompression with compressed air and, after that failed, employed various combinations of alcoholic beverages and analgesics before seeking recompression. The futility of in-water recompression with compressed air is well

known.[1,2] The need for further education of the diving community about these things is clearly evident.

In general, treatment of these patients was conducted according to the guidelines promulgated in standard references.[12,15] However, during the four-year period covered by this study at least eight different physicians were involved in treating these 60 patients, and the primary treating physician usually did not consult with any other physician before commencing treatment. Thus, treatment was in no way controlled and reflected the individual biases of the treating physician.

When evaluating the efficacy of treatment for these diseases it should be remembered that the diagnoses of DCS and DAE are purely clinical diagnoses based on the occurrence of characteristic symptoms and signs after appropriate diving or other pressure exposures, there being no specific laboratory or other diagnostic tests, and that recompression treatment is a dynamic process in which the variables of pressure and oxygen breathing time, which determine the "oxygen dose," are titrated according to the patient's response.

Although several different recompression protocols have been advocated for the treatment of DCS, the ones most widely used in this country are the oxygen treatment tables promulgated by the US Navy.[12] All patients in this series were recompressed according to these protocols, except for many of the patients being given additional oxygen breathing periods. When treating DCS the physician may extend the recompression schedule at a given pressure if the patient demonstrates continued improvement at that pressure, which would be either 2.8 or 1.9 atmospheres absolute when Navy Tables 5 or 6 are used. In a multiplace recompression chamber, several additional oxygen breathing periods can be given because of the ability to give periodic air breaks, which decrease the risk of oxygen toxicity. As many as four or five extensions were given in some of the cases in this series.

Another factor that must be considered when evaluating the efficacy of recompression treatment is that the US Navy treatment tables were derived for healthy, well-conditioned Navy divers who had been doing controlled diving. No recompression treatment tables have ever been developed specifically for civilian sport divers, ie, ones that take into account poor physical conditioning, the presence of concomitant medical problems, uncertain depth and bottom time factors, and other factors that complicate the treatment of civilian diving casualties. Nonetheless, the Navy treatment tables have worked reasonably well for treating civilian divers over the years, having overall success rates of 80% to 90% in patients who have been treated with delays of less than 12 hours,[1,2,9,10,13,14] which is comparable with the results achieved in this series of cases having more delayed treatment. When these results are viewed together with the rarity of untoward effects associated with recompression treatment, it is clear that all persons suspected of having untreated DCS must be referred for recompression even if such has been delayed for several days or, possibly, longer.

It is apparent from this series, though, that a larger oxygen dose may be needed when treating delayed cases. Except for the three patients who had minimal symptoms remaining when recompression commenced, US Navy Treatment Table 5 was not sufficient to achieve a maximal therapeutic effect in the other 21 cases of pain-only DCS. Likewise, Treatment Table 6 needed extension by two or more oxygen periods in more than half of the serious DCS cases. Overall, the treatment table had to be lengthened in 69% of the cases (34 of the 49 cases in which information was available). Whether recompression at higher pressures with Nitrox, a 50% combination nitrogen-oxygen mixture, or with saturation treatment, as has been advocated for difficult cases by some investigators,[20] would be helpful in cases of delayed treatment has not been investigated.

Although recompression is the essential treatment of DCS and DAE, several adjunctive pharmacologic agents have been recommended for treating neurological dysbarism in recent years. In particular, corticosteroids have been widely recommended, although their efficacy has never been determined by controlled studies. Interestingly, though, steroids and osmotic agents have at times produced apparent therapeutic effects independent of recompression.[4,5] Thus, the greater recovery rate observed in the steroid-treated cases in this series was notable and supports the notion that their use may have a beneficial effect in neurological DCS. It is important to note, however, that the retrospective and uncontrolled nature of this study, as well as the lack of long-term follow-up, precludes deriving any definite conclusion about the efficacy of steroids in these diseases.

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DIVING EMERGENCIES

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A. Whenever humans breathe compressed air in the underwater environment they are subject to various physical and biologic hazards, the most unique of which are the pressure-related maladies. Most diving-related emergencies can be properly diagnosed by history and physical examination alone. The history is most important and, at a minimum, should determine the type of diving done (e.g., scuba diving versus snorkeling); the equipment used (e.g., depth gauge, diving watch); the number, depth, and submergence time of all dives in the 48 to 72 hours preceding symptom onset; the surface interval(s) between repetitive dives; whether in-water recompression was taken, and if so, how much; the specific site of diving and its environmental conditions (e.g., presence of a strong current and water temperature); primary diving activity engaged in (e.g., photography, spearfishing); whether the dive was complicated by running out of air, marine animal injury, trauma, or other notable event; what the diver did after diving; and, importantly, when the first symptoms were noticed and how they have changed, either spontaneously or as a result of something the diver did. Many of the most serious diving emergencies are caused by formation of bubbles of nitrogen in the diver's tissues.

B. Nitrogen gas uptake during diving depends on depth and time. When safe diving depth-time limits are exceeded, specific time must be taken for "decompression" to allow excess dissolved nitrogen in the diver's tissues to be off-gassed. This must be done to avoid bubble formation in tissues, the penultimate condition for decompression sickness. The U.S. Navy Standard Decompression Tables are the most widely accepted protocols for determining whether the period of required decompression has been adequate.

C. Decompression sickness (DCS) is a multisystem disorder resulting from the liberation of inert gas (e.g., nitrogen) from solution and the resultant formation of gas bubbles in blood and body tissues when ambient pressure is decreased. The pathogenesis of DCS involves both bubble formation and the activation of Hageman factor and other biophysical phenomena at the blood-bubble interface. Most often affected by DCS are the musculoskeletal and neurologic systems. When more than about 10 percent of the pulmonary venous system is occluded by intravascular bubbles, a serious condition known as "the chokes" results. About half of persons suffering DCS develop symptoms within 10 minutes of surfacing from diving; more than 95 percent have symptoms within 6 hours after diving. Rarely, the onset of symptoms may be delayed for as long as 24 hours after diving.

D. In some cases, neurologic decompression sickness is not readily distinguishable from air embolism.

E. Dysbaric air embolism (DAE) is the most feared complication of pulmonary barotrauma and is a major cause of death and disability among sport divers. Symptoms of DAE always occur within 10 minutes after surfacing from a dive, and usually within the first 2 minutes. The neurologic manifestations are typical of an acute stroke, asymmetric multiplegia being the most common presentation overall.

F. Even though a diver may report no omitted decompression, this is sometimes unreliable (e.g., when a depth gauge and a diving watch are not used). Likewise, a very small number of people may suffer decompression sickness despite having adhered to the diving tables.

G. Gas pressure in the various air-filled spaces of the body is normally in equilibrium with the environment; however, if something (e.g., mucosal inflammation or a nasal polyp) should obstruct the portals of gas exchange for these spaces, a pressure disequilibrium will develop. *Barotrauma* is the term used to denote the tissue damage resulting from such an imbalance.

H. The ears and paranasal sinuses are most often affected by barotrauma.

I. Pulmonary barotrauma may occur during ascent from depth with a closed glottis (as when holding one's breath) or as a result of a focal problem (e.g., a mucus plug or granuloma). The most common manifestations of pulmonary barotrauma are mediastinal emphysema and subcutaneous emphysema; however, pneumopericardium, pneumoperitoneum, and/or (rarely) pneumothorax may occur singly or in combination. Recompression is contraindicated unless there is concomitant decompression sickness or air embolism. Management of the extra-alveolar air depends on its location and amount.

J. Although gastrointestinal air accumulation is generally easily relieved and self-limited, gastric rupture has occurred in rare instances.

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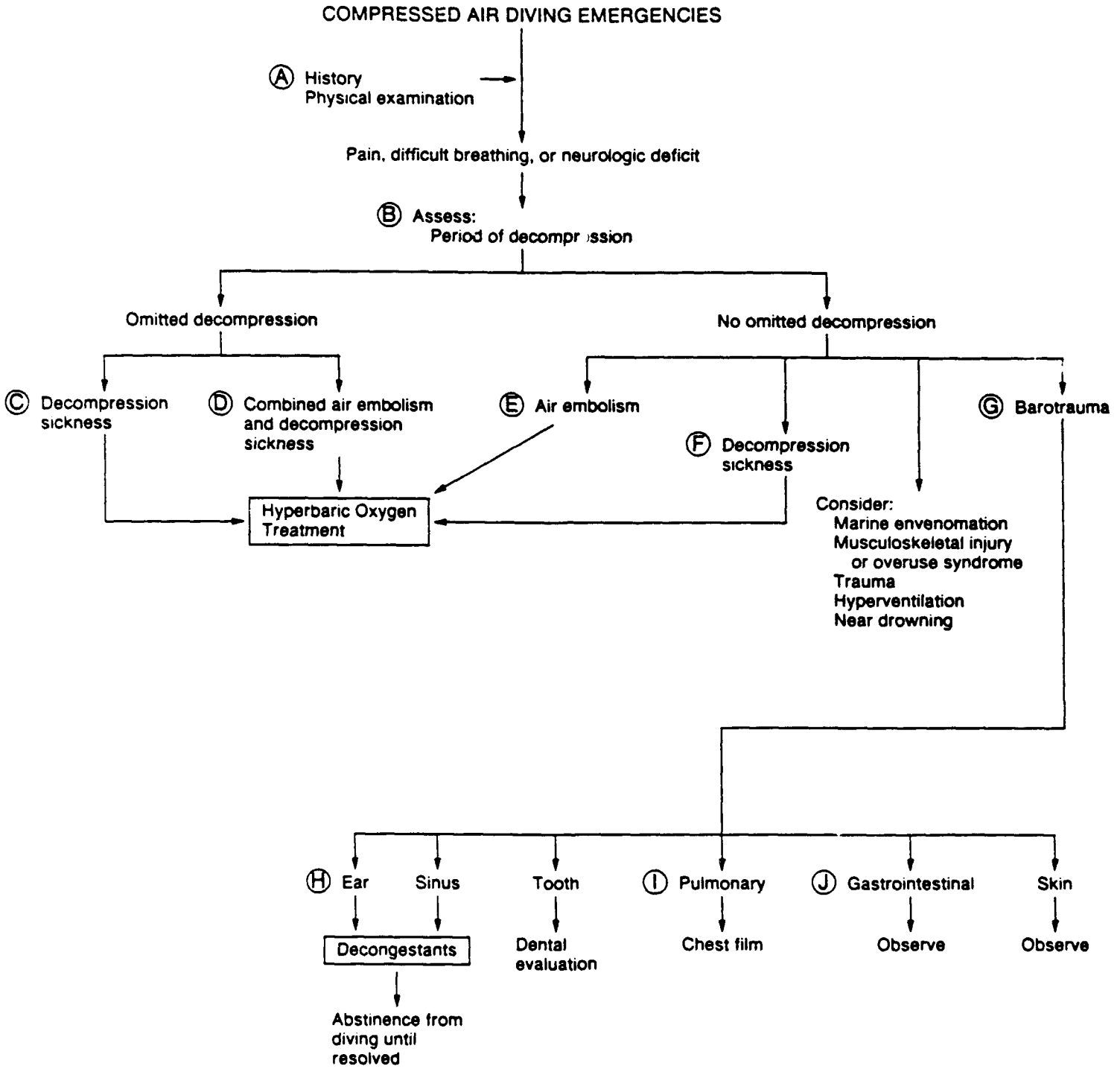
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COMPRESSED AIR DIVING EMERGENCIES
Ⓐ History
Physical examination
Pain, difficult breathing, or neurologic deficit
Ⓑ Assess:
Period of decompression
Omitted decompression
No omitted decompression
Ⓒ Decompression sickness
Ⓓ Combined air embolism and decompression sickness
Ⓔ Air embolism
Ⓕ Decompression sickness
Ⓖ Barotrauma
Hyperbaric Oxygen Treatment
Consider:
Marine envenomation
Musculoskeletal injury or overuse syndrome
Trauma
Hyperventilation
Near drowning
Ⓗ Ear
Sinus
Tooth
Ⓘ Pulmonary
Ⓙ Gastrointestinal
Skin
Decongestants
Dental evaluation
Chest film
Observe
Observe
Abstinence from diving until resolved

MAN AT ALTITUDE
ADAPTATION AND MALADAPTATION

WILDERNESS MEDICAL SOCIETY, 1991

Dr. Peter H. Hackett

Director, Denali Medical Research
Affiliate Associate Professor,
University of Washington School of Medicine and
University of Alaska Anchorage

EDUCATIONAL OBJECTIVES

1. To understand the normal physiological adaptations that take place on ascent to altitude.

2. To appreciate the limitations imposed upon exercise at high altitude and how to maximize performance.

3. To understand the pathophysiology of the problems of high altitude, including acute mountain sickness, the edemas of altitude, and retinal hemorrhage.

4. To gain a working knowledge of the prevention, diagnosis and treatment of these conditions.

OUTLINE

I. THE PHYSIOLOGY OF ACCLIMATIZATION

A. Struggle responses (first few days)

1. Hyperventilation

a. Attempt to maintain alveolar PO_2

b. Produces hypocapnic alkalosis, secondary bicarbonate diuresis

c. Determined partly by carotid body function (hypoxic ventilatory response) and CO_2 sensitivity, attenuated by alkalosis, alcohol, sleeping meds. Augmented by respiratory stimulants.

2. Increased cardiac output (mostly increased heart rate)

3. Fluid shifts

   a. Increased intracellular fluid

   b. Diuresis secondary to veno-constriction with central shift of blood, and bicarbonate excretion

   c. Loss of 15% of plasma volume in first few days, reduces stroke volume.

4. Increased hemoglobin concentration

   a. Caused initially by decrease in plasma volume

   b. Increases oxygen carrying capacity per unit of blood.

   c. Increased erythropoeisis within hours, but weeks to increase red cell mass

5. Changes in cerebral blood flow depend upon interaction of O_2 and CO_2-- generally increases 25-40% at 14000 feet, day one, then returns to normal over five days.

6. Cellular metabolic changes

   a. Some cells (endothelial) produce hypoxic associated proteins (HAP)

   b. Increased oxidative enzymes (muscle)

B. Adaptive changes (weeks to months)

1. Hyperventilation continues but renal bicarbonate excretion brings pH toward normal

2. Cardiac output decreases

3. Increased RBC mass

4. Increased density of capillaries in tissues and mitochondria in cells.

II. EFFECTS OF ALTITUDE ON EXERCISE PERFORMANCE

A. VO_2 max. decreases by 3% per 1000 feet, starting at 5,000 feet, improves with acclimatization very slowly and not by much.

B. Submaximal exercise (endurance time)--starts to increase at day 5, up 50% by day 15.

C. Factors limiting exercise are not well understood. Possiblities:

1. Decreased motor meuron drive (CNS) to muscles.

2. Decreased diffusion of O_2 to muscle cells.

D. Efforts to increase performance at altitude include blood doping, nutrition, hyperbaric and hypobaric training, and various drugs. Acclimatization seems best.

E. Altitude exposure may benefit sea level performance.

III. ACUTE MOUNTAIN SICKNESS (AMS)

A. General

1. Incidence of 15-30% of Colorado resort skiers, 50% McKinley climbers, 70% Rainier climbers, 70-100% if flown to 14,000 ft.

2. Related to **RATE OF ASCENT**, altitude reached, **SLEEPING ALTITUDE**, and contributory factors. No relationship to physical fitness. Younger may be more susceptible.

3. Individual susceptibility and reproducibility. Low vital capacity, low HVR, exaggerated pulmonary hypertension in response to hypoxia all contribute to illness. Past history a risk factor.

B. Diagnosis--**THINK AMS!**

1. Setting: rapid ascent to a higher altitude in unacclimatized persons.

2. Symptoms: headache, anorexia, dizziness, nausea, insomnia, lassitude, dyspnea. Note: mostly neurological symptoms. Periodic breathing common, not a sign of AMS. Early AMS exactly like a hangover.

3. In early stages, a lack of physical findings. Advanced: findings of pulmonary and cerebral edema. Ataxia and nailbed cyanosis most useful indicators of serious illness.

4. Differential diagnosis: dehydration, exhaustion, CO poisoning infections of lung or brain, hypothermia, drugs, psychiatric problems.

C. Pathophysiology

1. Ambient hypoxia leads to arterial hypoxemia and cerebral hypoxia.

   a. Hypoxia at a given altitude depends upon degree of hyperventilation. The lower the P_ACO_2, the greater the P_AO_2.

   b. Brain is sensitive to arterial PO_2, not ambient PO_2.

2. Inadequate ventilation (low HVR) causes greater hypoxemia.

3. Impaired gas exchange leads to even greater hypoxemia-increase alveolar-arterial oxygen difference.

4. Fluid retention and redistribution.

   a. Diuresis with acclimatization, antidiuresis with AMS.

   b. Role of ADH, renin-angiotensin-aldosterone systems.

   c. Brain and lungs overhydrated.

5. Brain Swelling.

   a. Cytotoxic edema--shift of fluid into cells.

   b. Vasogenic edema--permeability vs increased capillary filtration.

   c. Role of cerebral blood flow--too much or too little?

6. Theory of generalized "capillary leak syndrome"--lung, brain, kidney, retina.

D. Treatment

1. Descent--1,000ft may be adequate--as far as necessary for results.

2. Acclimatization at same altitude ok for self-limited illness--sick person **never** left behind alone.

3. Oxygen if available, especially good for headaches and encephalopathy.

4. Analgesics--acetaminophen, aspirin, codeine.

5. Anti-emetics--prochlorperazine (Compazine) 10mg IM, mg po (increases HVR). Benadryl for extrapyramidal reactions.

6. Acetazolamide 125-250mg every 8-12 hours (a sulfa drug).

7. Dexamethasone 4mg po, IM, IV every 6 hours if ataxia or change in consciousness present.

8. May re-ascend with staged acclimatization, acetazolamide.

E. Prevention

1. Slow ascent; climb high, sleep low.

2. High carbohydrate diet-->70%, improves respiratory quotient.

3. Avoidance of respiratory depressants (esp. sleeping pills) and use of alcohol in small amounts only.

4. Chemoprophylaxis:

a. indications are forced rapid ascent or history or recurrent illness.

b. Acetazolamide 5mg/kg/day divided into 2 or 3 doses, for one day prior and one day after ascent.

c. Dexametherone 4mg every 6 to 12 hours--for those intolerant of acetozolamide, or for insertion to extreme altitude.

IV. HIGH ALTITUDE PULMONARY EDEMA (HAPE)

A. General

1. Strikes 1-2% above 12,000 ft.

2. Most common on 2nd night.

3. Related to rate of ascent, exertion, use of sleeping medications, cold.

4. Many (most?) people have transient, milder forms of illness.

B. Diagnosis

Early: dry cough, increased heart rate, decreased exercise performance, shortness of breath with exercise and increased exercise recovery time.

Late: dyspnea at rest, tachycardia, tachypnea, cyanosis, productive cough, rales.

Atypical presentations: sudden death, cerebral manifestations only (esp. ataxia), acclimatized person, mixed with respiratory infection, bronchospasm.

C. Management

1. Rest, keep victim warm

2. Oxygen

3. Descent

4. CPAP mask

5. Medications which may be helpful (usually not necessary)

   a. Nifedipine 10 mg po stat, 30 mg extended release tid

   b. Acetazolamide 5 mg/kg/day

   c. Furosemide single dose of 40 mg

   d. Morphine small incremental doses

6. Victim may reascend when HAPE resolved

D. Pathophysiology

1. Non-cardiogenic; normal wedge pressures, high pulmonary artery pressure.

2. May be a form of neurogenic pulmonary edema (NPE).

3. May be a combination of high pressure and high-permeability edemas.

E. Prevention same as for AMS--acetazolamide apparently effective, also nifedipine recently shown good for prophylaxis.

V. HIGH ALTITUDE CEREBRAL EDEMA (HACE)

A. Diagnosed by ominous progression of cerebral AMS symptoms and findings of ataxia and change in consciousness.

B. Only effective therapy is descent; oxygen temporizes, drugs of little value, except perhaps Decadron given early.

C. Mechanism same as AMS-more advanced.

D. Prevention same as for AMS.

VI. RETINAL HEMORRHAGES

A. Common, affect vision only if in macula (rare).

B. Probably an hypoxic mechanism, leak is in capillary bed.

C. No treatment, no known prevention (acetazolamide?), clear spontaneously in 7-14 days.

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THE WORLD STATUS OF WILDERNESS MEDICINE

Coordinators: Blair Dillard Erb, M.D. and Bruce C. Paton, M.D.

Moderators: Peter Hackett, M.D., Oswald Oelz, M.D., Paul C. Auerbach, M.D.

Kenneth Kizer, M.D., Bruno Durrer, M.D., and Warren D. Bowman, M.D.

The **First World Congress on Wilderness Medicine** has been structured to address issues related to health and medical care of individuals who venture into remote and/or hazardous environments wherever they may be.

Wilderness is global in scope and knows no national boundaries. Since the aims and goals of medicine are the same the world around, and because nations do not contain identical geographic, environmental or medical features, a series of three 1½ hour sessions has been designed for participants to share their experiences and their perception of the principles of wilderness medicine with other health professionals from around the world.

Titled **"The World Status of Wilderness Medicine"**, these sessions are an effort to bring together the various elements that constitute the diverse fields of wilderness medicine. Since wilderness medicine is traditional medicine with no single discipline, this is an opportunity for the different specialties such as Cardiology, Pulmonology, Family Medicine, Emergency Medicine, Sports Medicine, etc., to examine their individual roles in wilderness medicine with a focus on the uniqueness of the requirements of medical care under the circumstances. Various organizations, institutions, centers, agencies, and leaders will discuss the current status of their activities in research, education, and service in wilderness medicine.

Attendees are invited to participate actively in these sessions. The objectives of the sessions will be:

1) To define the current world status of various aspects of wilderness medicine.
2) To outline needs for development of the field of wilderness medicine.
3) To propose mechanisms for enhancing research, education, and service in wilderness medicine.

Representatives from Mountaineering Medicine, Aquatic Medicine, Human Performance, Environmental Medicine (including heat, cold, and altitude stress), Medical Services, Public Health, and others will review their special areas of interest and stimulate discussion by participants.

Emphasis during these three **World Status** sessions will be on planning for cooperation for future development of medical resources in the broad field of wilderness medicine.

Please consider during your personal deliberations the question, **"Should the World Congress on Wilderness Medicine evolve into a consortium concerned with wilderness medicine?"**. Perhaps some form of loosely knit cooperative consortium could arise as a "United Nations" involved in wilderness medicine.

Please complete the enclosed questionnaire and bring it with you to the sessions. From this we hope to develop a directory. These are your sessions, and the product will be a result of your participation.

SCHEDULE

WORLD STATUS OF WILDERNESS MEDICINE

Monday, July 15 (2:00-3:30 p.m.)- **Mountaineering and Environmental Stresses.**
Moderators: Oswald Oelz, M.D.
Peter Hackett, M.D.

Tuesday, July 16 (2:00-3:30 p.m.)- **Aquatic Medicine and Environmental Stresses.**
Moderators: Kenneth Kizer, M.D.
Paul Auerbach, M.D.

Thursday, July 18 (2:00-3:30 p.m.)- **Delivery of Services including Search and Rescue and Emergency Care.**
Moderators: Bruno Durrer, M.D.
Warren Bowman, M.D.

Friday, July 19 (12:00-12:30 p.m.)- **Summary of the Proceedings.**
Moderators: Blair D. Erb, M.D.
Bruce C. Paton, M.D.

QUESTIONNAIRE

WILDERNESS MEDICINE ORGANIZATIONS

This questionnaire will assist in developing a directory of organizations and centers involved in wilderness medicine. It focuses on two types of groups: 1) Those whose primary mission is medicine in the wilderness setting, and 2) Those involved in wilderness activities which have a significant interest in medicine. Division into these groups is based on the degree of emphasis on the medical aspect of wilderness activity.

Each individual or organization is asked to complete the questionnaire and return to the Wilderness Medical Society staff. Thank you.

I. Division (Check One)

_____ Medical Organization with Wilderness Interest.

_____ Wilderness Organization with Medical Interest.

II. Organization

Name: _____

Address: _____

_____

_____

III. Chief Executive Officer of Organization

Name: _____

Address: _____

_____

_____

IV. Aim of the Organization (Check All That Apply)

_____ Education

_____ Research

_____ Service

_____ Other (Specify)

_____

_____

V. **Category** (Check All That Apply)

_____	Mountaineering	_____	Search and Rescue
_____	Environmental Stresses	_____	Research
_____	Aquatic Medicine	_____	Education
_____	Administration	_____	Other (Specify)

_____

VI. **Purpose** (Please Describe)

_____

_____

_____

_____

_____

_____

VII. Other organizations with which you are familiar that should be included in a directory:

_____

_____

_____

_____

VIII. For individual completing questionnaire:

Your Name: _____

Your Address: _____

_____

Your relationship to the organization you described:

_____

Please return to:

Blair Dillard Erb, M.D.
President, Wilderness Medical Society
1729 North Highland Avenue
Jackson, Tennessee 38301
USA
(901) 422-0330

NOTES FOR THE WILDERNESS MEDICINE RESEARCH SEMINAR

I. Find a question worth answering
  A. Attend conferences
    1. Find out what areas need investigating
  B. Observe in your own area
  C. Test hypotheses
  D. Try to study something within your area of interest or expertise

II. Figure out if it's feasible to study
  A. Can denominators be calculated?
  B. Do people all use one medical clinic?
  C. Do they start and finish at the same point?
  D. Can the problem be defined (such as rock-climbing hand injuries)?
  E. Would it be too expensive to study?

III. Fund it yourself if it's under $2000.
  A. Figure out your fixed expenses
    1. Printing, copying, mailing
    2. Computer software or computer expert
    3. Secretarial or research assistant help
    4. Having figures made (for publication)

IV. Design the study
  A. Define your study population
  B. Define a "case"
  C. Define the numerator
  D. Decide if it will be prospective or retrospective
  E. Who will collect the data?
  F. Design a questionnaire
  G. Design your computer database before you finish your quesionnaire. Make sure you understand the type of data you will collect at each point in the database. [Example: Are you in good physical condition? Give scale 1 to 10, words such as sedentary, occasionally exercise, exercise regularly, fanatic]
  H. Decide how long the study will run
  I. Define the authors in advance, and designate first author if that is possible. Get everyone's agreement in advance. This can cause a lot of trouble later if left unspoken.

V. Analyze your data
  A. Figure out every angle of what you have learned
    1. Tabulate, take means, etc.
  B. Don't go fishing for statistical significance [Give examples]
  C. Calculate statistical significance only when you had a hypothesis to test. [For example: You may have postulated that men have more skiing injuries than women because they

tend to ski faster. This can be tested statistically at the end of your study. But if you happen to detect that people who wear red jackets get hurt more often, you can't necessarily assume significance to that finding]

D. Decide on your major findings

1. You don't have to report on everything you learned

VI. Write your paper

A. Write a first draft including everything that you think of to say. You can always take it out later.

B. Organize the paper strictly along the lines of Introduction, Methods, Results, and Discussion. Read other papers with this in mind. Do not mix these sections. This is the most common mistake of new paper writers.

C. Make the introduction short

D. Do not justify your methods in the methods section, unless there is an overwhelming reason to do so.

E. Give the results as plainly as possible, using appropriate graphs, charts, etc. Simplify whenever possible.

F. Use the discussion to point out what you have learned, the strengths and weaknesses of the study, its significance to the world of wilderness medicine, possible advice to the study population based on your research, and possible future studies.

G Resist witticisms and the desire to be too clever, unless you are so overwhelmingly clever that it can't be resisted.

VII. Submit your paper

A. You can only submit to one journal at a time

B. The review process can take several months

C. So choose carefully where the article might have a good chance

D. Follow the "instructions for authors" for the selected journal carefully. Write a cover letter explaining why your paper is significant, and have your co-authors sign the required statements, if any.

E. Don't get angry at the reviewer's comments; they are told to be critical.

F. Expect to have to change a lot of the article, or shorten it drastically.

VII. Article or letter?

A. Articles much more significant in terms of CV

B. Letters much easier to produce and to get published.

C. Letters get published more quickly

D. Letters best medium for brief case presentations

E. Articles carry more weight because they are peer reviewed.

DETAILS:

1. Computer database programs
2. Statistical programs, such as Epiinfo
3. Choosing the right statistical test: review very basic tests, such as comparing two ratios, or comparing means.
4. Informed consent and releases, if necessary
5. How to make figures, graphs, etc. (Harvard Graphics or hire an artist)
6. Epidemiologic methods: mechanisms of bias in selection of study groups, populations, clinics, control groups. For example: do all ski injuries go to the first aid hut first, or do the more serious ones go straight off the mountain?

Work through an example, such as the one I suggested, studying whether men lose more weight on trek than women. In general keep the whole presentation simple, direct, practical. We have two 90 minute sessions. The first session could be an overview as outlined above. The second session could be working through a sample study, and dealing with details and questions.

WORKSHOP ON COLD WEATHER SURVIVAL
FIRST WORLD CONGRESS ON WILDERNESS MEDICINE
Whistler Mountain, British Columbia,
July, 1991
Warren D. Bowman, MD

The cold weather survival workshop will be conducted in the snow as much as possible. If snow conditions are unsuitable for snow shelter construction, model shelters will be demonstrated. Please try to attend the didactic session on cold weather survival or at least read the syllabus material before attending this workshop.

Objectives: After attending this workshop, the student should be able to:

1. Explain the layer system of clothing and describe preferred fabrics, clothing items, and layering sequences for cold weather outdoor travel.
2. Appreciate the insulating capacity of snow; describe and demonstrate the principles and building techniques of emergency snow trenches and multiday oversnow travel snow trenches, snow caves, igloos, and lean-tos.
3. Describe and demonstrate the techniques of gathering fuel and starting a fire under winter conditions.
4. List recommended items of winter survival equipment.

Since the end of World War II, more and more people have been venturing out into the winter wilderness to pursue hunting, snowmobiling, cross-country skiing, winter mountaineering, alpine skiing, winter camping, and other cold weather sports. Growth in expeditionary mountaineering places many climbers in regions where winter exists all year round. When modern conveniences are left behind, life is reduced to the basics and individuals become completely dependent on their own resources. Our ancestors' hard-won knowledge of how to survive in the cold may need to be relearned, sometimes at considerable cost.

This workshop will emphasize preplanning, anticipation, always carrying a minimum of emergency survival equipment, and prevention of environmental emergencies rather than panic-stricken improvization.

The most important human organ for survival is the brain, since voluntary actions such as preparedness, regulation of energy expenditure, adjustment of clothing, and seeking shelter are more important than involuntary mechanisms of adaptation to heat or cold. Since regulation of core temperature is the most important short-term goal in cold weather survival, the subject must either be carrying enough insulation or know how to improvise enough insulation and/or provide enough external heat to maintain a satisfactory core temperature for an indefinite period of time.

Since decreasing heat loss through intelligent use of insulation

is the most energy efficient method of avoiding dangerous degrees of cooling, it will be discussed and demonstrated in detail.

Clothing should be worn in a number of thin layers so as to trap several layers of still air, which are warmed by body heat. The most effective fabrics are those which trap air and prevent its motion, and whose effectiveness is not reduced by becoming wet. The best of these currently are wool, polypropylene, and such types of treated polyester as Capilene and Thermax. Cotton has poor insulating value, especially when wet, and should be avoided in cold weather. Orlon, polyester, acrylic, and similar synthetics are also good insulators. The above fibers are typically worn as the innermost 2 or 3 layers---long underwear, shirt, sweater, and pants. Outer layers are normally chosen from fibers that produce loft, such as polyester and nylon pile and fleece, down, Dacron, Hollofil II, Quallofil, Thinsulate, and Thermolite. These are made into jackets, vests, and overpants. The outermost layer is preferably of a windproof and water resistant fabric such as Gore-Tex, a nylon/cotton blend, or similar material. Two or three layers are used when skiing, hiking or building shelters, with a fourth layer added when stopping for lunch or a rest.

SAMPLE COLD WEATHER CLOTHING:
a 4-layer system

Underwear: 100% wool or 85% wool/15% nylon is the gold standard, but hard to obtain. Thermax, Capilene, polypropylene and blends of these are the new standard. Combinations such as Duofold and Thermolactyl are less warm.

Shirt: Wool, Capilene, polypropylene and similar materials are good; they should open completely in front or at least have a half-zipper.

Pants: The hard-finish wool pants found in military surplus stores are durable and reasonably priced. Knickers are preferred by some.

Parka: The most versatile combination is a windproof and water-resistant shell worn over a pile jacket. The pile jacket should have a nylon outer cover to prevent snow sticking to it when it is worn alone. The shell should have a hood with drawstring closure and be fingertip length. Metal zipper pulls and metal snaps should be shielded or situated so they do not touch bare skin and zippers should have a weather flap. "Pit-zips" are useful. Handwarmer pockets should be included and the main pockets should be accessible when you are wearing a fanny pack or backpack with the waist belt fastened.

Wind Pants: These are a must for cold, windy weather or for digging a snow cave. They should be made of windproof and water-resistant material.

Fourth Layer: for very cold conditions, a down, Dacron,

Thinsulate, etc., filled quilted garment such as a jacket or vest, and a pair of pile pants.

Socks: A good combination is a pair of polypropylene socks next to the skin with 1-2 pairs of heavy, wool socks over them.

Hat: A wool, polypropylene or acrylic stocking-type that can be pulled down to cover the ears. Unless a neck gaiter is worn also, choose a hat with a face mask or balaclava feature.

Mittens or Gloves: Mittens are warmer than gloves but are less useful when delicate finger movements are required. Several manufacturers make three-layer mitten and glove sets that include windproof shells with leather palms and two sets of removable pile inner layers that velcro in place. Polypropylene glove liners are useful when working on ski bindings and doing other delicate work.

Boots: The choice depends on the type of activity and the expected temperature. Double boots generally are best for cold temperatures. They can be winter mountaineering boots, telemarking boots, ski mountaineering boots, or non-technical boots such as the Sorel.

Rain Gear: In moderate climates or very wet conditions where rain or wet snow may be encountered, you may prefer to carry one set of windproof outer garments plus a second set of waterproof ones.

Vapor Barrier Garments: These are waterproof socks, underwear tops and bottoms, and sleeping bag liners worn over a thin polypropylene garment. This system traps a warm film of moisture next to the skin, theoretically decreases water requirements by reducing sweating, and maintains the insulating properties of outer garments by keeping sweat out of them. Try them under non-survival conditions before you commit yourself to them since some don't like them.

Anyone who ventures out of doors in cold weather should carry spare clothing for the most extreme environmental conditions likely to be experienced.

The ability to improvize a wind and cold-proof survival shelter is an essential survival skill. If the snow cover is adequate, this should be constructed of snow, which is a very good insulator with heat conductivity 1/10,000 that of copper and about the same as wool felt (Table 1). Satisfactory survival snow shelter designs should allow quick construction without wetting or chilling the builders. Every cold weather survival kit should contain a collapsable snow shovel of the small grain-scoop type, and a plastic or nylon tarp measuring about 8 X 10 feet. With the shovel, a triangular trench 4-6 feet wide, 6-8 feet long, and 2-3 feet wide at the narrow end can be dug in about 20 minutes. The wide end is roofed over with the tarp and a fire built at the narrow end. You lie snug and warm

under the tarp on your pack or natural insulation such as evergreen branches.

Snow caves are comfortable and warm but the classic type with a small, narrow entrance takes at least two hours to dig during which the builders usually become quite wet. You should have both a small grain scoop and a short, flat shovel of the French type for best results. A better and faster survival cave is an open snow-hole dug into the side of a hill, with the opening closed with snow blocks. If this option is elected, a snow saw is a useful tool since cutting blocks with a shovel or ski tail is difficult. Igloos are the Taj Mahal of snow shelters but require practice and some engineering skill to erect properly and quickly.

If the snow cover is light, the best emergency shelter is probably an open lean-to made of the tarp, with a reflector fire built in front and the sides closed with brush.

In the author's opinion, the most practical snow shelter for multiday deep snow camping is a special trench design that allows for the maximum of snow around and above the occupants. Such a trench can be built on the level or a slight incline. The door, which opens onto an entranceway where packs are kept, is narrow like that of a snow cave. The trench is 4X8 feet at the surface, and is undercut so that the bottom is 6X10 feet. It should be at least 3 feet deep. Two pairs of skis and ski poles are placed on top and the tarp laid over these, with snow piled around its edges to hold it down. If the trench will be used as a basecamp, poles can be cut and used instead of skis and ski poles. Above timberline, snow blocks can be laid over the skis instead of the tarp.

With the entrance closed with a poncho, two candles alone will warm the inside of the trench to >20oF no matter what the outside temperature. You can safely cook inside the trench if you place a ventilation hole above the stove. The author has slept comfortably in such a trench at -19oF.

The ability to build a fire under adverse conditions is another important survival skill that needs to be practiced. A cold weather survival kit should contain water proof matches, a candle, firestarter, a sturdy knife, and a collapsable saw. The fire should be laid out of the wind. Tinder---shavings or the small dead branches on lower trunks of evergreens---is laid against a large dead branch in lean-to fashion, with the small twigs on the bottom and larger ones on top. All the tender is arranged so that air can reach each piece. A candle or fire starter is needed if the wood is damp; if wet, the wood should be shaved or split as well. The amount of wood needed to last all night is easy to under-estimate, and is two or three times more than you think.

Because of the time and fuel required to melt snow or ice for water, advantage should be taken of open water when found. It should be treated by filtration, chemical disinfection, or boiling if there

is a question about its purity. Simply bringing water to a boil is effective in killing Giardia cysts, pathogenic bacteria and viruses at altitudes below 18,000 feet (5500 meters).

LITTERS AND LITTER PACKAGING

Michael V. Callahan, MSPH
Ministrie des Montagnas-Alpine Guard
National Association for Search and Rescue
Cambridge, Massachusetts U.S.A.

OBJECTIVES

Following this practical skills workshop, participants will be able to:

1. Properly assess and package the injured patient in soft, rigid, and improvised rescue litters.
2. Construct semi-rigid, vertically-loaded litters from expedition materials (Vauhgn system).
3. Develop a coordinated litter evacuation plan with regard to patient injuries, team resources, and terrain.
4. Transport the loaded litter over moderate terrain with rescue and improvised equipement

IMPROVISED SELF RESCUE FOR THE SPORT CLIMBER

Lanny Johnson, RN, EMT-P

One of the worst situations a mountaineer can face is having a leader fall with an injured climber hanging on the rope above the belayer.

This workshop will cover techniques for rescuing the fallen/incapacitated climber with common climbing gear.

Participants will observe demonstrations of the "Counter Balance Rescue" system as well as some simple raising and lowering systems using climbing slings and pulleys.

Objectives

1. Become familiar with technical self rescue techniques.

2. Learn knots to use during self rescue operations.

AN OVERVIEW OF HELICOPTER RESCUE IN BRITISH COLUMBIA

British Columbia, located on the west coast of Canada, is a region of arid plain, timbered hills and snow capped mountain peaks. The province extends over an area of approximately 800,000 sq. km. and extremes of weather and terrain may be encountered at any time of the year.

The majority of British Columbia's 3.25 million inhabitants live in the south western corner of the province and in a number of smaller centres. The industry and recreation of the province create a venue where accidents are fairly common and when rescue can be assisted by helicopter, a multi-agency, task force rises to the occasion.

National Search and Rescue Program

Canada is a member of both the International Maritime and International Civil Aviation Organizations. As signatory to the conventions on life saving established by these organizations, the federal government maintains a highly efficient search and rescue (SAR) program.

In Canada, the lead federal ministry for SAR is the Department of National Defence. This organization is responsible for providing the aviation component of SAR, with marine SAR assets being provided by Transport Canada Coast Guard.

Armed Forces SAR helicopters are based in each of the four SAR regions in Canada. The Pacific Region, which includes all of British Columbia as well as a substantial area of open ocean is approximately 1.6 million square Km. in size.

The 442 Transport and Rescue Squadron, located at Comox on Vancouver Island, is equipped with five Boeing Labrador Helicopters and five DeHavilland Buffalo STOL aircraft. In addition, electronic top cover is frequently provided by Aurora anti-submarine aircraft also based at Comox.

In 1990, the 442 Rescue Squadron responded to 45 marine emergencies and approximately 45 rescue incidents ranging from injured climbers to aircraft accidents.

In the National Parks, the Canadian Parks Service conduct helicopter rescue with Bell 206 and Hughes 500 aircraft under commercial contract.

In 1990, the Park Warden Rescue Service carried out 12 helicopter flight rescue system (HFRS) extractions in the British Columbia side of the Rockie Mountains and the Pacific Rim National Park on the West Coast of Vancouver Island.

Civilian Helicopter Rescue in British Columbia

Because of the distances involved in British Columbia the SAR program provided by the federal government is frequently augmented by aircraft and personnel from the commercial sector.

Rescue capability is usually in the form of a fixed-rope suspension system (HFRS) attached to Bell 206 Jetranger aircraft. This requires that the rescuers and patients be transported on the end of the rope system outside the aircraft. An improvement over HFRS is now being offered by one helicopter company which is operating a MBB BK 117 aircraft equipped with a personnel rescue hoist.

These aircraft may be chartered by the Royal Canadian Mounted Police, The Rescue co-ordination centre (military), Provincial Ambulance Service, or the Provincial Emergency Program.

In British Columbia, Workers Compensation Board regulations make the employer responsable for the initial evacuation of injured workers to medical attention. Accidents in resource based industries such as logging and Hydro electric projects may require helicopter rescue (HFRS) and a number of companies have developed this capability.

The British Columbia Forest Service has a well established forest fire attack program which utilizes helicopter rappelling for rapid response. A HFRS capability is also available and although intended for the rescue of Forest Service personnel, can be used in the event of non-fire related emergencies.

Already offering a highly efficient air ambulance service, the Provincial Government is presently investigating a province wide rescue/medical helicopter program. Currently, the British Columbia Ambulance Service conducts more than 6200 helicopter and fixed wing medevacs each year.

Over the years, the task of providing an efficient and safe helicopter rescue response has evolved into a co-operative multi-agency effort. It is indeed reassuring to know that in British Columbia, when a request comes in for the rescue helicopter it can be answered with all the dedication, skill and speed possible.

The HEALING FOREST

The Search For New Jungle Medicines

BY MARK J. PLOTKIN

The rate of extinction is increasing at an alarming pace. The majority of the world's threatened species—both animal and plant—inhabit the tropical forests. These forests cover only 2% of the earth's surface, but are believed to contain more than half the world's species. Among the devastating impacts of the current destruction of tropical forests will be the loss of raw materials for future medicines and the loss of the knowledge needed to utilize them. Rain forest plants are complex chemical storehouses that contain many undiscovered biodynamic compounds with unrealized potential for use in modern medicine. We can gain access to these materials only if we study and conserve the species that contain them. It is estimated that some 250,000 species of flowering plants exist worldwide, 90% of which are already known to science. Nevertheless, expeditions to the tropics, particularly the Amazon region, continue to bring back new species.



MARK J. PLOTKIN

The flora of the tropics faces serious threats. For example, as much as 95% of the Atlantic coastal forest of eastern Brazil has already been destroyed, according to Russell A. Mittermeier of the World Wildlife Fund. On the island of Madagascar, where it is believed that 80% of the flowering plants exist nowhere else on earth, well over half of the original forest cover has been removed or seriously disturbed, says Mittermeier. In the Hawaiian archipelago, where more than 90% of the plant species exist nowhere else, as much as 14% are believed to be extinct.

NATURAL & UNNATURAL EXTINCTION

Extinction is a natural process. Since the origin of life three billion years ago, many species have disappeared. Yet to view these recent extinctions as natural is to misinterpret the geological record. E.O. Wilson of Harvard University postulates that the present rate of global species extinctions is 400 times faster than in the recent geological past and that this rate is rapidly accelerating. The only similar examples in the history of this planet were the massive species die-outs at the end of the Paleozoic and Mesozoic eras.

A striking feature of these historic natural disasters, however, is that the extinctions were of primarily animal rather than plant species. In the past, plants were presumably more resistant to extinction than the dominant animal life forms such as dinosaurs. Consequently, plant diversity has increased through time. The unpleasant conclusion is that man is causing the first major reduction of global plant diversity since the origin of life.

PLANTS AND MEDICINE

Plants have traditionally served as man's most important weapon against the bacteria that cause disease. It seems that even the Neanderthals made use of medicinal plants. As early as 2000 B.C., the Chinese were using molds to treat festering ulcers, and the ancient Egyptians are known to have applied moldy bread to open wounds.

It is only relatively recently, with the advent of modern technology and synthetic chemistry, that we have been able to reduce our almost total dependence on the plant kingdom as a source of medicine. Nonetheless, we continue to rely on plants to a much greater degree than is commonly realized. Almost half

of all prescriptions dispensed in the United States contain substances of natural origin. More than 50% of these medicines contain a plant-derived active ingredient. In 1974 alone, the United States imported $24.4 million worth of medicinal plants.

Only a fraction of plant species have been investigated in the laboratory. This poor understanding is particularly acute in the tropics. Brazilian plant chemist Otto Gottlieb wrote in 1981, "Nothing at all is known about the chemical composition of 99.6% of our flora." It is worth noting that Brazil probably has more species of flowering plants — approximately 55,000 — than any other country on earth.

ETHNOBOTANY

Ethnobotany is the study of tribal peoples and their utilization of tropical plants. The importance of ethnobotanical inquiry as a cost-effective means of locating new and useful tropical-plant compounds cannot be overemphasized. Most of the secondary plant compounds employed in modern medicine were first "discovered" through ethnobotanical investigation. There are some 119 pure chemical substances extracted from higher plants that are used in medicine throughout the world, and 74% of these compounds have the same or related use as the plants from which they were derived.

The rosy periwinkle represents a classic example of the importance of plants used by local peoples. This herbaceous plant, native to southeastern Madagascar, is the source of over 75 alkaloids, two of which are used to treat childhood leukemia and Hodgkin's disease with a very high success rate. Annual sales of these alkaloids worldwide in 1980 were estimated to reach $50 million wholesale prior to 100% markup for the retail market, according to International Marketing Statistics. This species was first investigated in the laboratory because of its use by local people as an oral hypoglycemic agent. Thus, we can see that investigation of plants used for medicinal purposes by "unsophisticated" peoples can provide us with new biodynamic compounds that may have very important applications in our own society.

NATIVE KNOWLEDGE

Tropical-forest peoples represent the key to understanding, utilizing, and protecting tropical-plant diversity. The degree to which they understand and are able to sustainably use this diversity is astounding. The Barasana Indians of Amazonian Colombia can identify all of the tree species in their territory without having to refer to the fruit or flowers — a feat that no university-trained botanist is able to accomplish.

What can modern medicine learn from the witch doctor? Much more than one might think. Medicine men usually have a profound knowledge of tropical plants and the healing properties for which they may be employed. A single shaman of the Wayana tribe in the northeast Amazon, for example, may use more than a hundred different species for medicinal purposes alone. Furthermore, a great



MARK J. PLOTKIN

This Tirió Indian relaxes by playing the flute. The Tiriós use more than 300 forest plants as medicine.

many of the remedies are effective. Fungal infections of the skin are common in the humid tropics, and modern medicine can only suppress — not cure — serious cases. On more than one occassion, I have had serious infections successfully treated by shamans using jungle plants.

Unfortunately, however, the oral tradition of these medicine men is not being passed on to the next generation. With the advent of Western medicine in many of these remote areas, young tribal members demonstrate little interest in learning the traditional ethnomedical lore. Of all the shamans with whom I have lived and worked in the northeast Amazon, not a single one had an apprentice. We are, in my opinion, facing a critical situation — unless we act now, thousands and thousands of years of accumulated knowledge about how to use rain forest plants is going to disappear before the turn of the century.

THE SEARCH FOR NEW JUNGLE MEDICINES

Many reasons for species conservation have been presented to the general public — aesthetic, ethical, etc. — but one of the most relevant is the utilitarian: that species are of direct benefit to us. The few examples I have mentioned are indicative of the kinds of undiscovered compounds that are undoubtedly out there.

We now know that synthetics are not the only answers to our medical needs. European pharmaceutical firms are showing renewed interest in the potential of the tropical flora; heightened awareness exists in the United States as well. The National Cancer Institute awarded more than $2.5 million in contracts to the New York Botanical Garden, the Missouri Botanical Garden, and the University of Illinois to collect and test tropical-plant species for anti-tumor activity.

We must also consider the importance of medicinal plants in the developing countries themselves. The World Health Organization has estimated that 80% of the people in the world rely on traditional medicine for primary healthcare needs. In many cases, developing countries simply cannot afford to spend millions of dollars on imported medicines that they could produce or extract from tropical-forest plants.

Several African and Asian nations have begun to encourage traditional medicine as an integral component of their public healthcare programs. Indigenous medicines are relatively inexpensive, locally available, and usually readily accepted.

The ideal scenario for the future would be the establishment of local pharmaceutical firms in developing countries of the tropics. These firms would create jobs, reduce unemployment, reduce import expenditures, and generate foreign exchange. Beyond the economic advantages, however, such enterprises would encourage documentation of traditional ethnomedical lore and would promote the conservation and sustainable use of the tropical forest. ▯

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HIGH ALTITUDE NEUROLOGIC PROBLEMS

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LEARNING OBJECTIVES:

Following this presentation, participants will be able to:

1. Recognize clinically the neurologic syndromes that may present at high altitude, including high altitude cerebral edema, cerebral vascular spasm, stroke, and TIA.
2. Understand the pathophysiology of neurologic syndromes at altitude.
3. Discuss the proven and hypothetically effective treatments for these neurologic syndromes.
4. Appreciate the current research on pathophysiology and the new methodologies being employed to study these syndromes.

I. NEUROLOGIC SYNDROMES CAUSED BY HIGH ALTITUDE

A. Acute mountain sickness (AMS) and high altitude cerebral edema (HACE)

1. Pathophysiology

a. Probably the same pathophysiology, with HACE being the end-stage.

b. Possible brain swelling mechanisms:

- Intracellular Na^+ and H_2O accumulation.
- Vasogenic edema: Cause of permeability?
- Role of cerebral blood flow

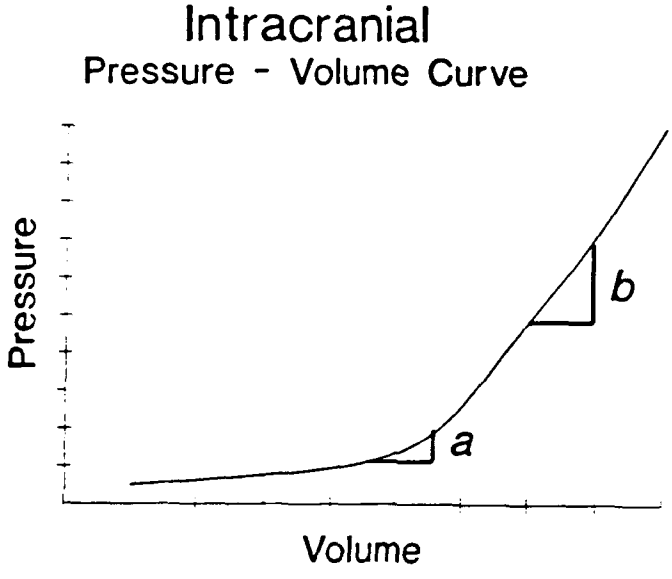
B. High altitude neurophysiology

1. Intracranial dynamics

a. Intracranial contents

- Average brain 1500 mls

- Average blood 75 mls
- Average CSF 75 mls
- Semi-elastic membranes not very distensible
- Volume-pressure relationship
- Volume can increase some without increased pressure



b. Cerebral blood flow

- If CO_2 constant, CBF inversely related to SaO_2%
- At altitude CBF varies widely, since CO_2 as well as SaO_2 depend on ventilatory response
- Xenon techniques show 20-40% increase of CBF in days 1-3, then return close to baseline despite hypocapnia
- Transcranial doppler shows little change in CBF

c. Cerebral blood volume

- Function of vascular tone
- Increased with vasodilation

d. Intracranial pressure (ICP)

- Increase in ICP in animals at altitude quite variable

- ICP clearly elevated in severe acute mountain sickness

- Both cytotoxic and vasogenic mechanisms may be operant

- Preliminary study indicates ICP **not** cause of mild AMS

2. Cerebral O_2 delivery

a. O_2 delivery = CBF X CaO_2 (arterial O_2 content)

- CaO_2 = Hb X 1.34 X SaO_2%

- Example: a 10% drop in SaO_2 and 10% increase in CBF = no net change in cerebral O_2 delivery

b. Is the brain always hypoxic at high altitude?

- O_2 delivery may be maintained

- Cerebral venous PO_2 normal until SaO_2 < 70% (5,000 meters) in normocapnia, but hypocapnic vasoconstriction at altitude changes this.

- O_2 extraction varies among individuals, may adapt.

c. Matching of O_2 delivery with $CMRO_2$

- Animal studies show $CMRO_2$ goes up transiently but perfusion and O_2 delivery exceed needs: "luxury perfusion" or damaging overperfusion?

3. Effects of hypoxia on CNS

- Neurotransmitter failure; e.g., tryptophan hydroxylase

- Cell membrane dysfunction, cell swelling

- Synthesis of various peptides, proteins: "hypoxic shock" model

- Effect on energetics? ATP probably not decreased

C. Neurologic Syndromes

1. Acute mountain sickness and high altitude cerebral edema

a. Neurologic symptoms and signs

- Headache, vomiting, sleep disorder, ataxia, altered mental status

b. Pathophysiology

- Overhydration of brain, hypoxic brain, or both?

- Fluid retention systemically; ADH, ACTH, aldosterone

- Fluid shift into cells (cytotoxic); Na^+/K^+ pump dysfunction?
- Leakage from vessels from overperfusion (vasogenic) or permeability change?
- Direct effects of hypoxia or brain fluid dynamics?
- Hypoxic effects on neurotransmitters or metabolism

D. Treatment

- Descent or O_2
- Dexamethasone very effective, earlier the better
- Acetazolamide effective early; less dramatic improvement than dexamethasone
- Diuretics (furosamide) also shown effective

E. Prevention

- Acclimatization
- Supplemental O_2
- Acetazolamide or dexamethasone

II. FOCAL NEUROLOGIC CONDITIONS

A. Stroke

- Apparently high incidence at high altitude
- Probably multifactorial
- Dehydration, polycythemia, brain compression?
- Possible role of vasoconstriction, vasospasm
- Cerebral venous thrombosis more common

B. Reversible neurologic deficits

1. Symptoms and signs

- Cortical blindness

- Hemiparesis and hemiplegia

- Aphasia

- Various reversible focal signs

2. Pathophysiology

- "Hypoxic/eschemic/thromboembolic"

- Migraine equivalent?

- True TID?

- Hypocapnic vasoconstriction?

3. Treatment

- O_2 or CO_2 breathing (or both)

- Descent

- NSAID's?

- Complete evaluation to rule out cerebrovascular pathology

4. Conditions exacerbated by high altitude

a. Seizure disorder

- Reports of 1st onset seizure at altitude

- Increased seizures in those not on medication

- No reports of increased seizures in those controlled on medication

- Mechanism: Hyperventilation?

b. Multiple Sclerosis

- Any alterations of blood brain barrier may exacerbate MS

- Few case reports, need study

c. Brain tumors

- Become symptomatic at altitude

- May be true of any space occupying lesion

- Probably secondary to brain swelling/vasodilation

d. Cerebrovascular lesions

- AVS's and aneurysm may bleed

- Secondary to vasodilation, increase venous pressure?

- Perhaps a contraindication to sleeping of 3,000 meters

e. Cerebral arteriosclerosis

- Case reports of patients marginally compensated at sea level becoming symptomatic on ascent to altitude.

- Normal area of brain may vasodilate, "steal" from area of fixed circulation.

MEDICAL PROBLEMS OF SPACE FLIGHT

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Despite the highly technological aspects associated with manned spaceflight and its emphasis on precise control, there are some facets over which we have little influence and yet which have large impact on the way we operate. While we are able to control environmental factors such as the total pressure and composition of the cabin atmosphere there is one factor we don't control at all - gravity. This absence of gravity turns out to be the root cause of virtually all the physiologic problems a crewmember is likely to encounter during flight. The major areas of concern and the operational problems to which they contribute follow.

VESTIBULAR

First-time exposure to 0-g (weightlessness) has resulted in an attack rate of Space Motion Sickness of 67% in Shuttle crews. The etiology of this syndrome is still not clear. Sensory conflict seems to be the most likely cause although factors such as fluid shift may play a role. The severity of symptoms is highly variable but most are manifest and subsequently resolve within the first 2-3 days of flight. The symptoms resemble those of motion sickness though certain significant differences occur.

CALCIUM

Bone demineralization is known to occur on exposure to 0-g and continues at a steady rate for many months. There is some evidence that a new equilibrium is reached after many months but this information is only preliminary. While the bone demineralization which occurs poses no problem while the crewmember is in a 0-g environment, return to a 1-g environment after a long exposure to 0-g would have a higher associated risk of spontaneous (or pathologic) fractures. While this problem is not significant for Space Shuttle operations, future Space Station and interplanetary missions will be tremendously impacted by this problem.

CARDIOVASCULAR

Exposure to 0-g causes a redistribution of intravascular volume within the body. This fluid shift begins immediately upon achieving the weightless condition. The body adapts to this 0-g environment rapidly, in less than 2 days, and as a result the intravascular volume shrinks. It is also postulated that with the loss of orthostatic stress in 0-g the autonomic response to an orthostatic load on return to 1-g becomes blunted. Although this reduction in intravascular volume and autonomic response to orthostatic

stimuli is not a problem while at 0-g, return in a precipitous fashion to a 1-g environment (i.e. re-entry and landing of the Space Shuttle) can have catastrophic results. As a result, countermeasures are used to minimize the symptoms crewmembers experience on return to 1-g. These countermeasures include fluid loading and anti-g suit use. While these countermeasures are effective, further investigation into other methods of protection is ongoing.

At the present time our ability to function during Space Shuttle flights is not significantly impacted; however a better understanding of the previously mentioned physiologic changes would serve to make the flight environment more hospitable. While this increased insight is desirable for Shuttle flights, it will become absolutely mandatory for long-duration operations such as those contemplated for Space Station or a Mars Mission.

Designing a Health Care System for Space Flight

Joey B. Boyce M.D.
Former Manager, NASA Health Maintenance Facility
Houston, Texas, USA

Learning Objectives: Following this presentation, participants will be able to -

1. Have a better understanding of the issues and dilemmas involved in development of a health care system for long duration space flight.

2. Understand the limitations of microgravity medical care and the effects of space flight on humans.

3. Appreciate where some of their tax dollars are going.

Designing a Health Care System for Space Flight

Joey B. Boyce M.D.
Former Manager, NASA Health Maintenance Facility
Houston, Texas, USA

Abstract -
The development of a health care system for space environments demands many modifications of existing terrestrial practices. Weight, power consumption, size, and personnel restrictions force adaptation of equipment, techniques, and protocols for preventive, diagnostic, and therapeutic medical care. Maximizing return from scarce resource allotments requires numerous trade studies and balancing of risk against benefit prior to development of basic system requirements. Cost of providing a certain level of care must also be balanced against larger program goals and capabilities. This paper will discuss some of these issues, and current approaches to medical care in the development of the International Space Station Freedom Crew Health Care System (CHeCS).

Introduction -
The Crew Health Care System (CHeCS) of the proposed International Space Station Freedom will provide essential operational biomedical support needs for the Freedom crews, including medical care, environmental habitat monitoring, and countermeasures for the potentially maladaptive physiologic effects of space flight. To meet these challenges, CHeCS will consist of three integral parts, the Health Maintenance Facility (HMF), the Environmental Health System (EHS), and the Exercise Countermeasures Facility (ECF). The unique microgravity closed environment of Space Station Freedom will force adaptation of standard Earth based procedures in each one of these areas. In addition, each CHeCS area will operate under significant constraints of power, weight, volume, and other operational limitations. At present, design requirements have been defined by NASA for CHeCs which will allow construction and operation no later than the permanent manned capability (PMC) phase of the Freedom Station in 1999 (1). The McDonnell Douglas Space Station Corporation has been chosen as the prime contractor for development of the CHeCS equipment, with technical and scientific guidance provided by NASA. Many areas that must also be considered for a complete picture of Space Station Freedom Medical Operations will not be discussed in depth

in this paper. A brief background of previous long duration space flight, physiologic effects of space flight, and the Space Station Freedom program is needed for understanding the basis upon which CHeCS is founded.

Background

The United State's experience to date in long duration space flight is limited to the three Skylab flights, the longest of which was 84 days. The Soviet MIR program has had flights over one year, and has gained invaluable experience as a result. Planned CHeCS equipment and technology will be significantly more sophisticated and more capable than either of these programs. For Skylab, crew medical officers were limited to only rudimentary medical procedures and first aid. No extended inpatient care was possible, and any medical event beyond a minor problem might have required a return to Earth for all crewmembers. Environmental monitoring of the 5 psia, 70% oxygen/30% nitrogen Skylab cabin atmosphere was limited to oxygen and carbon dioxide sensors on orbit, with detailed post mission analysis of other constituents. In addition, life support monitoring did not have to take into account the recycling of resources that will be an essential feature of Space Station Freedom. Exercise facilities and monitoring on Skylab were likewise less capable in comparison with Freedom, with little real time physiologic monitoring of crew's performance.

Despite the previous work done during US and Soviet flights, none of the physiologic changes that accompany space flight are well understood at this point. On short term flights, approximately two-thirds of U.S. space travelers have been afflicted by Space Motion Sickness (SMS), a self limiting malady that varies in severity from mild headache to nausea and vomiting (2). Fortunately, SMS typically last no more than three to four days, and should not be a problem during longer flights. Microgravity exposure over a period of more than one to two weeks causes several physiologic changes, including progressive bone mineral losses and musculoskeletal deconditioning. At present, exercise is the "prescription" of choice as a countermeasure to this deconditioning, although it is not clear how effective it will be in preventing long term losses or performance decrements. The effects of space flight must be added to the "routine" occupational and medical risks of the crew for a complete medical risk appreciation.

Although only approximately 250 nautical miles away from the Earth's surface in low Earth orbit, the Freedom Station will be an extremely isolated habitat that must be self sufficient. As shown in Figure 1, Freedom Station will consist of 4 cylindrical modules connected by "nodes" in a building block fashion over many flights of the Space Shuttle. Two of the smaller modules and all the nodes will be supplied by the United States, with the European Space Agency (ESA) and Japan's NASDA providing one full size module each. Essential station support services (power, thermal control, etc) and launch systems via the Space Shuttle

will be provided by the United States in this international cooperative effort. The Freedom systems are targeted for a 30 year lifespan at a minimum. Missions may last from 45 to 180 days or more. In the event of a contingency or missed resupply mission, essential backup supplies and equipment (including CHeCS) will be distributed throughout the Station. At present, there are no provisions for a return to Earth capability other than the Space Shuttle, although the NASA is currently investigating an Assured Crew Return Vehicle (ACRV) "lifeboat" for crew return in the event of contingencies. Life support for the Freedom Station will be supplied by the Environmental Control and Life Support System (ECLSS), which has been described previously (3). This system will provide a respirable atmosphere, hygiene and potable water, and other essential needs for the crew while recycling crew wastes and by-products to the maximum extent possible.

The Health Maintenance Facility

The medical care arm of CHeCS is provided by the Health Maintenance Facility (HMF), a clinical care facility for inpatient care of one crewmember and outpatient care of an entire crew of four for no more than three days. In present configurations, the HMF is located in a center node. Essential HMF equipment will take up the majority of one Space Station double rack. A diagram of HMF equipment in the current anticipated configuration is shown in Figure 2. The HMF will also be able to provide medical care in the Freedom hyperbaric airlock at up to 2.8 atmospheres absolute pressure for treatment of decompression sickness and air embolism suffered during extravehicular activity (EVA).

The subsystems that make up the HMF and their components are shown in Table 1. The determination of the appropriate mix and types of equipment for the HMF has been the subject of considerable effort since the inception of the Freedom program. Despite considerable demand for a numerical cost/benefit determination for each piece of equipment, it was not appropriate to "pare down" the myriad number of medical scenarios and base the HMF equipment list on what is needed in an arbitrary "top ten" (or one hundred). Instead, the focus was on general medical functions that would be needed in selected medical fields (i.e. equipment needed for basic cardiopulmonary diagnosis and treatment). In effect, a "rule out" process has been used to exclude equipment or functions that were not feasible. For instance, computerized tomography (too large), many specialized laboratory procedures (rare use), and major surgical treatments (appropriate personnel and equipment not feasible) are all beyond the scope of practical medical care in the Freedom Station program. In addition, the personnel may also have limited training. The HMF will support, however, the vast majority of medical problems that may arise.

The Environmental Health System

To ensure a safe and healthy environment for station crewmembers, the Environmental Health System (EHS) will regularly monitor the cabin atmosphere, potable water supplies, and other general environmental health parameters.

The EHS will be composed of six subsystems: Microbiology, Toxicology, Water Quality, Radiological Health, Vibroacoustics, and Barothermal Physiology (4). In present plans, the EHS system has one rack in a central node location, with portable monitoring equipment distributed throughout the Station's main elements for continuous monitoring of combustible products and total hydrocarbons. The node rack mounted EHS equipment will provide analysis of samples of air, water, microbial swabs, and other samples from various station sites on a regular schedule. Special monitors for specific toxins or experiments are also expected to augment the more general monitoring equipment.

Freedom environmental samples results will be evaluated using adaptations of Earth based standards. Because of the limited environmental reserves of atmospheric gases and water, however, responses and recovery from environmental contingencies will be limited. For example, current Station contingency response plans may include evacuation of a toxic module's atmosphere followed by repressurization. Even if this were 100% effective in eliminating the hazard, the repressurization would use a large percentage of available gases for a mission, and further repress/depresses might not be possible. Operational modeling of EHS and ECLSS systems will provide a better understanding of optimal sensor location, number, functional requirements, and other important data in the future.

Exercise Countermeasures Facility

Long duration space flight has well documented deconditioning effects on the cardiovascular, musculoskeletal, and neurosensory systems (5). To counteract these effects, the Exercise Countermeasures Facility (ECF) of SS Freedom will provide exercise equipment, physiologic monitors, and other facilities in one double rack located in a node. Present plans call for approximately two hours of exercise per crewmember for six days/week, with a capability for two crewmembers to exercise simultaneously. Motivational aids and ground monitoring "coaching" will be provided to assist the crew and optimize the exercise regimen for crewmembers. The exercise equipment will utilize three primary devices: a treadmill, a multi-functional exercise device, and a resistive exercise device, along with portable exercise devices. These devices will provide loads necessary for maintenance of aerobic fitness, strength, and skeletal integrity. The performance of the crewmembers will be monitored via video links and physiologic parameters including pulse, blood pressure, ECG, and metabolic gas monitoring.

The three CHeCS areas of HMF, EHS, and ECF must fit within the planned operational environment of Space Station to allow function with minimal demand on the scarce station resources. In fact, the CHeCS equipment has been downsized many times as the Freedom program struggled to meet constraints imposed by Congress and the available budget. The largest "scrub" occurred in 1990, when CHeCS went from a seven double rack design to the three rack system just described. Numerous developmental systems that will eventually be necessary for isolated long duration space flight were delayed or canceled. The regular use of CHeCS equipment as we learn about man's responses to space will require a significant portion of the available power, crew time, and attention. Each of the systems will have many interfaces with a variety of Freedom distributed systems including the data management system, environmental control, power, thermal systems, and the fluid management systems. Each system will also require ground support from the Space Station Control Center on a continual basis. At present, plans call for a 24 hour ground monitor of CHeCS systems and performance. In the event of a contingency or special event, additional expertise will be available via a ground telemedical network.

The CHeCS facilities on Space Station Freedom will play a significant role in the advancement of medical care in space flight. The provision of health care, environmental monitoring, and exercise countermeasures will be essential operational needs in this new environment. For future missions such as manned exploration of the moon and Mars, it will be necessary to build upon these capabilities and go on to develop more complete answers to the many potentially limiting problems facing manned exploration of the cosmos. CHeCS is a first step in providing comprehensive biomedical care and monitoring for a long duration crew. This endeavor will continue to provide numerous benefits for Earth based science by the constant demand for improved equipment, methods, and understanding of the biomedical issues involved.

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HMF SUBSYSTEMS

-- prevention, diagnosis, treatment and transport

1. **Anesthesia** – Peripheral Nerve Stimulator

2. **Dental** - Dental Hand Drill, Dental Instrument Tray, Laminar Flow/Suction Particle Containment Device

3. **Fluid Therapy** - Sterile Water for Injection System (SWIS), IV Solution Reconstitution Device, Large and Small Volume Parenteral Bags, Fluid Administration Kit, IV Catheters, Powered and Non-powered Infusion pumps, Accessories Kit, Blood Collection and Administration Kit, Parenteral Nutrition Kit

4. **Hyperbaric Therapy** - Built in Breathing Units

5. **Imaging** - Diagnostic Radiographic Imaging System (DRIS), Macroscopic Imaging System, Microscopic Imaging System

6. **Medical Analytical Lab** - Clinical Chemistry Analyzer, Blood Gas Analyzer, Hematology Analyzer, Coagulation Analyzer, Reagent Supplies Module, Sample Acquisition and Processing Module, Centrifuge, Prep Tent, (Microscope, Incubator, Slide stainer, Microbial Analysis system)

7. **Medical Decision Support** - Medical Database, Diagnostic Support System, Medical Library Reference System, Support Hardware, Medical Communication System, Medical Mobile Computer

8. **Medical Life Support** - Advanced Life Support Pack, Defibrillator, Doppler Flow Probe, Patient Monitor (EKG), Pneumatic Anti-Shock Garment, Pulse Oximeter

9. **Pharmacy & Central Supply** - Pharmacy, Central Supply

10. **Physician's Instruments** - Non-powered hand-held diagnostic instruments, Powered hand-held diagnostic instruments, Electronic Stethoscope System

11. **Respiratory Support** - Airway management equipment, Automated Ventilator equipment, Portable oxygen supply, Pulmonary manual resuscitator, Respiratory Monitoring

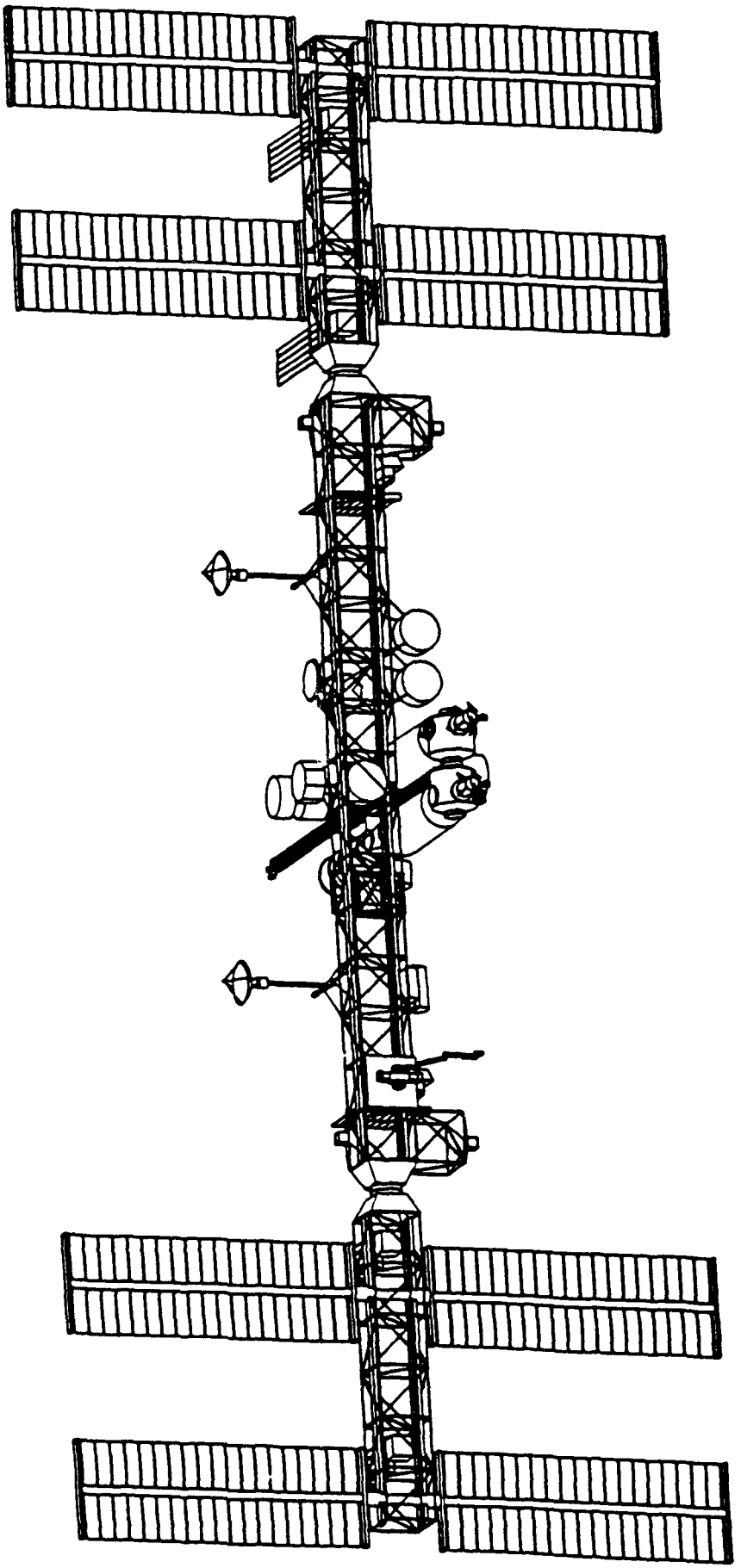
12. **Safe Haven** - Safe Haven equipment, Medical supplies, Pharmaceutical supplies

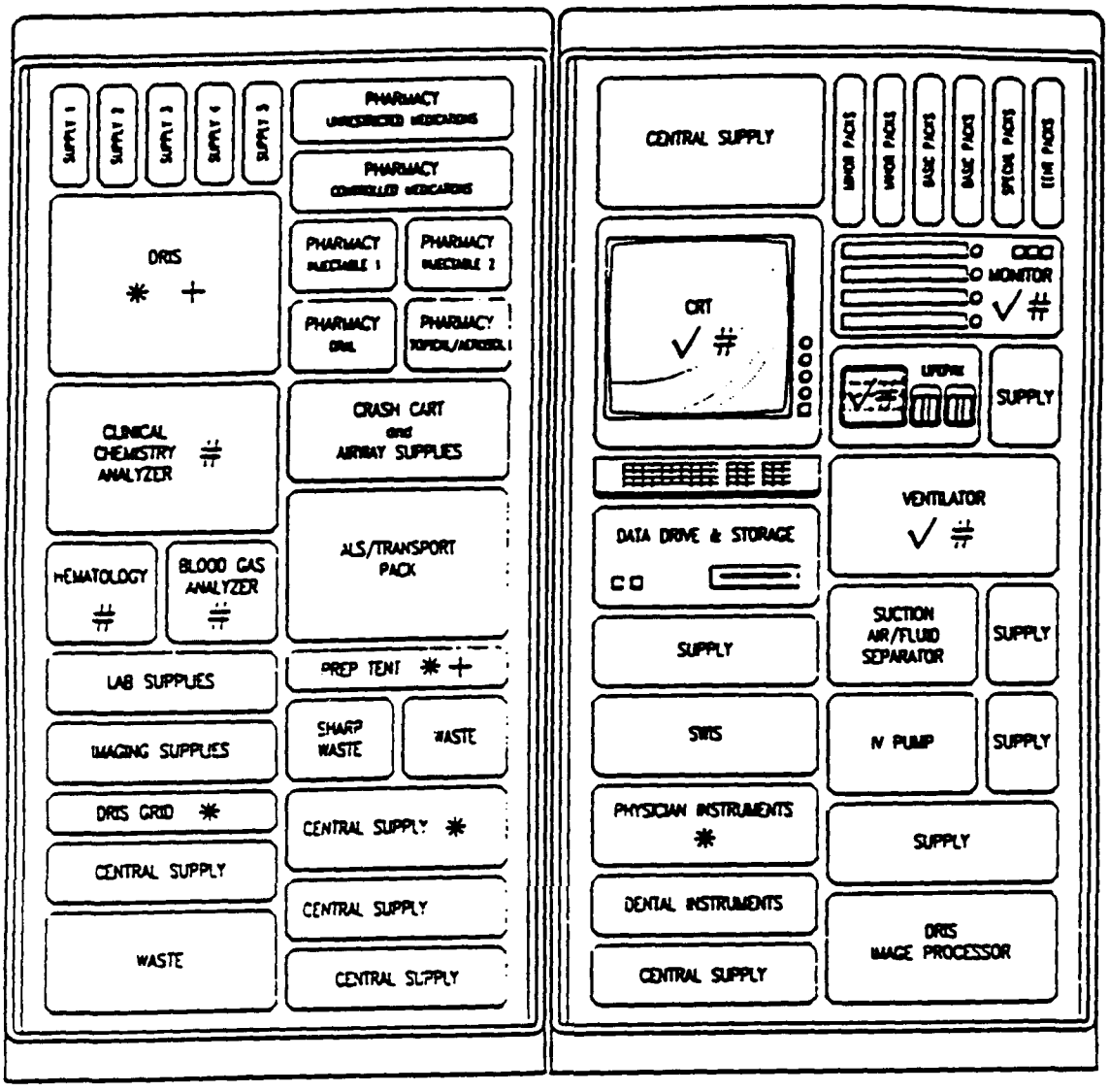
13. **Surgery** - Cautery device, Task Lighting, Medical Restraint System (MRS), Surgical Instruments and Supplies

14. **Transport** - Transport Monitor, Transport Aspirator

15. **Waste Management** - Air-Fluid Separator/Suction, Sharp Trash Container, Soft Trash Container, Fecal Collection Bag, Urine Collection system, Body Bag

TABLE 1





LEGEND

- ✱ PULLOUT
- ✓ LIGHTUP
- # KNOBS & BUTTONS
- \+ DEPLOY

FIGURE 2 - GENERAL LAYOUT AND FUNCTIONALITY OF PROPOSED HMF DISPLAY MOCKUP

RECENT ADVANCES IN HIGH ALTITUDE PULMONARY EDEMA (HAPE)

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Objectives

At the completion of the lecture on recent advances in HAPE, the participant will be able to:

1. Delineate the epidemiology and prevalence of HAPE.
2. Discuss the pathophysiology, clinical presentation and laboratory findings of HAPE.
3. Discuss the prophylaxis by natural means as well as by nifedipine.
4. Discuss the various options of treatment for HAPE which are descent, oxygen, nifedipine and the pressure bag.

PREVALENCE OF HIGH ALTITUDE PULMONARY EDEMA:

The reported prevalence of HAPE varies with altitude, rate of ascent, physical activity and altitude of residence. Skiers in the Alps very rarely, if ever, develop HAPE, since they generally stay overnight below 2000 m. In contrast one in 10'000 skiers in Colorado develops serious HAPE at altitudes of 2500 to 3000 m. Regiments of the Indian army flown above 3300 m had an attack rate of 15%. The prevalence of HAPE in trekkers in Nepal was reported to be 2.5 to 4.5% in the late seventies and has since decreased due to educational measures and awareness of the disease. The reported prevalence of 1 to 2% in climbers on Mount McKinley has also declined in recent years. In the Swiss Alps approximately 10 patients with HAPE per year have to be evacuated by air rescue organizations.

RISK FACTORS FOR THE DEVELOPMENT OF HAPE:

These include rapid ascent without prior acclimatization, continued ascent in spite of worsening symptoms of acute mountain sickness, a history of previous HAPE and male sex. In most studies males were 5 to 10 times more frequently affected by HAPE than females. The risk to develop HAPE upon very rapid ascent to 4550 m is 5 to 10 times higher in persons with a history of HAPE than in those with a good tolerance to high altitude in the past.

PATHOPHYSIOLOGICAL SEQUENCE OF HAPE:

1. Hypoxia:

   a) More pronounced hypoxemia in HAPE susceptible subjects due to relative alveolar hypoventilation (blunted hypoxic ventilatory response).

   b) Exercise-induced oxygen desaturation.

2. Sodium and Water Retention:

   HAPE-susceptible subjects are characterized by:

   a) More pronounced hypoxemia upon ascent to high altitude.

   b) Enhanced ACTH-secretion particularly during exercise.

   c) Enhanced secretion of norepinephrine, epinephrine and cortisol at rest and during exercise.

   d) Enhanced secretion of renin and angiotensin at rest and exercise.

   e) Enhanced secretion of aldosterone at rest and during exercise.

f) Enhanced secretion of ADH during exercise.

g) Enhanced secretion of ANP at rest.

As a consequence HAPE-susceptible subjects retain water and sodium, gain weight and develop peripheral and periorbital edemas.

3. Hypoxic Pulmonary Hypertension:

Marked hypoxic pulmonary hypertension develops in HAPE-susceptible subjects as a consequence of alveolar hypoxia. There seems to be exaggerated pulmonary vasoconstriction.

Uneven pulmonary vasoconstriction leads to over-perfusion injury of non-vasoconstricted vascular areas of the lung and thus to permeability edema. This could be either due to ruptur of endothelial structures or be solely a consequence of enhanced filtration pressure.

4. Leak:

The protein content of the edema fluid in HAPE is comparable to that of serum thus characterising HAPE as a permeability edema.

CLINICAL AND LABORATORY CHARACTERISTICS OF HAPE:

People developing HAPE initially, suffer from a non-productive cough, shortness of breath, particularly in the supine position, weakness, fatigue and other symptoms of acute mountain sickness. Lateron tachypnea develops. Subsequently the cough may become more productive with watery and sometimes bloody sputum. Rhonchi and gurgling sounds can eventually be heard without a stethoscope. However, the common clinical signs are crepitant rales, rhonchi, tachypnea, cyanosis and tachycardia. Later on, patients become irrational and hallucinating due to hypoxia and/or accompaning cerebral edema. This is rapidly followed by coma and death.

The systolic pulmonary artery pressure is markedly elevated in the range of 40 to 130 mm Hg. Arterial blood gas analysis shows marked hypoxemia and relative hypoventilation. The AaDO2 is elevated above 12 mm Hg. The radiomorphology is uncharacteristic with alveolar and interstitial edema. The right lower lung field is significantly more affected than other lung fields.

PREVENTION OF HAPE:

HAPE can in most cases be avoided by graded slow ascent, rest days when minor symptoms of acute mountain sickness develop and by descent, if symptoms of acute mountain sickness get worse. There is circumstantial evidence that prophylaxis with acetazolamide and/or dexamethason may protect HAPE-susceptible subjects to some extent, however, there have been no controlled studies with these medications.

In a recent study mountaineers with a history of radiographically documented HAPE were randomly allocated to receive 20 mg of a nifedipine slow relase preparation or identical placebo every 8 hours while ascending from low altitude within 22 hours to 4559 m and during the subsequent stay of 3 days at this altitude. Diagnosis of HAPE was based on chest radiographs. Pulmonary artery pressure was assessed by Doppler echocardiography and AaDO2 was measured in simultaneously sampled arterial blood and end-expiratory air. 7 of 11 subjects on placebo, but only one of 10 subjects on nifedipine developed HAPE (p-0.01). Systolic pulmonary artery pressure, AaDO2 and symptom score of acute mountain sickness were significantly lower in subjects receiving nifedipine than in those receiving placebo. Thus prophylactic administation of nifedipine is effective in lowering pulmonary artery pressure in preventing HAPE in susceptible subjects. This observation should not lead to uncontrolled intake of nifedipine for prophylaxis of HAPE. Intake of drugs in mountain sports can be considered acceptable, only when the most important preventive measure, which is slow ascent to high altitude, has failed.

TREATMENT OF HAPE:

HAPE is best managed by descent, evacuation and/or if impossible by oxygen in a flow rate of 1 to 4 liters per minute. In mild cases bed rest has been tested in a controlled setting. Furosemide has been advocated but in other hands it rendered patients susceptible to circulatory troubles, pulmonary embolism and cerebral edema. Morphin, another inadequately tested remedy, could do more harm than good by depressing the respiratory centres at a time when more respiration is needed. Positive end-exspiratory pressure has been applied briefly but it long-term effects may be harmful. Another possible means of short-term improvement is postural drainage.

Treatment of HAPE patients in a laboratory at 4559 m with nifedipine 10-20 mg sublingually, followed by 20 mg of a slow release preparation every 6 hours resulted in clinical improvement, better oxygenation, reduction of AaDO2 as well as pulmonary artery pressure and progressive clearing of alveolar edema. In these patients no other therapeutic measures were taken and they continued their mountaineering activities at altitudes above 4000 m. Thus nifedipine offers a potential emergency treatment for HAPE, when descent or evacuation is impossible and oxygen is not available.

EFFECTS OF ALTITUDE UPON CARDIOVASCULAR DISEASES

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Effects of Altitude Upon Cardiovascular Diseases

Learning Objectives:

1. Immediate and long term effects of high altitude upon the cardiovascular system.

2. Aggravation of angina pectoris by ascent to altitude including responsible mechanisms, prevention and treatment.

3. Increase in blood pressure in hypertensive patients going to high altitude. Responsible mechanisms, prevention and treatment.

4. Effect of high altitude upon pulmonary hypertension in patients with primary pulmonary hypertension, congenital heart disease and mitral stenosis: Methods of evaulation, prevention and treatment.

Effects of Altitude Upon Cardiovascular Diseases

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Physicians should be aware of sea level cardiovascular problems that may be aggravated by exposure to high altitude. Increasing numbers of middle aged and elderly individuals are visiting high altitude areas to ski, trek or attend conferences. Many Elder Hostel programs are held in mountain environments. A recent survey of 1909 adult visitors to Keystone, Colorado 9,300 feet (2837 m) revealed that 48% were 40 to 60 years of age and 15% were over 60. Sixty-six percent were males (1). Approximately 10% of trekkers in Nepal were 50 years of age or older (2). The prevalence of many cardiac conditions rises rapidly after 50 years. For example the mean age of entry of 2234 men into 3 large randomized studies of surgery in coronary artery disease was 50-51 years (3).

This presentation will review altitude induced changes that affect the circulatory system and examine 3 cardiovascular conditions that are adversely affected by altitude. These will include coronary artery disease, systemic hypertension and pulmonary hypertension. High altitude for the purpose of this presentation is defined as elevations exceeding 8,000 feet (2440 m), since altitude illness and altitude aggravation of sea level conditions are rare below this altitude.

1. Effect of High Altitude Upon The Circulation

Several important circulatory changes occur during exposure to high altitude (4,5). These include:

1. Increase in resting and exercise heart rate.
2. Increase in cardiac output and velocity of cardiac contraction
3. Increase in systemic vascular resistance and systemic blood pressure.
4. Contraction of veins and vessels in the skin, muscles and viscera with an increase in central blood volume.

The sum effect of these changes is an increase in cardiac work, cardiac oxygen consumption and an increase in coronary blood flow. These changes are largely due to an increase in sympathetic activity mediated by the effect of hypoxia upon the chemoreceptors in the carotid bodies (5; 6). Epinephrine secretion is transiently increased and Norepinephrine secretion is increased for several days (7).

Pulmonary ventilation is increased in proportion to the altitude resulting in a respiratory alkalosis which is partially corrected after several days. A prompt decrease in plasma volume occurs with ascent

resulting in a 10 - 20% decrease in total blood volume with a resulting rise in hemoglobin and hematocrit (4). After 2 - 3 weeks increased red cell production restores the total blood volume to normal pre-ascent values (8). Maximal physical working capacity as measured by the maximal oxygen consumption (V_{O_2} max) is decreased (9). The decrease is proportional to the altitude and amounts to a decrease of approximately 3% for every thousand feet of altitude above 5,000 feet.

A modest increase in pulmonary vascular (pre-capillary) resistance occurs with a resulting increase in pulmonary artery pressure. In some patients with an elevated pulmonary artery pressure at sea level a marked rise in pulmonary artery pressure may occur.

After 4 - 8 days at high altitude most circulatory changes subside and during a longer period of altitude exposure, several values will fall to levels below those seen at sea level. For example resting and exercise cardiac output are decreased (10). Left ventricular and atrial chamber size decrease without changes in cardiac function (11,12). The systemic blood pressure decreases and after a prolonged stay at altitude the blood pressure may fall to below sea level values (13,14). These effects are due to a decrease in sympathetic nervous system activity probably in part due to a decrease in peripheral receptors (15). Pulmonary artery pressure remains elevated however.

2. Coronary Artery Disease

The increase in cardiac work associated with ascent to high altitude may result in an increase in severity of angina, the new onset of angina or unstable angina.

An example of angina made worse by exposure to high altitude is represented by the following case report: A 70 year old businessman had angina pectoris since a myocardial infarct 22 years previously. His angina had progressed slowly so that he experiences chest pain with minimal physical effort. By limiting his activity he is able to carry on a sedentary existence with only 1-2 episodes of chest pain a day. He occasionally had nocturnal angina. His medications include Nitroglycerine prn, aspirin 325 mg/day, Propanolol 40 mg t.i.d. and Diltiazem 60 mg t.i.d.. He has never had hypertension. He has had frequent treadmill tests but stops after 1-2 minutes, because of chest pain. He had not had coronary arteriography or coronary bypass surgery. Recently he and his wife travelled to Denver, Colorado, where they spent the night. No increase in symptoms was noted. The following day they drove to Vail, Colorado altitude 8200 ft.(2500 m). Upon crossing Loveland Pass, altitude 11,900 ft. (3630 m) the patient began to experience frequent episodes of angina and dyspnea relieved by nitroglycerine. His wife noted he was quite blue and clearly in distress. Upon arrival at Vail his anginal attacks continued, but he was able to sleep moderately well. The following day repeated episodes of chest pain and dyspnea caused them to return to Denver. Once over Loveland Pass the patient felt very much better and upon

return to sea level his original attacks returned to the pre-ascent frequency.

Comment:

With such severe angina and limitation of effort at sea level the patient should not have gone to Vail. When his angina became worse at Vail a physician should have been called. It is very possible that low flow oxygen might have substantially relieved his symptoms. It is of interest that he did not experience an increase in symptoms at Denver 5400 feet (1647 m).

An analysis of 21 coronary events occurring within 5 days of arrival at Keystone, Colorado has recently been completed by this author. Seventeen patients had severe angina, two experienced an acute infarction and there were two sudden deaths. Fifteen patients were males. The mean age of all patients was 58 years (range 40 to 74). An elevated blood pressure and heart rate was present in most cases. Symptoms subsided with bed-rest, medical therapy, oxygen and descent. One patient (RM) developed severe angina upon arrival at Keystone. The pain subsided upon descent to a lower altitude and became severe upon return to Keystone 5 days later. The electrocardiogram revealed T wave inversion in the precordial leads during chest pain. After medical treatment a normal coronary arteriogram was found and a diagnosis of altitude induced coronary spasm was made. Coronary spasm can be produced by sympathetic stimulation and alkalosis, both of which occur at high altitude (16).

The most likely mechanism of altitude aggravated angina is the increase in cardiac work primarily due to the increase in systolic blood pressure and heart rate resulting in an increase in the heart rate/systolic blood pressure product which is an estimate of cardiac work. Morgan and his associates studied nine men with angina by graded treadmill testing at 5248 ft. (1600 m) and upon arrival at 10,150 ft. (3100 m). Mean maximal oxygen uptake was reduced by 11%. Angina and/or ST segment depression occurred at the same heart rate/systolic pressure product but at lower work loads and a shorter duration of treadmill time (17). The effect of hypoxia was via the increase in cardiac work and not an impairment of myocardial function. If myocardial function had been impaired by hypoxia, angina would have occurred at a lower double product than at the lower altitude.

The prevalence of coronary disease among the skiing population at Keystone is unknown. An estimate of the prevalence however can be made from telemetry electrocardiograms obtained by Grover in 149 men during recreational skiing at altitudes above 10,150 ft. (3100 m). Tachycardia was significant and heart rates exceeded 80% of the predicted maximum in two-thirds of the subjects. Five men developed abnormal ST segment depression during or immediately after exercise. All five were older than 40 years. Thus the incidence of ST abnormal-

ities in this skiing population was 5.6%. This is similar to the incidence in asymptomatic men during submaximal exercise at low altitude. Grover concluded that mountain skiing does not appear to pose a greater coronary stress than does comparable exercise at low altitude in men without known heart disease. Only one of his subjects had coronary disease (18). The risks of trekking in patients with coronary disease has been reviewed by the author (19).

In summary ascent to 9300 ft. (2837 m) may precipitate coronary events within a few days of arrival. Accelerated angina or new onset angina are the most common presentation, but acute infarction or sudden death may occur. Systemic hypertension and a rapid heart rate are commonly present and may be causative factors. Acute coronary spasm may occur. Many patients had experienced chest pain or infarction prior to ascent. Physicians should caution such patients that symptoms may appear or become worse with rapid ascent to high altitude. The use of anti-anginal or anti-hypertensive medications should be employed in appropriate patients. Acclimatization for a few days at an intermediate altitude (6,000 ft. - 7,000 ft. (1830 m to 2135 m) may be helpful. Restricted activity for a few days after arrival may minimize symptoms. Bed rest, low flow oxygen or descent may be necessary if symptoms are severe. Asymptomatic patients without known heart disease may ski at altitude without a greater prevalence of myocardial ischemia than performing a similar level of moderate to heavy exercise at a lower elevation.

Systemic Hypertension

A. Normal Subjects

Normal subjects usually exhibit a modest rise in blood pressure during the first week or two after ascent to high altitude. Several studies have indicated an elevation of both systolic and diastolic pressures as well as an increase in heart rate during high altitude exposure. Systemic vascular resistance is increased (4). Kamat observed a rise in blood pressure in 31 of 32 subjects who ascended to an altitude between 11,500 ft. (3506 m) and 13,000 ft. (3906 m). Systolic pressure rose from 115 mm Hg to 125 mm Hg and diastolic pressure rose from 78 mm Hg to 93 mm Hg. In 7 young men exposed to 14,000 ft.(4300 m) for 21 days blood pressure rose from 124/71 mm to 145/88m (mean values). Systemic resistance increased by 65 percent (8).

Blood pressure has been shown to rise even at lower altitudes. Palatini and his workers recorded 24-hour blood pressures at sea level after arrival at 3,969 ft. (1210 m) in 12 normotensives and 12 patients with borderline or mild hypertension. Modest increases in daytime blood pressures were observed in both groups. Systolic pressure was increased by 6 mm Hg in both groups with an increase in diastolic pressure only in the hypertensives of 5.5 mm Hg. Individual variations were present with some individuals exhibiting increases of

17 to 16 mm Hg in systolic and diastolic pressures respectively. Heart rates were increased by 3 to 4 beats per minute in both groups. Plasma Norepinephrine and Epinephrine levels increased suggesting that the changes observed could be due to sympathetic stimulation (21).

Hypertensive Patients

Patients with hypertension at sea level may experience a further rise in blood pressure with ascent to high altitude. In some individuals a marked rise in pressure may occur. This is illustrated by the following case report: A 62 year old Caucasian woman who resides in Florida had moderate, asymptomatic hypertension for 5 years. Her blood pressure was controlled on Enalapril maleate 5 mg. daily. Her pressure on this regimen varied between 115/75 - 120/82 mm Hg. She had no complications from her hypertension. There was no history of stroke, angina or left ventricular failure. The electrocardiogram was normal. Her renal function was normal. She had no retinal abnormalities. She was not obese weighing 127 lbs. She had a ventricular demand pacemaker implanted 2 years previously for a "sick sinus syndrome". Upon arrival at Aspen, Colorado 8,000 feet (2440 m) she had a diffuse, moderately severe headache during the first 2 days after arrival. Her blood pressure rose to 180/110 mm Hg. She consulted a local internist who administered Nifedipine sublingually and started Nifedipine (slow release tablets) 20 mg daily. Clonidine 5 mg patches were started daily. On this regimen her blood pressure was controlled to 110/80 - 130/100 mm Hg and she no longer had a headache. She has had previous similar rises in blood pressure upon visiting Aspen. On one occasion her pressure rose to 200/140 mm Hg. She experiences only mild acute mountain sickness with each ascent.

The effect of altitude upon hypertension is illustrated by a record of twice daily blood pressures taken by a physician during a motor trip to the Rocky Mountains. The data are shown in Figure 1. Systolic and diastolic pressures rose with the altitude attained and decreased to subnormal levels after descent. There was no significant change in heart rate. The patient continued his usual medication consisting of Propanolol 40 mg. daily throughout the trip. At 9,300 ft. (2850 m) the morning blood pressures were higher than evening pressures. This is a reversal of the usual diurnal variation and could be related to a greater degree of hypoxia during sleep. Blood pressure elevations in hypertensive patients going to high altitude are not a trivial problem. Eight percent of adult visitors to Keystone, Colorado gave a history of prior hypertension and 26% of these individuals were taking medications for their hypertension (22).

Patients with hypertension who go to high altitude should consult their physician in advance so that appropriate preventive measures can be employed. Usually an increase in medication, a low salt diet and increased rest during the first few days of the altitude stay may suffice. If patients record their own blood pressure a physician

should be consulted if high pressures are encountered. Low flow oxygen and rest may be helpful. The appropriate anti-hypertensive medications for altitude aggravated hypertension has not been determined. It is possible that beta blockers may not be effective in controlling high altitude blood pressure elevations. Altitude hypertension may be primarily related to increased sympathic activity, and release of Epinephrine and Norepinephrine. Beta blockade lowers blood pressure primarily by reductions in cardiac output and plasma renin activity. Clonidine may be more effective in the management of altitude hypertension since it produces a diffuse inhibition of sympathetic neural outflow of the central nervous system (23). Prazosin hydrochloride may also be more effective than beta blockers since this preparation blocks alpha-adrenoceptors and the rise in blood pressure during altitude exposure probably principally involves alpha stimulation. Terazosin is a new alpha blocking agent which has a longer duration (half life-12 hours), than Prazosin. Twenty mg. once daily has a similar hypotensive effect to that of Prazosin. Syncope may occur, but can be obviated by omitting the night dose (24). Calcium channel blockers may also be effective. Nifedipine may be used if hypertension in severe and symptoms are present and rapid reduction in blood pressure is desired. It is evident that controlled trials of several forms of medication at high altitude are indicated to develop more specific recommendations.

Pulmonary Hypertension

Several congenital and acquired cardiac conditions are associated with an increase in pulmonary vascular (pre-capillary) resistance and an elevated pulmonary artery pressure. Most important of these conditons are: primary pulmonary hypertension, several forms of congenital heart disease and mitral stenosis. Many patients with these conditions will exhibit a rise in pulmonary artery pressure when exposed to high altitude and some may have substantial increases in pressure even at moderate altitudes. Not all patients however may exhibit unpleasant symptoms and many may tolerate altitude exposure with little adverse circulatory effects.

Primary Pulmonary Hypertension

Most patients with primary pulmonary hypertension will exhibit an increase in symptoms upon ascent to high or even moderate altitudes. Increased dyspnea, weakness on exertion and an increase in syncopal episodes may occur. Breathing a low oxygen mixture will result in a rise in pulmonary artery pressure in many of these patients. The pressure rise can now be non-invasively determined by echo-doppler studies (25). In many patients Diltiazem or other calcium channel blockers may prevent the rise in pressure (26). If patients with primary pulmonary hypertension must visit a higher altitude the use of pulmonary vasodilators, reduced physical activity after arrival and low flow oxygen may prevent or ameliorate symptoms. Patients who live at

moderate or high altitudes may experience considerable relief of symptoms by moving to sea level and by the use of selected pulmonary vasodilators as illustrated by the following case report (27). A 20 year old man who resided in Denver, Colorado 5,400 feet (1647 m) experienced increasing exertional dyspnea and frequent episodes of exertional near and true syncope. Hemodynamic studies had established the diagnosis of primary pulmonary hypertension. The patient moved to the San Francisco Bay Area upon the advice of his physician. After arrival his symptoms were greatly relieved and his walking distance greatly improved. Hemodynamic studies indicated a moderate reduction in pulmonary artery resistance and pressure. Intravenous and sublingual Isoproterenol further reduced pulmonary artery resistance and improved walking distance as evaluated by a treadmill test. The patient was followed for 6 years. Symptomatic benefit from 20 mg. of Isoproternol 6 to 12 times daily persisted, but slow progression of his disease and symptoms were noted (27,28).

Congenital Heart Disease

It has long been known that several varieties of congenital cardiac conditions may be associated with an increased pulmonary artery pressure due to an increased pulmonary vascular resistance. These conditions include ventricular septal defects, patent ductus arterious and atrial septal defects. Patients with secundum atrial septal defects born at moderate to high altitudes have a higher pulmonary artery pressure than patients born at lower elevations (29). Patients with ventricular septal defects born at Denver, Colorado have pulmonary vascular resistances twice as high as patients at sea level (30). Many of these patients will exhibit an increase in pressure with exposure to altitude.

While most of the above congenital cardiac conditions can be treated surgically some patients cannot be operated on because of severe pulmonary hypertension. Such patients may experience adverse effects at high altitude due to a rise in pulmonary artery pressure and an increase in cyanosis due to a greater right to left shunt. Right heart failure may occur. The effect of oxygen and pulmonary vasodilators upon pulmonary vascular resistance should be evaluated prior to ascent and, if effective, employed to minimize symptoms at altitude. A decrease in physical activity is mandatory.

Mitral Stenosis

Symptomatic mitral stenosis is now rarely seen in the United States due to the wide application of surgical treatment. However severe mitral stenosis is common especially in young patients in many other parts of the world, including India and Mexico. Of the various acquired valvular lesions mitral stenosis is the only lesion commonly associated with an increased pulmonary vascular resistance and pulmonary hypertension. In some patients systemic levels of pulmonary

artery pressure has been observed. Patients with mitral stenosis exposed to hypoxia commonly exhibit an increase in vascular resistance (31). Such patients may exhibit an increase in symptoms during high altitude exposure. Increased dyspnea may be due to the increase in heart rate with a resulting higher left atrial pressure. Right heart failure may occur due to the increased afterload. Atrial fibrillation may be precipitated. Precautions are similar to those in other patients with pulmonary hypertension. Restricted activity, oxygen administration and pulmonary vasodilators may relieve symptoms. If atrial fibrillation is present additional Digitalis or Verapamil may be necessary for control of the heart rate.

In summary ascent to high altitude may have significant effects upon certain varieties of cardiovascular disease. The most common complication is an increase in angina or new onset angina which occurs within a day or two after arrival. Systemic blood pressure may become elevated especially in individuals with pre-existing hypertension. These complications are largely related to increased activity of the sympathetic nervous system. Patients with pulmonary hypertension due to congenital or acquired heart disease may experience an increase in symptoms at altitude due to hypoxic pulmonary vasoconstriction and a rise in pulmonary artery pressure. Physicians should be aware of these potential effects of altitude so that appropriate preventive and therapeutic measures can be employed.

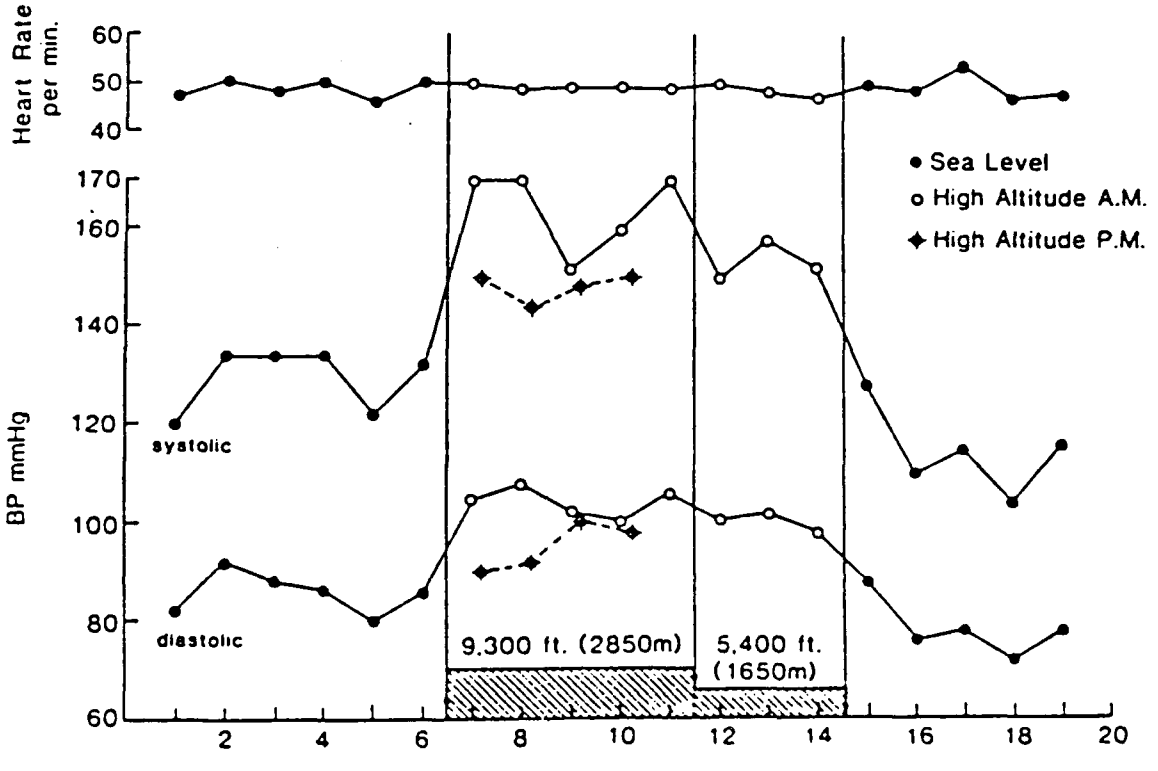


Figure I.

Heart rate, systolic and diastolic blood pressure in a 64 year old physician at sea level and during an auto tour of the Rocky Mountains. Note the rise in systolic and diastolic pressure related to altitude. At 9,300 feet morning pressures were higher than afternoon pressures. Propanolol 40 mg. daily was continued throughout the trip.

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WATER DISINFECTION PRODUCTS

Howard Backer, MD

Water Disinfection Products

Page 1A

Product/Manufacturer	Price	Structure/Function
Ketadyne U.S.A., Inc. 3020 N Scottsdale Rd Scottsdale, AZ 85251 (602) 990-3131		0.2 micron ceramic filter; silver impregnated to decrease bacterial growth.
Ketadyne pocket filter Replacement filter element	$185 $90	40" intake hose & strainer, zipper case; size: 10"x2"; wgt: 1 lb 9 oz; hand pump, flow: 3/4 qt/min.
Handpump filter Replacement filter element	$600 $40	Steel stand and large hand pump; size (packed in case): 23x6x8"; weight: 10lbs 13oz; flow: 1 1/2-3 qt/min.
Drip filter (same filter element as handpump)	$300	Gravity drip from one plastic container to another through 3 filter elements; size: 18" high x 11" dia. (26" high when assembled); weight: 8lbs 5oz; flow: 1 pt/hr.
General Ecology, Inc. 151 Sheree Blvd. Lionville, Pa. 19353 (215) 363-0412		0.4 micron (absolute) filter--carbon matrix, silver impregnated; cannister removable:
First-Need purifier extra cannister pre-filter replacement	$39.95 $24.95 $6.50	hand pump with intake strainer; 800 pint capacity; size: 6"x6", wgt: 12 oz. flow: 1 pint/min;
Seagull IV (X-1P) replacement cartridge	$320 $50	Stainless steel casing and hand pump; capacity 1000 gal; size 4.8x5.4"; weight: 3 lbs; flow: 3-4 qts/min
Timberline Filter PO Box 12007 Boulder, CO, 80303		2 micron fiberglass and polyethylene matrix;
Timberline filter replacement element	$24 $12	hand pump; flow: 1 qt in 1.5 min; wgt: 6 oz; size: 9"x1-3".
Outback Filter (Distributed by Hiker Supply of Washington)	$29.95	1 qt water bottle with drink-through filter element from timberline filter.

Claims	Comments
Removes bacterial pathogens, protozoan cysts, parasites, nuclear debris. Clarifies cloudy water. If filter clogs, flow can be restored by brushing filter element--this can be done hundreds of times before needing to replace filter element. Claims for removal of viruses not made in USA, although testimonials offered implying effective in all polluted waters.	Effective for claims. Expensive, but durable. Complete virus removal cannot be expected, although it is true that viruses usually clump to larger particles that can be filtered. Silver impregnation probably does not prevent all bacterial growth in filter; recommend flushing with bleach solution after each trip.
0.1 micron retention (0.4 absolute) microstrains bacteria, cysts, parasites; no specific claims for viruses. "Ionic charges from surface chemistry...remove particles even smaller than microfiltration alone can remove." Carbon adsorbers remove chemicals and organic pollutants that cause color and taste. Does not remove all dissolved minerals and salts.	Reasonable design, cost, and effectiveness. Most testing with E. coli and Giardia cysts show complete removal. Charcoal matrix will remove chemical pollutants. Will clarify cloudy water. Despite testimonials for effectiveness in undeveloped countries, recommend caution where viruses may be a problem; prior disinfection with halogen would guarantee disinfection, and carbon would remove halogen.
Removes Giardia cysts. No claims for bacteria or viruses.	Effective for claims; intended only for domestic backcountry use where Giardia is the primary contaminant. Lightest pump filter available. Drink-through filters likely to give frustratingly slow flow and require high pressures as particles begin to clog.

Water Disinfection Products

Page 2A

Product/Manufacturer	Price	Structure/Function
Water One (Available in camping stores.) Calco Ltd 7011 Barry Ave Rosemont Illinois 60018 (312) 296-6615	$49.95	Cartridge containing charcoal block, 0.5 micron filter; prefilter sponge 30-50 micron. Bulb hand-pump generates 3-5 lbs pressure; can back-wash to unclog. Size: 3"x6" cartridge with about 7 ft of tubing and bulb pump; Wgt: 1 lb. Capacity 400 gallons or 3 yrs.
Water Works Total Filtration System Dromedary Beverage Bag (All filter elements and parts replaceable.) Mountain Safety Research Box 3978 Terminal Station Seattle, WA 98124 (206) 624-7948	$139.95 $12.95-19.95	Four filter elements of decreasing pore size: porous foam, 10 micron stainless steel wire mesh screen, activated carbon filter, then 0.1 micron absolute membrane filter. Hand pump with inlet tubing. Storage bag (2 or 4 liter) attaches directly to outlet of pump. Size: 9x4x3"; Wgt 18.4 oz; Flow rate: 1 liter/90 sec.
Sierra Water Purifier 2801 Rodeo Rd Suite B-518 Santa Fe, NM 87505 (505) 986-0617		Chlorine crystals (calcium hypochlorite) and 30% hydrogen peroxide in separate small plastic bottles with dropper and scoop. Uses extremely high concentrations of chlorine for disinfection, then dechlorination with peroxide, which causes formation of soluble calcium chloride (non-toxic). Excess peroxide bubbles off as oxygen.
Sierra Water Purifier starter kit (hikers) marine and RV kit Refills available (reduced price)	$12.95 $13.00	Total weight: 5 oz; treats 160 gallons treats 720 gallons

Manufacturer and Claims	Comments
Removes Giardia; no claims for bacteria, viruses. Unbreakable, 2 year guarantee. Comes with dye to test filter integrity.	Should be effective for claims made. Charcoal will remove most chemical pollutants and halogens--so filter could be used as second stage after halogen disinfection of water which may contain bacteria and viruses.
Removes protozoa (including Giardia), bacteria, pesticides, herbicides, chlorine, discoloration. Design and ease of use are distinct advantages. Filter can be easily maintained in the field; maintainance kit and all replacement parts available.	Excellent filter design. Prefilters protect more expensive inner, fine pore filters. Should be effective for claims made. No claims made for viruses. Many would be removed by clumping and adherence to larger particles, but this should not be considered reliable for highly polluted waters in developing countries. Attaching bag is a nice addition.
Kills all microorganisms. Voluminous experimental evidence exists for efficiency of chlorination, especially with use of high concentrations. Hydrogen peroxide is also a weak disinfectant. Treated water has no chlorine taste; in fact, taste is improved from oxygenation.	Sound use of chlorination and dechlorination. Minor disadvantage of two-step process. Peroxide is titrated to estimated amount of chlorine; measurements do not need to be exact, but takes some experience to balance the two and achieve optimal results. 30% peroxide is extremely corrosive, and burns skin; use cautiously. Very good technique for highly polluted or cloudy waters and for disinfecting large quantities. By far the best technique for storing water on boats: maintaining high levels of chlorination prevents growth of algae or bacteria during storage, then dechlorinate when ready to use.

Product/Manufacturer	Price	Structure/Function
Water Technologies Corp 14405 21st Ave N, Suite 120 Plymouth, MN 55447 (800) 627-0044		Pentacide™ iodine resin; iodine molecules complexed to quaternary ammonia base and attached to inert resin. Water is poured or pumped through resin, contacting iodine.
Personal Traveler cup	$19.99	gravity pour through: 6oz/45sec; capacity 100 gallons; size: 3"x4" wgt: 3.5 oz.
Puri-jug	$29.95	Collapsible 2 gallon container water passes through iodine resin at outpour spout. 20-30 oz/ min; collapses to 8.5x8.5x7"; 1 lb.
The straw		Drink-through straw; 8" cartridge with prefilter, pentacide resin and granular activated carbon filter. capacity: 25 gal; 5.5" long; wgt: 1 oz.
Micro-Pure hand pump replacement cartridge *(EPA registration not yet submitted. Sold outside USA. Contact company for this product and for those listed below.)*	$130 $90	Hand or foot operated bulb pump; Pentacide/carbon cartridge and sediment filter; capacity 500 gallons; flow: 1.5 qts/min; wgt: 1 lb 12 oz; size: 7x2" cartridge, 50" long.
Mt Everest expedition model replacement cartridge	 $250 $110	Hand pump; Pentacide/carbon cartridge; flow: 1 gal/min; size: 15x9x5"; weight: 6.5lbs Capacity 500 Gal or 6 mos.
Penta-Pour Bucket replacement cartridge	$200 $100	Gravity drip bucket with 3 gal holding capacity; sediment filter and pentacide/carbon cartridge; capacity: 2,000 gallons; 10 Gal/hr; 12"x30", wgt: 2.5 kg.
Travel-Pure faucet filter		Sediment filter, Pentacide resin and carbon bed adapter to easily attach to any faucet without tools. Capacity 200 Gal or 12 months; flow rate 0.5 Gal/min; length: 6";

Claims	Comments
Resin releases iodine "on demand", on contact with microorganisms; minimal iodine dissolves in water: effluent 1.0-2.0 ppm iodine. Charcoal removes any residual dissolved iodine. Tested effective for bacteria, giardia, shistosomiasis, viruses--including hepatitis.	Test data is convincing; resin appears to be major breakthrough in water disinfection. Organisms effectively exposed to extremely high iodine concentrations when passing through resin, which is why minimal holding time is necessary. Iodine apparently binds to microorganisms aided by electrostatic forces and penetrates more readily than in a dilute iodine solution, but the exact mechanism of iodine transfer to organisms is not known. The company has developed a large number of new products applicable to different size groups and various settings. The resin disinfectant has already been approved by the EPA, however each product must be individually submitted and tested for approval--a long and arduous process. While all these products are already produced and sold in Europe, most are not yet available in the U.S. However, most are available through the company; contact the sales department. The most rational products are the hand pump models, the Puri-jug and pour bucket, and the faucet filter. The travel cup is much too slow and gives only small quantities. The straw is for survivalists.

Water Disinfection Products

Product	Price	Structure/Function
Polar Equipment 12881 Foothill Lane Saratoga, Ca. 95070		Iodine crystals, 8 gm in 3 oz bottle; separate 30-50 micron prefilter provided. "Trap" in bottle to catch crystals when pouring off water; bottle cap is used to measure--directions and dot thermometer on bottle (temperature affects iodine concentration in bottle). Recommends warming water to 20C (68F) before adding iodine (to shorten contact time). Capacity: 2000 quarts; Wgt: 5 oz.
Polar Pure (Widely available in camping stores.)	$9.75	
Polar Pure Plus (new product)		2 oz plastic dropper bottle of concentrated iodine/alcohol solution; thermometer and graph for determining dosage and contact time.
Potable Aqua (sold widely in camping stores)	$3.25	Iodine-containing tablets (tetraglycine hydroperiodide) release approximately 7-8 mg iodine when added to water. Instructions direct one table added to one quart of water. In cloudy or cold water, add two tablets. Contact time only 10 minutes in clear, warm water, more in cold, cloudy water. Refer to table. Weight: 2 oz.
Emergency Germicidal Drinking Water Tablets	$3.99	
Basic Designs 5815 Bennet Valley Rd Santa Rosa, CA 95404 (707) 575-1220		Candle Filter in a bag; ceramic element and charcoal core. Gravity drip or squeeze bag. Size: 3.5" x 8"; Wgt: 8 oz.
Ceramic Water Filter	$65	

Claims	Comments
Destroys all waterborne pathogens; yields 4 ppm iodine when appropriate dose is added to one quart of clean water.	Extensive data exists for effectiveness of iodine. Commercial source of crystalline iodine, an excellent source of iodine solution. Recommendations adequate for clear, warm water, but it may be impractical to warm water. Reports of glass bottles freezing (and breaking) in subzero temperatures. Polar Pure Plus uses disinfection chemistry to determine the minimal dose necessary for chosen contact time. This allows control over dose (and taste), but requires some intelligence in the user.
Kills bacteria, viruses, and protozoan cysts, including giardia.	Method developed by the military for troops in the field. Good data for effectiveness of iodine at these concentrations. Advantages of unit dose and short contact time, but these concentrations create strong taste that would not be acceptable to many wilderness users. May add one tablet to two quarts of clear water to yield about 4 mg/L with a minimum contact time of 20 minutes in warm water and at least one hour in cold water. In cloudy water, use one or two tablets per quart (better yet, clarify water first).
Removes Giardia, bacteria, parasites, cysts and bad taste.	New product--not all technical data received prior to writing this. Should be effective for claims; note virus removal is not claimed. Filtration rate may be slow.

Joseph B. Serra, M.D.
WMS First World Congress
Whistler, B.C. 1991

MANAGEMENT OF DISLOCATIONS & FRACTURES IN THE WILDERNESS ENVIRONMENT

GENERAL COMMENTS

Trauma to the musculoskeletal system, in the form of dislocations and fractures, is on the increase due simply to the fact that there are more people involved in the wilderness experience of remote travel, climbing, cross-country skiing, trekking, and major expeditions. The fractures and dislocations are the same as those seen in the urban and hospital setting; however, there are distinct differences in methods of diagnosis and acute management. It is a well known fact that the sooner a dislocation is reduced, or the sooner a fracture is realigned and splinted, the more comfortable and stable the victim will be. This presentation is directed at physicians, nurses, paramedical personnel, trek leaders, and anyone else who is involved in remote travel where transport to a medical facility is not available, for whatever reason. These may include distance, environmental risk factors or extreme terrain. In the situation where acute management and early transport is feasible, this should be the treatment of choice. Fractures should be realigned as well as possible and splinted prior to transport; the area of question involves dislocations. I would advocate reduction of the more common dislocations as soon after injury as possible, if the rescuer is comfortable with attempting a reduction and if the victim is agreeable. Identification of the injury as a dislocation or fracture; good clinical judgment regarding management; a good common sense approach to the problem are all important factors. These will be discussed and demonstrated.

DISLOCATIONS

Dislocations of most joints may be easily identified and are quite incapacitating to the victim. Some of the major advantages in early reduction of dislocations are as follows:

1. Reduction is easier immediately after injury, before swelling and muscular spasm have developed.
2. Reduction most often results in dramatic relief of pain.
3. Transport of the victim is easier after reduction.
4. Immobilization of the injured joint is much easier and stable after reduction.
5. Safety of the entire party may be jeopardized during the evacuation of a victim with a major joint dislocation.
6. Early reduction reduces the circulatory and neurologic risks to the involved extremity.

DIAGNOSIS

The major joints discussed are the shoulder, elbow, digits, hip, knee, patella, and ankle. There are many helpful signs in identifying a dislocation. There is nearly always restriction of motion; obvious deformity in comparison with the uninvolved extremity; often a typical, identifiable posture of the dislocated joint that the victim will maintain to minimize pain. Crepitus or grating of bone fragments; and gross instability are generally absent.

The provider of care or rescuer should not be concerned about causing additional damage to any fractures associated with a dislocation. At times, there are avulsion type fractures within the joint that has dislocated, but these fractures will generally be improved in alignment with reduction of the dislocation. The same is true of vessel and nerve impairment associated with a dislocation; reduction will reduce the impingement and traction injury to these structures, as well. In the situation in which there is a major long bone fracture associated with a dislocation, the dislocation may not even be diagnosed, in view of the more apparent major fracture. In these cases, splinting of the fracture is the treatment of choice and the dislocation, for all practical purposes, becomes a secondary issue and will, most likely, not be identified until x-rays are obtained.

DIAGNOSTIC TIPS FOR SPECIFIC JOINTS

SHOULDER - Anterior-inferior dislocation of the shoulder is most common, accounting for 95% of shoulder dislocations. Victim will stabilize the upper extremity in the most comfortable position; usually with the upper arm held away from the body and supported by the uninvolved arm. The dislocated extremity cannot be brought across the chest wall, i.e. sling position. Observe and palpate the involved shoulder, comparing it with the uninvolved side. There will be a loss of normal contour to the deltoid and a palpable defect where the humeral head should be. Check circulation, motor and sensory function to the hand and, also, sensory function along the outer aspect of the shoulder, and document findings. Mechanism of injury most commonly external rotation, abduction and extension. In the case of recurrent anterior dislocations, the victim will identify the problem and can be quite helpful in its reduction.

ELBOW - Obvious deformity is present when compared with uninvolved side. Restricted, painful motion is present. Most commonly posterior with resultant bony prominence of the olecranon process.

DIGITS - Obvious deformity at the joint with limited motion are main findings.

HIP - The majority of dislocations are posterior. The involved extremity will be moderately flexed, internally rotated and adducted. Any attempt to extend the extremity for splinting or easier transport will be resisted by the victim and mechanically impossible to obtain. The mechanism of injury is most often a fall in which the hip is flexed and the forces are transmitted longitudinally through the knee and femur, driving the femoral head posteriorly from the acetabulum - a dashboard injury is a good example. Anterior dislocations of the hip are less common and usually occur as the result of a fall directly on the lateral side of the hip, driving the femoral head anterior and medially. The posture is extension or mild flexion with external rotation and abduction of the thigh. Again, it is nearly impossible to bring the hip into full extension and neutral position.

PATELLA - Most often occur laterally with the knee held in moderate flexion for comfort. Position of patella in comparison to uninvolved side is diagnostic. May be recurrent with usual mechanism pivoting on a partially flexed, weight bearing knee.

KNEE - Truly a disaster with the probability of major ligamentous disruption. Vascular impairment is a major threat. Peripheral pulses, motor and sensory function at the foot and ankle should be evaluated and documented. The knee may not be dislocated at the time of initial examination, but gross instability is the major clue.

ANKLE - Vascular impairment to the foot is a major risk. Associated fractures are common. Prompt reduction or the best improvement of alignment should be performed immediately.

METHODS OF REDUCTION

SHOULDER - Two methods will be discussed: The abduction traction method, and the prone traction method. The first is steady traction applied to the shoulder with the upper arm abducted 90°. The victim is supine and, preferably, at waist level. Continual communication with the victim regarding the procedure and the need for relaxation is vital. Gently bring the upper arm 90° away from the side of the body; have an assistant apply countertraction in the axilla; while you gently apply traction using your body weight as the traction force. Create a loop, utilizing a jacket or shirt with the arms tied together, avalanche cord, belt or webbed strapping. With the victim's elbow flexed 90°, the loop is placed in the antecubital fossa

and around the waist of the reducer. Then, leaning back steadily, adequate traction is applied to the shoulder. Padding in the antecubital fossa and axilla prevents damage to neurovascular structures. Gentle, steady leaning backward using your body weight creates the traction to reduce the shoulder. Continue traction for two to three minutes, while gently internally and externally rotating the humerus, all the while communicating with the victim to attain maximum relaxation. Reduction is usually signalled by a clunking of the humeral head into the glenoid and noticeable relief of the victim. Proof of reduction is the ability to bring the arm across the chest wall. The extremity is immobilized with a sling and swathe.

The prone traction method consists of the victim in a prone position with the arm hanging downward and a 15-20 pound weight secured to the wrist. Relaxation is extremely important. This may require 15-20 minutes and, at times, is not feasible in rainy, cold, windy weather.

In first dislocation, immobilization is recommended for three weeks to allow adequate soft tissue healing. In recurrent dislocations, the victim will usually decide when to start using the shoulder again. Individual circumstances will dictate the need for evacuation of the victim.

ELBOW - Traction applied to the forearm with the elbow in a partially flexed position while countertraction is applied to the upper arm by an assistant. Slow, steady pull with medial or lateral pressure, if appropriate. Reduction is successful if the elbow can be flexed to 90°. Elbows can be quite difficult to reduce; give it your best try, but don't be disappointed if not successful. If not reduced, splint in a position most comfortable to the victim. Check peripheral pulses.

DIGITS - Reduction of phalanges is accomplished by traction applied to a partially flexed digit while actually pushing the base of the dislocated phalanx back into place. This is more successful than attempting to apply straight traction to the digit. After reduction and if the victim desires, buddy tape the reduced digit so that partial function can be maintained. Don't attempt reduction of a dislocated metacarpophalangeal joint of the index finger, since open reduction of this dislocation is necessary. Dislocation of the metacarpophalangeal joint of the thumb is often quite difficult to reduce, but should be attempted. In open dislocations, thoroughly cleanse the wound and proceed with reduction; leave the wound open; and apply a sterile dressing. Antibiotic therapy, if available, is indicated.

HIP - Although reducing a dislocated hip may be difficult and painful to the victim, it is worth doing, if at all

possible. The victim will be more comfortable after reduction, transport is facilitated, and impairment of circulation to the femoral head is reduced. Demerol, Morphine or Valium given intravenously is quite helpful, if available. Two people are required with one applying countertraction to the pelvis as the other reduces the dislocation. The victim is placed supine on the ground; the involved hip and knee are gently and slowly flexed to 90°. The reducer straddles the victim and applies steady traction in an upward direction. Victim's lower leg is placed between rescuer's thighs; hands locked behind the knee; and as the rescuer assumes a sitting position while applying traction upward, significant leverage is applied to the hip. This position also allows for internal and external rotation of the hip, as needed. The anterior dislocation is reduced by steady, gentle, longitudinal traction with internal and external rotation. Once reduced, the injured extremity must be splinted to the uninvolved extremity and the victim transported in a supine position.

PATELLA - Everyone should learn to do this reduction because it is so easy; provides immediate relief; and converts the victim to an ambulatory state. The knee will be partially flexed with the patella laterally displaced. Gently extend the knee and gently push the patella back towards its normal position. Splint the knee in extension from ankle to groin. Various materials are appropriate, including Ensolite pad wrapped around the leg, down parka wrapped in circular fashion several times around the knee, metal pack stays, or tree branches. It is safe for the victim to walk and far better than attempting to carry the victim. An ice axe or ski pole is a helpful crutch.

KNEE - Apply gentle traction to realign the joint as well as possible. Generally, this is easily done due to the massive ligamentous disruption. The advantages are reduction of vascular impairment. Splint the entire extremity; check pulses and document findings. The victim must be carried.

ANKLE - Dislocations of the ankle are almost always associated with fractures. The fractures and dislocated joint both benefit by reduction of the deformity as soon as possible. Circulation may be compromised and peripheral pulses should be evaluated.

Apply gentle traction to the foot and ankle, using the victim's leg as the countertraction, and this will significantly improve the alignment. Anatomical alignment is not necessary. By improving alignment, splinting is more stable and comfortable. These fracture-dislocations may be well splinted with a down parka or other comparable gear,

having the affect of a pillow splint and being securely wrapped or pinned in place.

FRACTURES

GENERAL COMMENTS

Diagnostic skills are most important in the suspected fracture and management should be dictated by the circumstances at hand. The needs of the victim must be considered in relation to his/her desire to ambulate on a suspicious ankle injury; the ability and availability of people to carry; the type of terrain involved in transport; and the need or desire of the victim to continue carrying a load. Fracture deformities should be reduced as well as possible and adequate splinting applied. Rest, elevation, ice and compression are part of basic fracture care. Improvisation is often required for splinting techniques and there are usually many materials available to be used. These include skis, ski poles, Ensolite pads, metal pack frames, straps, webbing, rope, tree branches, parkas, and maps, to mention a few.

UPPER EXTREMITY FRACTURES

SHOULDER GIRDLE - Gently palpate towards the area of pain; compare with uninvolved side; look for deformity. Sling is adequate immobilization. Ice, snow, cold compresses are helpful to reduce pain and swelling.

HUMERUS - The shaft of the humerus is palpable throughout its entire length along the inner aspect of the upper arm. Any area of point tenderness with significant trauma should be considered a fracture until checked radiologically. Apply sling and swathe. If there is obvious deformity in the humeral shaft, allow gravity to realign the fracture and apply appropriate splinting. Check radial nerve function by asking the victim to extend wrist, digits and thumb, and document findings.

ELBOW - May be grossly unstable. Apply appropriate splint, posterior and/or U-shaped, with elbow approximately 80-90°, and then sling.

FOREARM - Realign as well as possible and splint joint above and below; U-shaped splint is best. Check peripheral pulses. Sling, elevate and ice, if available.

WRIST - These are best splinted in the position found, unless there is a compromise of circulation. Place the

hand in a position of function with soft material, such as rolled up glove or socks, in the palm.

HAND OR PHALANX - Realign and splint in position of function with all joints in a partially flexed, relaxed position. Example would be holding a rolled Ace bandage in the palm of the hand. Digits should not be splinted in an extended position due to rapid onset of joint stiffness. Consider buddy taping, whenever feasible, to allow for partial usage of digits.

LOWER EXTREMITY FRACTURES

PELVIS - Pelvic fractures are suspected when there has been significant trauma to the pelvic region and tenderness to palpation over bony prominences. Crepitus is usually not present. Pain with weight bearing may also suggest an acetabular fracture. Suspected pelvic fractures require transport, unless pain and tenderness are mild over pubis or ischium, in which case, this may be a small, stable fracture and the victim may desire to ambulate with the aid of ice axe or ski pole. The general rule would be to transport victim with a suspected pelvic fracture. If the trauma is massive, be aware of the possibility of shock due to retroperitoneal bleeding.

HIP - Symptoms are pain, inability to bear weight. Fractures of the femoral neck may not reveal any deformity or instability, but rather pain with hip motion. Unstable hip fractures often reveal shortening and external rotation of the extremity. Immobilization of the injured extremity to the uninjured with padding between the thighs and under the knees is usually sufficient. If there is significant pain with transport, then traction may be helpful.

FEMORAL SHAFT - All femoral shaft fractures require traction to provide stability, reduce hemorrhage, prevent further vascular damage, and reduce pain. Temporary traction should be applied as soon as the injury is diagnosed. There are excellent lightweight traction devices which are available and should be strongly considered as standard equipment for expeditions. The Kendrick and Sager splints are very good; lightweight, easy to apply, and very efficient. Become familiar with their usage to facilitate their usage, if the need should arise. If makeshift traction is to be used, become familiar with the materials you plan to use and practice applications prior to their field application. It is difficult to remember the necessary steps. A rule of thumb for the amount of traction is 10% of body weight; but an equally good indicator is when the victim feels comfortable and the fracture feels more stable. Flex both knees 5-10° with padding beneath the knees.

This will not affect the traction and makes transport much more comfortable. Monitoring peripheral pulses and sensibility should be done every thirty minutes and documented.

An extended transport over very difficult terrain requires a minimum of eight to ten people to alternate in carrying the victim and also to carry packs. Therefore, if there is reasonable alternative, including helicopter evacuation, it should be strongly considered. Keep the victim warm and monitor for shock during all transport or while awaiting rescue.

PATELLA - Patellar fractures result from direct trauma to the patella. If there is tenderness, but no palpable defect or deformity, the knee can be wrapped in a cylinder splint, composed of an Ensolite pad or foam mattress pad, and the victim be allowed to ambulate, if possible. Ice axes or ski poles are helpful.

TIBIA - These fractures are often easily diagnosed due to deformity, crepitus and immediate swelling. Gently correct angular deformities and apply adequate splinting. An U-shaped splint running from the inner knee beneath the foot as a stirrup and up the outside of the leg to the knee with adequate wrapping is an excellent tibial splint. In an open fracture, the area should be gently cleansed with Betadine or any other antiseptic available or with soap and water; a sterile bandage or the cleanest available material placed over the wound and the fracture then treated with realignment and splinting. Broad spectrum antibiotic therapy is indicated, if available. The victim should not bear weight with a suspected tibial fracture.

FIBULA - In suspected fibular fractures, the victim may be allowed to ambulate once the fracture is adequately taped, wrapped or splinted. Walking aids are helpful.

ANKLE - Swelling and tenderness about the ankle may denote a fracture if directly over bony prominences. They are sometimes difficult to differentiate from ankle sprains. As a general rule, the fracture tenderness immediately after injury is over bone and not ligament. Therefore, treat all ankle injuries as if there may be a fracture involved. The ankle should be securely taped or wrapped and an U-shaped, stirrup splint applied. Weight bearing should be at the discretion of the victim, if the pain is not severe. A victim barely able to place weight on an injured ankle, should be advised not to do so.

TARSALS & PHALANGES - These are often stable fractures and, with adequate wrapping, weight bearing may be allowable, as tolerated. Walking aids are most helpful.

hand in a position of function with soft material, such as rolled up glove or socks, in the palm.

HAND OR PHALANX - Realign and splint in position of function with all joints in a partially flexed, relaxed position. Example would be holding a rolled Ace bandage in the palm of the hand. Digits should not be splinted in an extended position due to rapid onset of joint stiffness. Consider buddy taping, whenever feasible, to allow for partial usage of digits.

LOWER EXTREMITY FRACTURES

PELVIS - Pelvic fractures are suspected when there has been significant trauma to the pelvic region and tenderness to palpation over bony prominences. Crepitus is usually not present. Pain with weight bearing may also suggest an acetabular fracture. Suspected pelvic fractures require transport, unless pain and tenderness are mild over pubis or ischium, in which case, this may be a small, stable fracture and the victim may desire to ambulate with the aid of ice axe or ski pole. The general rule would be to transport victim with a suspected pelvic fracture. If the trauma is massive, be aware of the possibility of shock due to retroperitoneal bleeding.

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FEMORAL SHAFT - All femoral shaft fractures require traction to provide stability, reduce hemorrhage, prevent further vascular damage, and reduce pain. Temporary traction should be applied as soon as the injury is diagnosed. There are excellent lightweight traction devices which are available and should be strongly considered as standard equipment for expeditions. The Kendrick and Sager splints are very good; lightweight, easy to apply, and very efficient. Become familiar with their usage to facilitate their usage, if the need should arise. If makeshift traction is to be used, become familiar with the materials you plan to use and practice applications prior to their field application. It is difficult to remember the necessary steps. A rule of thumb for the amount of traction is 10% of body weight; but an equally good indicator is when the victim feels comfortable and the fracture feels more stable. Flex both knees 5-10° with padding beneath the knees.

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TARSALS & PHALANGES - These are often stable fractures and, with adequate wrapping, weight bearing may be allowable, as tolerated. Walking aids are most helpful.

SPLINT MATERIALS

In addition to improvised splinting materials, there are commercial products available which provide excellent immobilization and are lightweight. The Sam splint is an excellent addition to any wilderness pack. For more extended travel and major expeditions, air splints should be considered. The zippered variety with screw type air tube is most efficient. Keep in mind the variations in pressure, due to gain or loss in altitude, and monitor circulation on an hourly basis. They do provide excellent immobilization and stabilization of many fractures. The adult long leg and adult long arm sizes are adequate.

The commercially available air stirrup splint for ankle injuries is excellent and lightweight. This splint will often allow the victim to ambulate in relative comfort and stability.

All splints add weight to your medical pack but, if needed, their efficiency to provide adequate splinting and comfort far outweigh the disadvantages.

SUMMARY

Fractures and dislocations do occur in the wilderness environment. An understanding of the injury and quick, efficient handling of the problem will often convert a very painful, unstable and difficult injury into one which can be managed more comfortably for the victim and all concerned.

HIGH ANGLE RESCUE: RECENT DEVELOPMENTS

Michael V. Callahan, MSPH
Ministrie des Montagnas-Alpine Guard
National Association for Search and Rescue
Cambridge, Massachusetts, U.S.A.

OBJECTIVES

Following this workshop participants will be able to:

1. Identify factors surrounding alpine accidents through the analysis of recovered mountaineering equipement

2. Describe mountain rescue operations for several agencies in Europe and North America.

3. Describe the role of prototype medical and rescue gear developed for mountain rescue.



PRACTICAL EVACUATION SKILLS

Lanny Johnson, RN, EMT-P

Moving an injured or incapacitated mountaineer over rough terrain can be a difficult and strenuous task.

Participants will do hands-on practice with improvised carrys and formal litter carry on non-technical terrain.

Objectives

1. Learn improvised carrying techniques.
2. Be able to estimate manpower needed during evacuations involving carry outs.
3. Learn techniques for carrying and belaying litters safely on non-technical terrain.

ABSTRACT FORM
FIRST WORLD CONGRESS ON WILDERNESS MEDICINE
July 14-20, 1991
Whistler, British Columbia

TITLE: The Availability of Oxygen-saving Systems for Himalayan Climbing.

BODY OF ABSTRACT: The abstract must be typed double-spaced in no less than 10-point type size and no more than 12 characters per inch. It must be limited to the space on this page. Do not include references, illustrations, or funding sources. Tables, when appropriate, are acceptable. Do not list authors or institutions on this page.

[Introduction] Although O_2-less high altitude climbing is prevailing among elite climbers, Himalayan trecking has became more popular among non-career mountaineers. For the laters, O_2-less climbing is not recomendable. But oxygen is very expensive. So, availablity for Himalayan use of several clinical O_2-saving systems were tested at the Kyoto University Medical Research Expedition to Xixabangma 1990.
[Materials and Methods] Used apparatuses were; 1)Pendant O_2-conserving nasal cannula(Pendant), 2) Thermo-sensitive demand O_2 delivery system(Synchoxy), 3)Baro-sensitive demand O_2 delivery system (Sansosaver), 4)Vinyl face mask with constant O_2 flow as the control. They were tested at rest at Base Camp(BC)(5020m). The sabjects were 8 climbers. It was evaluated by elevation of arterial O_2 saturation measured by puls-oxymeter (SpO_2). And also they were tested on exercise at Advanced Base Camp(ABC)(5640m). The subject was only one and it was evaluated by walking speed on foot on the hill.
[Results] Average SpO_2 elevation by 0.5l/min.of O_2 inhalation for 10 minutes at rest at BC were as follows; Pendant:11.7%,Synchoxy:11.8%, Sansosaver:12.3% and face mask:3.3%. The walking speed were measured by consuming time on foot on the hill behind ABC with the elevation of about 40m.The results obtained were as follows; Air:5 min.22 sec. Pendant:4 min.28 sec.,Synchoxy:4 min.38sec.,Sansosaver:3 min.55sec..
[Discussion]Several clinical reports suggested that these apparatuses could save the O_2 consumption to about from 1/2 to 1/3 with considerable elevation of SpO_2.Our results supports their observations.
[Conclusion] It was suggested that the Pendant nasal cannula and the demand oxygen delivery systems, Synchoxy and Sansosaver, could be used effectively and relatively economicaly for Himalayan climbing.

ABSTRACT FORM
FIRST WORLD CONGRESS ON WILDERNESS MEDICINE
July 14-20, 1991
Whistler, British Columbia

TITLE: Carbohydrate Supplementation for Work at High Altitude: Liquid Versus Solid Food Supplements

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The anorexia associated with rapid ascent to high terrestrial elevations is well known to climbers and trekkers. Even with a gradual ascent and acclimation, appetite does not keep pace with energy demands associated with work in these high altitude environments. Carbohydrate is believed to be an especially critical energy source for work at high altitude. We studied energy intakes and expenditures of soldiers consuming liquid or solid carbohydrate food supplements while undergoing military training at several high altitude locations (Mauna Kea, HI; Pikes Peak, CO; Mt. Rainier, WA; and Potosi, Bolivia) ranging from 3000-4300 m elevation. Our results indicate that thirst is less negatively affected than appetite by high altitude. Consequently, sweetened beverages containing sucrose and/or glucose polymers were readily consumed and enabled the soldiers to ingest up to 200 g/man/day additional carbohydrate over that level consumed from the basal ration alone. However, when a solid rather than a liquid carbohydrate supplement was used, the soldiers ate less of the basal diet and did not receive significantly more total daily carbohydrate. The results of these studies strongly suggest that high altitude field operations are best supported by a liquid carbohydrate supplement.

ABSTRACT FORM
FIRST WORLD CONGRESS ON WILDERNESS MEDICINE
July 14-20, 1991
Whistler, British Columbia

TITLE: CENTRAL NERVOUS SYSTEM CHANGES THAT MAY PRECEDE SYMPTOMS OF ACUTE MOUNTAIN SICKNESS (AMS)

BODY OF ABSTRACT: The abstract must be typed double-spaced in no less than 10-point type size and no more than 12 characters per inch. It must be limited to the space on this page. Do not include references, illustrations, or funding sources. Tables, when appropriate, are acceptable. Do not list authors or institutions on this page.

A previous report has demonstrated that latencies of visual evoked potentials increase at high altitudes (Neurosurg. 21: 352, 1987). Furthermore, it was suggested that more prolonged latencies may occur in patients suffering from AMS. We sought to 1) determine if neural responses would also be altered at high altitudes in subjects taking acetazolamide (500 mg OD) and dexamethasone (4mg BID) or acetazolamide alone (500 mg OD) and 2) establish whether a relationship exists between AMS and changes in visual evoked potentials.

Visual evoked potentials and symptom reports were recorded for twenty-two subjects participating in a climbing expedition in Bolivia, S.A. Data were collected at sea level (day 0), 3,689 m (day 2) and 4,970 m (day 5). The second negative (N2) and positive wave (P2) brain responses to flashed-light stimuli were recorded over the occipital cortex. Latencies for the overall group were studied for changes upon ascent (ANOVA). Furthermore, subjects taking acetazolamide and dexamethasone were compared to those on acetazolamide alone (double-blind, randomized) for latency changes from sea level to 3,689 m and 4,970 m (T-test). Cumulative cerebral symptom scores (sums of intensity grading scores for symptoms present) at each altitude were compared between the two drug groups (Wilcoxon signed-rank test).

The overall group did not demonstrate a significant difference in waveform latencies upon ascent. However, those subjects taking acetazolamide alone exhibited 1) a significantly greater N2 latency change (eight percent) than the combined drug regimen at 3,689 m 2) and subsequently developed higher ($p=.02$) mean cumulative symptom scores at 4,980 m. No significant differences between the drug groups at 4,980 m existed for either the absolute latencies or for latency changes from sea level.

These data suggest that changes in evoked potential latencies in the acetazolamide only group at 3,689 m may be a subclinical sign of the developing pathophysiology but the latency impairment may be resolved when AMS symptoms are present.

ABSTRACT FORM
FIRST WORLD CONGRESS ON WILDERNESS MEDICINE
July 14-20, 1991
Whistler, British Columbia

TITLE: Effect of Cooling Time on Survival in Heatstroke: A Meta-Analysis Evaluation

BODY OF ABSTRACT: The abstract must be typed double-spaced in no less than 10-point type size and no more than 12 characters per inch. It must be limited to the space on this page. Do not include references, illustrations, or funding sources. Tables, when appropriate, are acceptable. Do not list authors or institutions on this page.

Although rapid cooling is considered a cornerstone of the treatment of heatstroke, there is little data to correlate rapid cooling with improved outcome. Two previous studies using similar methodologies demonstrated decreased mortality with rapid cooling but were unable to demonstrate statistical significance, probably secondary to small sample sizes. We performed a similar analysis on 31 patients with classical heatstroke and again demonstrated a much lower mortality (11%) in rapid coolers vs delayed coolers (33%) but were also unable to achieve statistical significance (P=.10). Because of the consistent pattern of improved survival with rapid cooling, meta-analysis of the 3 data sets was undertaken to explore whether a real difference in outcome existed between rapid and delayed cooling groups. Meta-analysis demonstrated that heatstroke patients that cool rapidly do have improved survival. Meta-analysis has not been commonly used to assess clinical studies of environmental emergencies, and may play an important role in clarifying treatment approaches to these infrequently encountered problems.

THE EFFECT OF AN ELECTRICAL CURRENT ON SNAKE VENOM TOXICITY

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[1]Division of Emergency Medicine, Department of Surgery, Section of Surgical Sciences, Vanderbilt University, Nashville,TN, USA.

[2]Department of Veterinary Science and [3]Department of Pharmacology and Toxicology, University of Arizona, Tucson, AZ, USA.

ABSTRACT

An electric current (20, 11 amp, 7000 volt spikes per second for 90 seconds) from a commercial stun gun was applied directly to a rattlesnake venom solution in a three compartment electrolysis cell, in order to evaluate the effect that high voltage electroshock might have on the lethality of the venom. Using LD_{50} determinations in mice, there was no measurable inactivation of the venom when it was electroshocked for 18 times longer than recommended by stun gun manufacturers. Using a constant voltage power supply, a venom sample was electrolyzed at a lower voltage, but 4 to 5 times the charge delivered from the stun gun. This inactivated the venom at the electrodes, but not within a central compartment, which simulated the condition of a human envenomation.

Key words: snake venom, envenomation, stun gun, electrical current

ABSTRACT.

Success of a high-altitude expedition may be limited by the nutritional status of the expedition members. This study describes a dietary survey carried out by members of The Everest Base Camp Clean Up Operation. These individuals were not mountaineers but the walk-in period completed by the volunteers is similar to that of most Himalayan expeditions. Ten healthy unacclimatised male subjects walked from an altitude of 2430 m to Everest Base Camp (5400 m) in 10 d. The average increase in altitude was 300 m/d. All food and fluids consumed during the 10 d walk-in period were weighed using digital dietary scales and recorded in food record books. The diet during this time was based on fresh, locally available foods such as potatoes, onions, cabbage, buffalo, goat and yak meat, dahl, barlotti beans and flour. Energy and nutrient intakes were calculated using food composition tables. The mean daily energy intake was 10.03 (SE 1.26) MJ. Once an altitude of 3475 m had been reached there was a significant decrease in percentage energy from fat (36 (SE 2)% on day 3 to 27 (SE 1) on day 10) with a corresponding increase in energy from carbohydrate (46 (SE 4) on day 3 to 61 (SE 1) on day 10, $p<0.001$). Mean daily intakes of vitamins and minerals are given in the Table.

	Mean	SE	RDA
Thiamin (mg)	1.3	0.2	1.3
Riboflavin (mg)	1.7	0.3	1.6
Niacin (mg)	37	6	18
Folic acid (ug)	147	15	300
Ascorbic acid (mg)	37	1	30
Iron (mg)	13	1	10
Zinc (mg)	9	1	-

(RDA, recommended daily amount, DHSS, 1979).

The RDA values refer to healthy populations but not to those exposed to high-altitude when nutrient requirements may be increased. The results indicate that if an expedition diet is based on locally available foods for at least 10 d, a vitamin and mineral supplement may be beneficial in order to prevent possible deficiencies.

ABSTRACT FORM
FIRST WORLD CONGRESS ON WILDERNESS MEDICINE
July 14-20, 1991
Whistler, British Columbia

TITLE:

A Modified Hyperbaric Bag and Inhalator Mask to Study Gas Exchange

BODY OF ABSTRACT: The abstract must be typed double-spaced in no less than 10-point type size and no more than 12 characters per inch. It must be limited to the space on this page. Do not include references, illustrations, or funding sources. Tables, when appropriate, are acceptable. Do not list authors or institutions on this page.

A pilot study of hyperbaria was performed utilizing a Gamow hyperbaric bag (HB) modified with a penetrating plug during the 1990 American Andes Biomedical Research Expedition (AABRE) climb to 4,970 m in Bolivia, South America.

Six subjects (4 at sea level without medication and 4 at high altitude taking medication for acute mountain sickness (AMS) prophylaxis) underwent three consecutive twenty minute exposures in the modified HB. During the first exposure subjects breathed compressed ambient air whilst in the portable chamber. Then each subject breathed 100% oxygen (0_2) on demand or compressed air through a modified Scott II inhalator mask using a nonrebreathing system. $Sa0_2$, $Tc0_2$, $TcC0_2$ and chamber $C0_2$ were measured. Gas analyses were done with both polarographic and chemical methods.

$Sa0_2$, $Tc0_2$, and $TcC0_2$ increased 14%, 50%, and 75% respectively, at the end of 20 minutes in subjects breathing compressed air in the HB at 4,970 m (bag pressure was maintained at 110 torr above ambient barometric pressure). P_1C0_2 increased .85 mm Hg in the bag when measures to prevent $C0_2$ rebreathing were not taken. $Sa0_2$ and $Tc0_2$ increased 26% and 700% respectively, when 100% 0_2 was breathed in the HB at high altitude.

Further studies are necessary to determine whether combining hyperbaria with 100% 0_2 delivered on demand through a mask is more effective in increasing $Sa0_2$ and ameliorating AMS symptoms than either therapy alone.

Orthopedic Field Splinting Devices

This presentation will demonstrate new field splinting devices for upper limb and neck injuries and for lower limb injuries. The splint for upper limb and neck injuries is made out of plastic and exceeds the capabilities of other available products. Performance highlights include: the splint does not become brittle at low temperatures, it is completely reusable and does not weaken with multiple use, it is simple to use, it is light weight, it rolls into a small compact cylinder shape, and it is economical.

The device for lower limb injuries is improvised out of available materials and therefore has no additional costs or additional weight. The splint is put together from internal stays from four expedition packs, a therma-rest sleeping pad or similar inflatable pad, webbing or cord, and wind or rain pants.

ABSTRACT FORM
FIRST WORLD CONGRESS ON WILDERNESS MEDICINE
July 14-20, 1991
Whistler, British Columbia

TITLE:

Oxygen Saturation and Neuropsychological Performance Changes With Altitude

BODY OF ABSTRACT: The abstract must be typed double-spaced in no less than 10-point type size and no more than 12 characters per inch. It must be limited to the space on this page. Do not include references, illustrations, or funding sources. Tables, when appropriate, are acceptable. Do not list authors or institutions on this page.

Exposure to hypoxic environments, as at altitude, may acutely cause decreased neuropsychological functioning. We questioned whether any deficit in performance can be easily measured at different altitudes during a climb. A pilot group of four recreational mountaineers participated in a climb to 5700 meters. Pulse, O_2-saturation, Pretrails, Trails A, Trails B, and Symbol Digit Modalities were evaluated at four different altitudes. These are well-standardized tests with established norms and are easily administered at altitude. Because this was a pilot study with a small number of subjects, only trends were identified. As expected, pulse rate increased and oxygen saturation decreased with altitude. Trails A seemed to be a sensitive test in that it did show deterioration with altitude. This effect was not due to exhaustion or cold because Pretrail speed remained constant at all altitudes. Trails B did not show this pattern, but there was a single outlier that may have skewed the results. Symbol digit modalities did not improve until return to 2240 m; this may indicate that the anticipated improvement in scores with repeated testing (learning effect) did not occur and may reflect an altitude effect. Altitude-induced neuropsychological dysfunction may be evaluated with this battery of simple tests. Larger numbers of subjects need to be studied for validation of these trends.

ABSTRACT FORM
FIRST WORLD CONGRESS ON WILDERNESS MEDICINE
July 14-20, 1991
Whistler, British Columbia

TITLE: Pseudoephedrine For The Prevention of Barotitis Media: A Controlled Clinical Trial in 120 Scuba Divers

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Purpose: To determine the efficacy and safety of decongestant prophylaxis among scuba divers in the prevention of barotitis media (middle ear squeeze).

Methods: A prospective, double-blind clinical trial was completed with 120 volunteer scuba divers under the supervision of certified instructors. Following randomization, each subject received a 60mg tablet of pseudoephedrine or placebo, 60 minutes prior to diving. Prospective data were collected, including subject demographics, signs and symptoms of middle ear squeeze during the dive, and possible drug side effects. The otoscopic appearance of the tympanic membrane was graded according to the amount of hemorrhage in the eardrum, with TEED scores running from 0 (normal) to 5 (gross hemorrhage and rupture). χ^2, t test, and the Mann-Whitney U test were applied with significance at $P<.05$.

Results: A total of 116 subjects met the inclusion criteria and completed the study; 60 received 60mg pseudoephedrine and 56 received placebo. Treatment groups were similar with regard to age, sex, medical history and depth of the first dive ($P>0.5$). Ear discomfort during the dive was present in 8% (5/60) of those receiving pseudoephedrine vs 32% (18/56) of the control group ($P=.001$). Likewise, the pseudoephedrine group tended to have smaller TEED scores after diving than the control subjects ($P=.003$). No adverse effects were noted.

Conclusions: These results suggest that use of an oral decongestant prior to diving is safe and decreases the incidence and severity of middle ear squeeze.

ABSTRACT FORM
FIRST WORLD CONGRESS ON WILDERNESS MEDICINE
July 14-20, 1991
Whistler, British Columbia

TITLE:

Radio frequency rewarming of hypothermia victims

BODY OF ABSTRACT: The abstract must be typed double-spaced in no less than 10-point type size and no more than 12 characters per inch. It must be limited to the space on this page. Do not include references, illustrations, or funding sources. Tables, when appropriate, are acceptable. Do not list authors or institution on this page.

The efficiency of warming after hypothermia depends on the method of rewarming and the control of temperature afterdrop. Rewarming in field conditions is often less efficient because of logistical or equipment limitations. To improve both efficiency and field applicability, rewarming with a radio-frequency (RF) coil operating at 13.56 MHz has been evaluated. In one study, anesthetized rhesus monkeys were used to compare the temperature effects and enzyme response to anesthesia only, rewarming using a surgical heating pad circulating 35°C and 40°C water, or RF rewarming from rectal temperatures (T_{re}) of 28° to 30°C. Rewarming to 35°C typically required only 50 minutes for RF vs 137 min for heating-pad rewarming with no significant differences in serum CPK, LDH or AST between the methods. Six additional sessions, performed at lower T_{re}, were not significantly different, except for one which resulted in cardiovascular collapse at T_{re} = 19.3°C, with spontaneous cardioversion upon RF rewarming. RF rewarming produced T_{re} that were significantly higher than heating pad rewarming beginning with the third minute of rewarming, and included a large reduction in the magnitude and duration of temperature afterdrop. RF has also been shown to produce more rapid rewarming to 30°C than warm humidified air (43°C, 450 cc $min^{-1} \cdot kg^{-1}$) (58 ± 13 vs 280 ± 114 min respectively) with anesthetized dogs at stable core temperatures of 25°C. Likewise, in mildly hypothermic humans with 0.5°C reduction in esophageal temperatures (T_{es}), RF has been shown to produce more rapid rewarming than either an insulated cocoon or rewarming in 41°C water (ΔT_{es} 1.15 ± 0.22°C/h, 0.18 ± 0.09°C/h, and 0.37 ± 0.16°C/h respectively). RF has been shown to be an efficient, field applicable method of rewarming from hypothermia and a vest based on this patented method has been approved by the FDA as an investigational device for human use.

ABSTRACT FORM
FIRST WORLD CONGRESS ON WILDERNESS MEDICINE
July 14-20, 1991
Whistler, British Columbia

TITLE: Respiratory function test(RFT) in normal subjects during a 7 days period at high altitude(HA) and correlation with AMS score.

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AIM of the study is the monitoring of flow-volume curves(FV) during a 7 days stay at HA and the correlation with AMS score METHODS:5 subjects(2F,3M)mean age 36±4 performed 3 FV at different altitude:sea level(sl),3600m in 3 days(A1,A2,A3),4550m in 4 days both in the morning and in the afternoon(B1m B1a,B2m B2a,B3m B3a,B4m B4a).A1 and B1 were 'climbing days' and the values were not used for the analysis.The best according to FEV1 was selected for the statistical analysis(paired t test and ANOVA).RESULTS(mean%of expected value):PEF sl 102% A 120% B 138%;FEV1 sl 96%,A3 B2a90%,B3a 96%;FVC sl 104%,A3 B2a 100%, B3a 108%;MEF25 sl 76%,A3 B2a 70%,B3a 79%;MEF25 B2m 64% B2a 70 B3m 72% B3a 79%. Statistical analysis:significant increase in PEF($p<.01$)between sl and HA;significant decrease($p<.01$)in FVC FEV1,MEF25 between sl and B2a.In B3a and B4a the values improve;the analysis between B2m-B2a and B3m-B3a showed a significant reduction($p<.01$)in MEF25 in the morning vs afternoon.In B2 B3 a even light AMS score was recorded.CONCLUSIONS the HA higher PEF is due to decreased air density;the decreased FEV1, FVC,MEF25 in A3 and B2 could be due to the higher lung tissue fluid(as pointed out by AMS score).The monitoring of MEF25 twice a day can detect a higher lung tissue fluid in the morning even when afternoon RFT are improved(defective acclimatitation)

A REVIEW OF EMS NEAR DROWNING CALLS IN CONTIGUOUS NORTH CAROLINA BEACH COMMUNITIES: APPROPRIATE USE OF BASIC ASSESSMENT AND MANAGEMENT SKILLS MUST BE BASED ON A THOROUGH UNDERSTANDING OF POTENTIAL INJURIES SUSTAINED IN THE MARINE ENVIRONMENT

Near drownings are a subset of environmental emergencies frequently seen in beach communities. This study retrospectively reviewed all EMS calls for the calendar year 1990 in the rural setting of Dare County, North Carolina. Several management problems were identified and protocols will be modified to positively affect outcome. Prehospital care was provided at the EMT-Advanced Intermediate (endotracheal intubation, intravenous access and basic ACLS drugs) level. Charts reviewed identified 109 EMS calls related to near drownings. Of these, 78 (71.6%) were male and 55 (50.5%) were 30 years or older, while only 5 (4.6%) were less than age 6. Eighteen (16.5%) victims were found in cardiopulmonary arrest, and 2 (1.8%) were considered dead. Surf and tidal rip were identified as significant factors in near drownings in 16 patients (14.7%), which is previously unreported. Spinal immobilization, a significant concern, was documented in only 11 (10.1%) patients, and for only 2 (1.8%) of the patients injured in the surf. Endotracheal intubation was accomplished in only 8 (44.4%) arrest victims. Hypothermia was documented in 19 (17%), with 12 (11%) occurring in June, July and August. These data suggest that specific education on near drowning and the water environment should be incorporated into EMS training for providers in water-related areas. Airway management and spinal immobilization skills must be stressed in relation to these injuries.

ABSTRACT

TREKKER MEDICINE:
The Impact of Western Medicine on the Health of Sherpas in the Khumbu Region of Nepal

Catherine A. Hagen, M.D.
Ian D. Schokking, M.D, C.C.F.P.

Each year, approximately 10,000 foreign trekkers visit Sagarmatha National Park region of Nepal (the Mount Everest Trek). Problems of health among trekkers have been documented: high altitude sickness, trauma, bacterial and protozoal infection. Less attention has been paid to the impact foreigners have had upon the health of the local 3,000 Sherpas.

Many visitors are doctors; all like to share the contents of well-stocked first aid kits with local residents. Unfortunately, the habit of trailside prescribing can undermine a system of primary health care established through Kunde Hospital and a system of village health clinics. Better communication could be fostered between visiting health professionals and Sherpa patients, in the interest of supporting WHO goals of primary health care through essential drugs and locally trained workers.

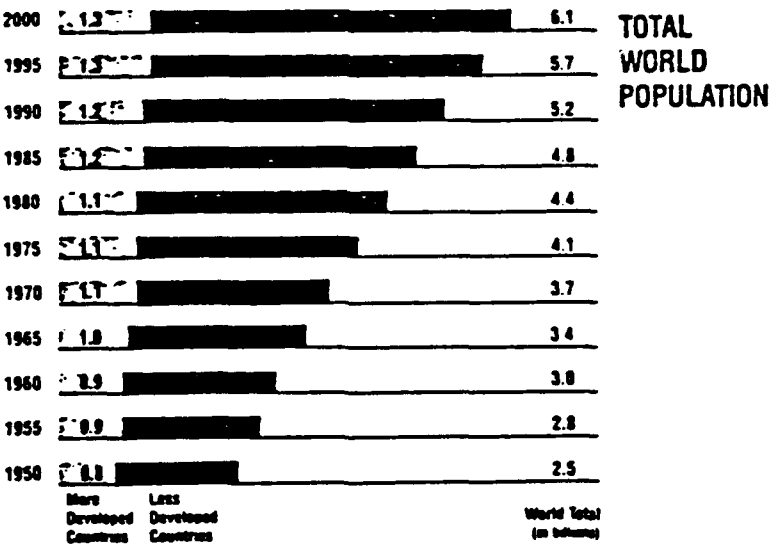
In a slide presentation, Doctors Hagen and Schokking document two years of primary health care practice and education among the Sherpas of the Mt. Everest region.

GLOBAL HEALTH ISSUES

Philip C. Rasori, M.D.

"Modern education is competitive, nationalistic and separative. It has trained the child to regard material values as of major importance, to believe that his nation is also of major importance and superior to other nations and peoples. The general level of world information is high but usually biased, influenced by national prejudices, serving to make us citizens of our nation but not of the world."

ALBERT EINSTEIN

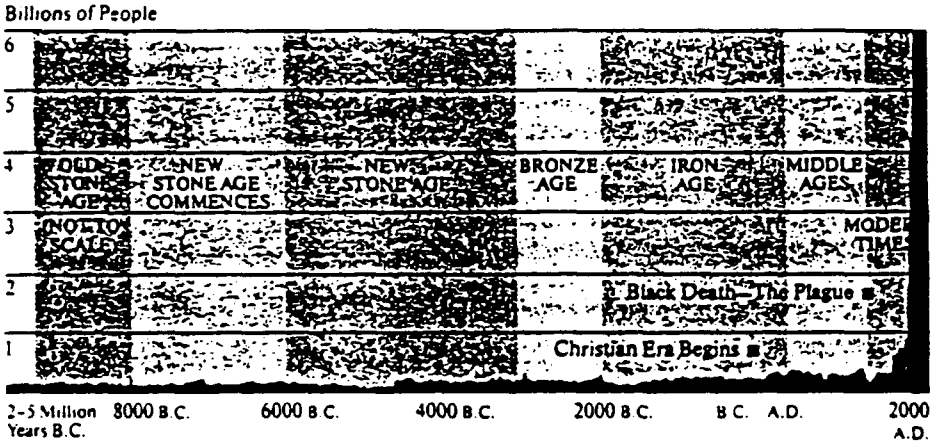


A projection of world population comparing the growth of developed countries with that of less developed countries.

Date	Estimated World Population	Doubling Time
8000 B.C.	5 million	1,500 years
A.D. 1650	500 million	200 years
A.D. 1850	1,000 million (1 billion)	80 years
A.D. 1930	2,000 million (2 billion)	45 years
A.D. 1975	4,000 million (4 billion)	35 years
A.D. 2010	8 billion?	30 years
A.D. 2040	16 billion?	

Figure 10.2 Doubling times for human population (After Keyfitz, 1962)

FIGURE P.9. WORLD POPULATION GROWTH THROUGH HISTORY



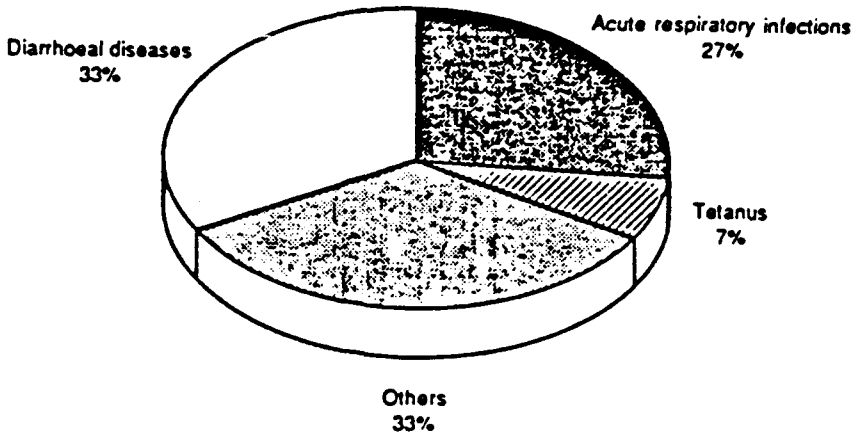
Source: Jean van der Tak, Carl Haub, and Elaine Murphy, "Our Population Predicament: A New Look," *Population Bulletin* 34, no. 5 (December 1979): 2.

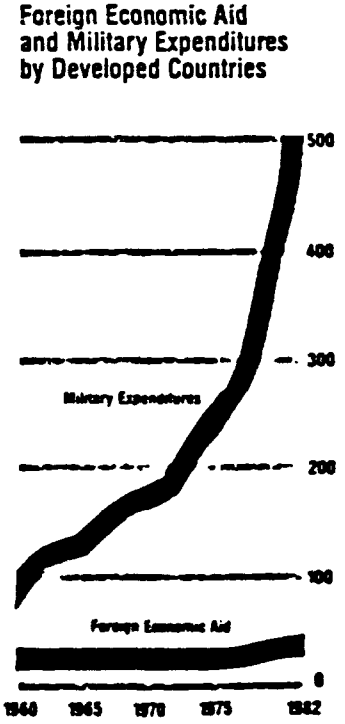
Table 9-1. Potential Reduction in Infant and Child Deaths with Proven Disease-Control Technologies

Disease	Estimated Deaths	Interventions	Effectiveness	Lives Potentially Saved
	(million)		(percent)	(million)
Diarrhea	5	Oral Rehydration	50–75	3
Immunizable Diseases	5	Vaccines	80–95	4
Pneumonia/Lower Respiratory Infection	4	Penicillin	50	2
Low Birth Weight, Malnutrition	3	Maternal Supplements; Treat Infections; Contraception	30	1
Total	17			10

SOURCE: J.E. Rohde, "Why the Other Half Dies," *Assignment Children* (UNICEF/Geneva), No. 61–62, 1983.

Fig. 1 **Causes of death in children under five years.**





A comparison of developed nations' spending on foreign economic aid with their military expenditures.

"Every gun that is made, every warship launched, every rocket fired signifies, in the final sense, a theft from those who hunger and are not fed, those who are cold and are not clothed.



This world in arms is not spending money alone. It is spending the sweat of its laborers, the genius of its scientists, the hopes of its children ....This is not a way of life at all in any true sense. Under the cloud of threatening war, it is humanity hanging from a cross of iron."

The Honorable Dwight D. Eisenhower
1890-1969
34th President of the United States
Supreme Commander, Integrated European
Defense Forces

MARINE ENVENOMATIONS

Dr John Williamson
Director of Hyperbaric Medicine
Royal Adelaide Hospital
Adelaide
South Australia

1. Learning Objectives:

Following this presentation, participants will:

a) have a perception of the international incidence and significance of the commonest human envenomations resulting from marine creatures.

b) be able to develop a systematic approach to the diagnosis, and first-aid and medical management of the more serious and the more common envenomations.

c) begin to have an understanding of the principles of prevention of such envenomations.

d) develop insight into the more pressing areas where research is needed to advance this subject.

2. Course Material:

i Brief historical introduction of marine envenomations as they have impacted upon human beings over the centuries shows that mythology, superstition and inaccurate observations have kept this subject in the realms of folklore until this century. Halstead has been a pioneer in bringing systematic documentation of observations to the subject.

ii Display of existing world epidemiological data relating to the incidence and identification of human stingings and envenomations indicates that the events affect sea shores the world over, but have their greatest preponderance in the warmer tropical and sub-tropical oceans (e.g. the Indo-Pacific region). As with all envenomations, children are a particularly susceptible risk group, with also relatively high exposure.

iii Illustration of the more common and/or more serious envenomations (*Chirodropid* and *Carybdeid* jellyfish, *Physalia* stings, sea snakes, blue-ringed octopus, sting ray

injuries, spinefish, Crown-of-thorns injuries, coral abrasions), using first-hand slide pictures and case reports enables recognition of injury patterns. Reference will also be made to Ciguatera poisoning. Human deaths most commonly result from *Chirodropid* stings in the Indo-Pacific regions. Lethal *Physalia* stingings are occurring in the United States.

iv Within the animal groupings, a suggested systematic approach to diagnosis of close skin sting or injury observation, nematocyst identification from skin scrapings (where applicable), and serum immunoglobulin G titres). First-aid management, retrieval, and subsequent medical treatment of the envenomation groupings is taking on a logical basis. Vinegar dousing and the controversial place of compression bandaging versus tourniquets will be discussed. A possible new advance in the treatment of acute ciguatera poisoning using intravenous mannitol infusion will be described.

v Reference to international specific marine antivenom availability, and usage principles includes correct animal diagnosis, selection of appropriate cases for indicated antivenom administration, appropriate route of administration, and suggested methods of avoidance of the potentially serious complications of systemic animal protein exposure.

vi An outline of current toxinological and immunological techniques which permit rational identification and management of human patients. The leading laboratory is that of Professor Joseph W. Burnett, MD, and colleagues, within the Department of Dermatology at the University of Maryland Medical School, in Baltimore, USA.

vii A discussion of specific prevention principles, which include public education, knowledge of risk times and areas of the coastlines, in-water behaviour, protective clothing, seashore patrolling, and safe enclosures.

viii A conclusion which addresses the need for better reporting of stings, envenomations and poisonings occurring in and from the seas worldwide, for high quality colour photographs of fresh stings and injuries, and for accurate first hand observations of envenomation cases to be documented. Further advances are now also

contingent upon the collection and isolation of the marine animal venoms, and their valid characterisation, both toxicologically and immunologically. Only then will specific therapeutic measures become possible for affected patients.

ix **The International Consortium for Jellyfish Stings** is currently working to the above ends, and seeks to establish an international reporting network, with some success to date. Countries participating at present are USA, Australia, Japan, Republic of China, Malaysia, Thailand, Pakistan, India, South Africa, Yugoslavia, Switzerland, Portugal, United Kingdom, and Russia. A collaborative immunological research effort between USA, Malaysia, India and Australia has resulted in several publications. It is hoped more funding by respective governments will be forthcoming for marine envenomation research - especially for scientists and clinicians working in the countries bordering the Indo-Pacific region.

3. Publications in the past 7 years:

Burnett JW, Calton GJ, Othman IB, Russo AJ and colleagues, and Russell FE and colleagues, in "Toxicon", "Ann Emerg Med", and "J Comp Biochem Physiol".

Endean R, Rifkin J and colleagues, in "Toxicon".

Edmonds C, in Med J Aust, and in book form ("Dangerous marine creatures", Reed Books Pty Ltd, 2 Aquatic Drive, French's Forest, NSW 2086, Australia.)

Williamson JA, Fenner PJ, Callanan VI, Hartwick RF and colleagues, in Med J Aust, and in several book publications (e.g. Queensland Museum Publications, and Queensland State Centre Inc., Surf Life Saving Association of Australia, Brisbane, Queensland, Australia).

4. Classical Historical Publications:

Pioneer publications by Light, Halstead, Barnes, Cleland, Southcott, Flecker, Freeman, Crone and colleagues throughout the Australian and American medical and toxicological literature.

JELLYFISH ENVENOMATION

in a Symposium on
Wilderness Toxinology

Dr John Williamson
Director of Hyperbaric Medicine
Royal Adelaide Hospital
Adelaide
South Australia

1. Learning Objectives:

At the conclusion of this Symposium Session, participants will:

a) appreciate better the world wide incidence, and frequency of jellyfish envenomation

b) learn of the diagnostic and management criteria that are presently understood

c) have an appreciation of the diverse clinical effects of jellyfish venoms, together with the common features of venoms from different species

d) be able to get a "handle" on the components of the venoms, their clinical characterisation, their toxicological and immunological propensities, and the direction of current research.

e) perceive the international research needs of the present and the future, and its possible relation to parallel research in other venom toxinology.

2. Course Material:

i An overview of jellyfish envenomation, based on the International Consortium for Jellyfish Stings' reporting system shows that these events are common, world-wide in distribution, frequently serious, and sometimes fatal. Serious jellyfish envenomation is the commonest marine mishap after drowning, near-drowning and immersion, and has been a neglected area of study.

ii Advances in biological understanding (e.g. the architecture of jellyfish tentacles, nematocyst function) and improved on-the-beach correlation of stinging case reports have permitted a

development of a systematic approach to the recognition of sting patterns on human skin.

Barnes' original observation, complimented by the original work of Cleland and Southcott, has evolved the technique of sting skin scraping and the harvesting of adherent nematocysts which permit "fingerprint-like" specific identification of the stinging species in the acute situation. This permits appropriate clinical response.

Advances in venom characterisation, especially "lethal" fractions enables more specific application of resuscitation and management practice at the clinical level.

An emerging appreciation of the antigenic properties of jellyfish venoms (complex mixtures of polypeptides and enzymes) is revealing a syndrome group of allergic reactions to jellyfish stings, including both severe (anaphylactic) and delayed effects.

iii Antigenic commonalities are being uncovered between components of jellyfish venoms, and other terrestrial venoms and toxins (e.g. the brown recluse spider and the cholera vibrio toxin). The significance of this observation remains obscure.

iv Titration of the patients days-old serum against specific venom antigens *in vitro* permits the identification of the offending venom with increasing accuracy. This not only assists clinical management, but throws light on the epidemiology of jellyfish stinging world wide.

Immunoglobulins G in human sera respond to jellyfish venom exposure by specific rise in antibody titres. These appear in a few days, and persist for many months. Significant numbers of patients exhibit cross reacting antibodies to other jellyfish venoms. No consistent pattern has yet been discerned.

v One of the most studied jellyfish venoms is that of the northern Australian lethal box-jellyfish *Chironex fleckeri*. This complex mixture contains "lethal", dermatonecrotic, savage pain-producing, and haemolytic fractions at least. THe lethal component is probably directly toxic to the human myocardium, causing cessation of contraction in systole? This action is dose- and may be calcium ion-

dependent? There is animal evidence that calcium channel blockade may offer some protection against the myocardial effects.

The existing Australian Commonwealth Serum Laboratories' specific sheep antiserum *Chironex fleckeri* antivenom is a powerful analgesic for the sting of this animal, probably protects against skin damage also, and may offer some myocardial protection. This latter seems to be enhanced in animals by the addition of verapamil.

vi Laboratory studies in Baltimore have successfully employed adsorption immunochromatography to separate and partially characterise various world jellyfish venoms, including the North American Sea Nettle (*Chrysaora quinquecirrha*), the world-wide Portuguese Man-o'-war (*Physalia* spp), and some of the Australian and South American jellyfish.

vii A dramatic new syndrome, the "Irukandji Syndrome", caused by the sting of a tiny tropical cubomedusan group (including *Carukia barnesi*) is characterised by delayed severe muscular pains and prostration, severe acute hypertension and other features suggestive of a catecholamine storm, and occasional progression to an acute global cardiac dilatation and pulmonary oedema with life-threatening hypoxia. Venom and clinical research is proceeding. Patients are assisted by the intravenous administration of phentolamine, and where indicated inotropic cardiac support.

viii There is evidence that certain jellyfish envenomations are accompanied by acute, severe, and directly localised arterial spasm and distal ischaemia of stung limbs following stinging. The pharmacokinetic explanation for this phenomenon is unknown.

ix The capture of jellyfish venoms, their preservation for transport, and their valid reconstitution and separation into components is a challenge which is currently being addressed. Freeze-drying techniques seem superior.

x Past toxicological studies may have been misled by the partial denaturation that resulted in these highly labile venom mixtures, from the crude fractionation techniques that were used at that time.

xi Happily a number of countries in the tropical regions whose shores are most commonly frequented by venomous jellyfish, are beginning toxicological research. Collaboration, experience sharing, training, and publication notification via the channels offered by the International Consortium for Jellyfish Stings is facilitating these advances. The United States and Australia presently lead these activities, but it is hoped increased government and private research funding in the different countries will stimulate these efforts in the future.

3. Recent Publications:

Burnett JW, Calton GJ, Othman IB, Russo AJ and colleagues, and Russell FE and colleagues, in "Toxicon", "Ann Emerg Med", and "J Comp Biochem Physiol".

Endean R, Rifkin J and colleagues, in "Toxicon".

Edmonds C, in Med J Aust, and in book form ("Dangerous marine creatures", Reed Books Pty Ltd, 2 Aquatic Drive, French's Forest, NSW 2086, Australia.)

Williamson JA, Fenner PJ, Callanan VI, Hartwick RF, Burnett JW and colleagues, in Med J Aust, and in several book publications (e.g. Queensland Museum Publications, and Queensland State Centre Inc., Surf Life Saving Association of Australia, Brisbane, Queensland, Australia).

Advances in Crotalid Antivenin will be taken from the attached paper and the review, *Snake Venom Immunology: Historical and Practical Considerations. J. Tox. Toxin Rev.* 7(1) 1-82, 1988, and updated.

Findlay E. Russell, MD, PhD, The University of Arizona, Tucson, Arizona 85721

BIOLOGY

In the USA, about 20 of the 120 species of snakes are sufficiently venomous to be a danger to humans. Most of these reptiles can be divided into the pit vipers (Crotalidae) and the coral snakes (Elapidae). Their distribution is shown in Table 1, and their habits and habitats are described in the references.

CHEMISTRY, PHARMACOLOGY, AND PATHOLOGY

Snake venoms are complex mixtures, chiefly proteins, many having enzymatic activity. Although the enzymes contribute to the deleterious effects of the venom, the lethal property and some other toxic effects may be due to certain of the relatively small polypeptides. These polypeptides appear to have specific chemical and physiologic receptor sites and activities.

In addition to the activities of various venom components and their metabolites, envenomation may be complicated by the release of autopharmacologic substances that can make diagnosis and treatment difficult. Thus, the arbitrary grouping of snake venoms into categories such as "neurotoxins," "hemotoxins," and "cardiotoxins" is pharmacologically superficial and can lead to grave errors in clinical judgment. A so-called neurotoxin can produce marked cardiovascular changes or direct hematologic effects. The so-called hemolytic venoms can also produce changes in the nervous system or in vascular dynamics. A patient with snake venom poisoning may present several toxic reactions -- hematological, cardiovascular, and neurological.

Rattlesnake and many viper venoms produce local tissue damage, blood cell changes, coagulation defects, blood vessel injury, and changes in vascular resistance. The Hct may fall rapidly, although hemoconcentration may occur during the very early stages. Thrombocytopenia is common, except in minimal envenomations. Pulmonary edema is common in severe poisoning, and bleeding may occur in the lungs, peritoneum, kidneys, brain and heart. These changes are often accompanied by alterations in cardiac and renal function. Renal failure may occur because of a critical deficit in glomerular filtration secondary to hypotension, or it may be due to the effects of hemolysis, or to the direct effects of the venom components. Although cardiac dynamics may be disturbed, the early cardiovascular collapse seen in an occasional patient bitten by a rattlesnake is caused, for the most part, by a fall in circulating blood volume. This appears to be associated with a loss of blood plasma and protein through the vessel walls, and to blood pooling. Most North American crotalid venoms produce relatively minor changes in neuromuscular transmission. The venom of the Mojave rattlesnake is the major exception.

Most elapid venoms cause changes in neuromuscular transmission, in nerve conduction, and to a much lesser extent in the CNS. Some elapid venoms, however, cause local tissue damage and necrosis, blood changes, and severe renal complications.

EPIDEMIOLOGY

Although 45,000 people per year are bitten by snakes in the USA, only about 8,000 cases are due to venomous snakes. Fewer than 12 fatalities per year occur, mostly in children, in untreated or mistreated cases, or in members of religious sects who handle venomous serpents. Rattlesnakes account for about 65% of venomous snake bites and for almost all of the deaths. Most other venomous snake bites can be attributed to the copperhead, and to a lesser extent the cottonmouth. Coral snakes inflict less than 1% of all bites. Imported snakes found in zoos, schools, snake farms, and amateur and professional collectors account for at least 15 bites per year.

SYMPTOMS, SIGNS AND DIAGNOSIS

Bites by venomous snakes are medical emergencies requiring immediate attention and the exercise of considerable judgment. Before treatment is started, it is essential to determine whether the snake was venomous, and whether or not venom was injected; snakes may bite and not inject venom. (No poisoning develops in about 20 to 30% of crotalid bites, and in about 40 to 50% of cobra and certain elapid bites.) When no envenomation occurs, or if the bite is inflicted by a nonvenomous snake, it should be treated as a puncture wound. It is advised that the necessary antitetanus agent be given.

Positive diagnosis for snake venom poisoning requires identification of the snake and evidence of envenomation. Although the identity of the offending snake can be suggested by the fang marks, these should never be relied on for positive identification. "Typical" fang mark patterns are often based on the anatomy of the snake's jaw and may not occur under field conditions. Rattlesnake bites may leave one or two fang marks as well as other tooth marks; single fang punctures are very common and are not uncommon in bites by some nonvenomous snakes. Double strikes also occur.

Numerical grading of rattlesnake bites is sometimes described in the literature, but it is wiser to describe cases as *minimal*, *moderate*, or *severe*, depending on all the symptoms, signs, and laboratory findings rather than on numerical grading: 1, 2, 3, 4, or 5, as based on pain and swelling. Bites by the Mojave rattlesnake, for example, can give rise to minimal edema, local tissue changes and pain, and therefore be graded as 1. The consequence can be the administration of insufficient antivenin resulting in a poor (even fatal) outcome. Diagnosis must be established on the basis of *all* manifestations.

Pit Viper Envenomation

The symptoms and signs of crotalid poisoning vary considerably, depending on the species of snake, the amount of venom injected, and other factors. If there is evidence of poisoning immediately after a bite, the possible consequences must not be underestimated. Bites by rattlesnakes, cottonmouths, and copperheads usually cause immediate swelling, edema, and pain; but contrary to popular opinion, severe pain is not a constant finding. Generally, however, some degree of pain immediately follows envenomation, and swelling and edema usually appear within 20 minutes. They are rarely delayed for more than 30 minutes, but this may occur. By the time the patient arrives at the doctor's office, a diagnosis of crotalid bite with envenomation can

usually be made (or envenomation excluded) on the basis of fang marks, swelling and edema, pain, and in bites by some species, tingling or numbness perioral or in the fingers or toes, or a metallic or rubbery taste.

Untreated, the edema progresses rapidly and may involve the entire extremity within several hours. There may be lymphangitis and enlarged, tender regional lymph nodes. Skin temperature over the injured part is usually elevated, although the patient may complain of chills. Weakness, syncope, sweating and nausea may be present. Vomiting may occur and the pulse may be rapid and weak. BP often drops and, in severe cases, shock may develop early. Particularly following bites by the Mojave rattlesnake, respiratory distress may occur, and muscle fasciculations, spasms and weakness are not uncommon. True paralysis may be seen in severe cases. The patient may complain of difficulty in swallowing, headache, blurred vision, ptosis, and marked thirst.

Ecchymosis is common in most cases of moderate or severe rattlesnake poisoning and usually appears around the bite within 3 to 6 hours. It is severe following bites by eastern and western diamondbacks, the prairie and Pacific rattlesnakes, and less severe following copperhead bites. The skin may appear tense and discolored, vesicles may form in the area of the bite within the first 8 hours, often becoming blood-filled. North American rattlesnake bites tend to be relatively superficial. Necrosis is common around the bite area in untreated cases, and superficial blood vessels in the area may be thrombosed. Most of the effects produced by snake venom reach their peak by the 4th day.

There may be hemorrhage from the gums, hematemesis, melena, and hematuria. Bleeding and clotting times are prolonged and platelet counts may fall sharply in moderate and severe envenomations. In most cases, an initial rise in the packed cell volume may occur, although in moderate or severe cases subsequent hemolysis may cause a rapid fall in the Hct.

Coral Snake Envenomation

The bite is usually associated with little or no pain, often transitory. Swelling is either absent or very minor. Paresthesia is often noted around the bitten area, and some weakness of the part becomes evident within several hours. Muscular incoordination may subsequently develop, and the patient may complain of marked weakness and lethargy. There may be increased salivation, difficulties in swallowing and phonation, and visual disturbances. Respiratory distress and failure may ensue. In fatal cases, shock, leading to complete cardiovascular failure, usually precedes death.

LABORATORY TESTS

In all but trivial cases, a CBC, platelet count, and urinalysis are essential. The following tests should also be obtained: Typing and hold; PT, PTT, and fibrinogen; other tests might include ESR, Na, K, Cl, and Ca blood levels; and CO_2 combining power. An ECG is indicated in all cases except minor ones.

TREATMENT

Pit Viper

If the patient is seen within 5 minutes of being bitten, and is several hours from the hospital, and there is evidence of envenomation, a constriction band should be placed close to the bite above the first joint proximal. The band should be tight enough to occlude lymph flow but not tight enough to impede venous or arterial flow. Suction, using Sawyer's "Extractor," applied directly over incisions or even over the fang punctures is of value during the first 30 to 60 minutes following the bite. The wound should be cleansed and covered with a sterile dressing. The affected part should be immobilized at heart level and in a functional position. The patient should be kept warm, at rest, rings and bands removed, and should be given reassurance. If the patient is within 30 minutes of a medical facility, he should be transported there, as quickly as possible, given reassurance, and the affected part immobilized.

At the hospital, if antivenin is needed, a skin test for horse serum sensitivity should be performed as described in the antivenin brochure. If the patient is mildly sensitive to horse serum, diphenhydramine IV is indicated before giving the antivenin. Some physicians routinely administer this drug prior to antivenin. A tourniquet, O_2, epinephrine, and other drugs and equipment for treating anaphylaxis should be available during antivenin administration. If the skin test is strongly sensitive, antivenin should be given *only* if life or limb is at stake, and then only under very controlled conditions.

The amount of Antivenin (Crotalidae) Polyvalent [Wyeth] to be given depends on many factors, most important of which are the severity and progression of the symptoms and signs. In minimal rattlesnake venom poisoning, 50 to 80 ml (5 to 8 vials), of antivenin (reconstituted) will usually suffice. Moderate cases may require 80 to 120 ml (8 to 12 vials); severe cases may need 150 to 400 ml (15 to 40 vials), or more. Water moccasin poisoning usually requires lesser doses; with copperhead bites, antivenin is usually required only for children and the elderly. Reconstituted antivenin should be diluted in sterile isotonic saline or 5% dextrose and given by IV drip in most cases. If necessary to inject IM, give in the buttocks. **Never inject antivenin into a toe or finger.** Measuring the circumference of the extremity at 3 points increasingly proximal to the bite and recording the measurements every 15 to 30 minutes provides a guide to antivenin dosage. If additional antivenin is needed, it is added to the IV drip and given over 3 to 4 hours. Antivenin is probably of less value if not administered within the first 12 hours. After 28 hours it is of questionable value, although it may reverse coagulation deficits even at 30 hours. IV fluids should be kept to a minimum, except when shock or hypovolemia are present.

The appropriate antitetanus agent should always be given, and a broad-spectrum antimicrobial administered in serious cases. In many cases, either early or late in the poisoning, blood volume decreases and perfusion fails, often with concomitant lysis of RBCs and platelet destruction, necessitating transfusions and parenteral fluids. Plasma or albumin may be used to treat hypovolemia. If there is a decrease in RBC mass, either through lysis or bleeding, packed cells or whole blood should be given. When these complications are accompanied by defects of hemostasis -- i.e., abnormal clotting or lysis of cells or clots, or a disturbance of platelet activity -- replace with specific clotting factors: fresh frozen plasma or platelet transfusions.

At the first sign of respiratory distress, O_2 should be given and preparations made to provide mechanical support. Tracheal intubation or tracheostomy may be indicated, particularly if trismus, laryngeal spasm, or excessive salivation is present. Aspirin or codeine may be used for pain, and meperidine or morphine if pain is severe. Mild sedation with diazepam is indicated in all severe bites if respiratory depression is not a problem.

Sedation should reduce the amount of analgesic necessary.

The wound should be cleansed and covered with a sterile dressing. The injured part should be immobilized in a position of function. Surgical debridement of blebs, bloody vesicles, or superficial necrosis, if present, should be carried out between the 3rd and 10th day, and may need to be done in stages. The injured part should be soaked in 1:20 Burow's solution t.i.d. for 15 minutes. Antihistamines and corticosteroids are of no proven value during the acute stages of poisoning and the latter may be contraindicated. Antihistamines may be of value as a sedative in children.

Fasciotomy should be discouraged. It is usually unnecessary and reflects the use of insufficient antivenin during the first 12 hours of the poisoning. It may be necessary, however, when there is objective evidence (wick measurements) of a constant, elevated intracompartment pressure (above 40 mm/Hg) over an extended period of time and other evidence of severe vascular embarrassment. These, however, are very rare findings.

Follow-up care is of the utmost importance. Contractures can be avoided by early corrective measures and exercises. Within several days of the bite, a complete evaluation should be made of joint motion, muscle strength, sensation measurements and girth. Follow-up care should include sterile whirlpool baths and daily cleansing of the wound with 3% hydrogen peroxide followed by 15 minute soaks in 1:20 Burow's solution.

Coral Snake

The general principles for pit viper envenomation should also be considered in coral snake bites. The value of suction and other such first aid measures have not been determined. Three vials of antivenin (*Micrurus fulvius*) should be given when a diagnosis of coral snake envenomation has been established. If symptoms develop, 3 to 5 additional vials may be indicated. The physician should contact a poison control center, zoo, or Wyeth Laboratories for the nearest source of this antivenin. In severe cases, cardiopulmonary and intensive care may be indicated.

All data in this review are taken from:

1. *Snake Venom Poisoning*, Scholium International, Port Washington, N.Y. 11050, 1983.
2. *Merck Manual*, 15th & 16th ed., 1988, 1990.
3. *Conns Current Therapy*, Saunders, Philadelphia, 1988.
4. Snake Venom Immunology. *J. Toxicol Toxin Rev.* 7:11-82, 1988.
5. Aids, Cancer & Snakebite. *West. J. Med.* 148: 84, 1988.

Extractor™ Pump Kits can be obtained from Sawyer Products, Box 188, Safety Harbor, FL 34695.

Table 1
Some Medically Important Snakes of the United States*

Snakes	WA, OR, ID	CA, NE	AZ, NM	TX	MT, MI, WI, MN, SD, ND, NE, IA, WY, UT, CO	KS, OK, AR, MO	TN, KY, IL, IN, OH	NC, SC, GA, AL, MS, LA	FL	PA, NJ, MD, DE, VA, WV, NY, New England
Pit Vipers (Crotalidae)										
Cottonmouths and copperheads (*Agkistrodon*)										
Cottonmouths (*A. piscivorus*)				X	NE, IA	X	TN, KY, IL	X	X	VA
Copperheads (*A. contortrix*)				X	NE, IA	X	X	X	X	X
Rattlesnakes (*Crotalus*)										
Eastern diamondback (*C. adamanteus*)								X	X	
Western diamondback (*C. atrox*)		X	X	X		OK, AR				
Sidewinder (*C. cerastes*)		X	AZ		UT					
Timber (*C. horridus*)				X	MN, WI, NE, IA	X	X	X	X	X
Rock (*C. lepidus*)			X	X						
Speckled (*C. mitchelli*)		X	AZ							
Black-tailed (*C. molossus*)			X	X						
Twin-spotted (*C. pricei*)			AZ							
Red diamond (*C. ruber*)		CA								
Mojave (*C. scutulatus*)		X	X	X						
Tiger (*C. tigris*)			AZ							
Prairie (*C. viridis viridis*)	ID		X	X	Not MI, WI, MN	KS, OK				
Grand Canyon (*C. v. oreganus*)		AZ								

* Certain groups of adjoining states are treated as units. The symbol "X" indicates that distribution of the species is widespread within the unit. Restriction of a species to a part of a unit is indicated appropriately.
From: Russell FE. *Snake Venom Poisoning*. Port Washington, NY: Scholium International, 1983.

GROUP COHESION AND THE SELECTION OF EXPEDITION MEMBERS

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This paper concerns the way expedition members are selected, and how this process may affect group cohesion. Selecting people for dangerous or challenging enterprises is as old as history. The Bible describes how Gideon defeated the enemy with 300 men selected from an army of over 30,000. Greek history tells of Leonidas and 300 Spartans holding out to the last man against Xerxes's huge force at Thermopylae; the ultimate cohesion. On his expedition in search of the Golden Fleece, Jason took "fifty heroes of Greece" with him (apparently his selection procedures were heavily influenced by public affairs officers). Each of Columbus's four expedition recruited sailors differently, depending on supply and demand; at times picking through crowds of volunteers, and at times recruiting convicts. Col. William Travis, trapped in the Alamo by Santa Anna's superior forces, allowed each man to choose for himself whether to leave, or to stay and fight to the death.

For the "Endurance" expedition of 1914, Shackleton selected 56 members from over 5000 volunteers. Thirty-five years later, Sir Edmund Hilary, describing the selection of the members of the expedition which finally conquered Mt. Everest, observed rather cynically that when it came to choosing between two well-qualified applicants for the final assault team, the fact that one had enough money to help finance the expedition was the tie-breaker. Jimmy Doolittle asked American bomber crews to volunteer to fly with him on a secret mission which he would lead, and only after training was complete and they were en route to the target did he reveal their mission, to bomb Tokyo. Morale was so high that the stand-by crew members who did not get to go on the extremely dangerous flight were quite disappointed.

Finally, even today, the selection of a new class of U.S. astronauts involves starting with some 3000 volunteers and ending with about 20 astronaut trainees.

How is this accomplished, this process which begins with a group of strangers and ends with a group bound so tightly that its members will sacrifice their lives for each other? We will not consider physical fitness criteria, or developing the proper technical skills. Instead, using a

background of aerospace medicine and psychiatry, we will look at how criteria of personality and motivation may be addressed in enterprises which may involve deadly danger, where members may depend utterly upon each other for survival.

CRITERIA AND MATHEMATICS

Aerospace medicine is the medical specialty involved in the selection, medical care, and health maintenance of aviators and, today, astronauts. Aeromedical standards generally pertain to two factors, safety and health, which may be translated into statements about selection for expeditions: standards should pertain to 1) accomplishing the expedition competently, dependably and safely, and 2) maintaining the health and safety of the individual member. Among the first challenges faced by early flight surgeons was that of medical and psychological selection of volunteers for flying training. This process began about the time of the First World War, when the early practice of transferring men worn out or unfit for army service into the air service led to a disastrously high accident rate. Something had to be done to select men who were truly fit to fly.

Early U.S. standards were based on the normal values from a textbook of physiology, but after about two months the flight surgeons found that these requirements were so severe that no one had been able to pass them, and so the standards had to be changed. Much attention was given to eye, ear, nose and throat examinations, as well as tests of equilibrium and resistance to hypoxia. In addition, applicants in some countries underwent a variety of somewhat more exotic tests: timed response to colored lights, tremor in response to an unexpected bomb blast as a measure of emotional control, ability to raise a board which supported a rod standing on end without toppling the rod, counting simulated artillery positions on a panorama rolled past a viewing slit, ability to hold one's breath against 50 mm Hg pressure, and so on. (Armstrong, p. 35 ff.)

Psychological fitness was determined through a semi-structured half-hour interview, designed to test the quality of the applicant's motivation to fly as well as to judge whether his personality fit the current ideas of what sort of person an aviator should be. In fact, most of these standards were based on the medical officers' ideas of what fliers should be like, with little or no validation of the standards against reality. Did they really discriminate between successful and unsuccessful pilot trainees--or safe and unsafe aviators--and how did one know? The few research protocols intended to determine this were inconclusive. Still, the morale of the fliers was generally good, as any reading of biographical or historical documents of the

period will show. Late in the 1930s, the Army Air Corps only had some 3000 aviators, but they regarded themselves as a special breed, sharing skills and experiences beyond those of those who did not fly.

With the vast expansion of aviation training at the outbreak of World War II, the mathematics of selection changed from one in which the Air Corps had the luxury of selecting only the best of the many applicants. Now U.S. Army and Navy aviation needed tens of thousands of men. One of the great mathematical lessons of selection quickly became clear: the larger the number of standards used for selection, or the more rigorous those standards are, then the smaller the number of people who can meet them all.

If you wish to choose four people who will be guaranteed successful, based on qualities that may be found in only one percent of the population, then you must examine 400 people and reject 396 to get your four. This may be practical if you have lots of applicants and lots of examiners, but not if you are short of one or the other. If you are willing to choose your people based on criteria that are wrong 20% of the time, but which involve qualities found in 50% of the applicants, then you need to examine ten, accept five, and wash one out later. Your life will be a lot simpler, especially if you only <u>have</u> 10 applicants! Let me remind you again that the more criteria you have, the more difficult your job will be, since the person with the really good vision may not be clever enough, while the one who is clever may wear glasses. How good must the vision be? How clever must the applicant be? Can contact lenses be substituted for the glasses? How do you know these criteria have anything to do with the success of your project? By asking such questions, the number or the severity of the selection criteria may be diminished, and the pool of potential selectees enlarged. For this reason, the selection criteria for aviation training have gotten less strict over the years, and many criteria have been dropped altogether; e.g., right bundle branch block.

Another mathematical fact is that the specificity (false positives) and selectivity (false negatives) of your criteria, calculated against the prevalence of the disorder, must be considered. The rarer the condition, the more expensive your testing will be, the more false positives you will encounter, and the more applicants you will lose. I do not propose to discuss this further, but anyone developing a set of medical selection criteria should ask a statistician whether Bayes's Theorem applies.

People going on an expedition need to develop cohesion--to bond together, to accept the expedition as a valued personal experience, and to accept its goals as their own so strongly that they will choose to act for the good of the expedition over their own personal interests, perhaps even to the point of sacrifice. Many of the traditions of such choices arise from military experiences, where the individual feels helpless to affect the outcome of a battle, but becomes bold when able to identify with someone or something perceived as stronger or more powerful, such as a charismatic leader, or a tightly knit group, or a powerful piece of equipment. Borrowing power from such a source, the individual once more becomes master of the situation. Imagine the difference in spirit of a fighter pilot flying in an F-16, and the same pilot caught on the ground in a mortar attack.

Coping skills

People bonded by dramatic experiences feel they're special--different--better. They are more willing to forego ordinary pleasures and rewards to win the esteem of a valued leader or comrades. Some feeling of atruism--that others will benefit from the project, justifying one's sacrifice--is seen as being of greater value or good than any benefit to one's self. Thus, the difficulty or suffering involved in achieving the goal is accepted.

Everyone who plans for endeavors such as expeditions understands the need to anticipate what may come--to plan, to think ahead and link what one knows to what might happen. Another mature coping skill is a healthy sense of humor, frequently self-effacing, that keeps things in perspective. Look carefully at the quality of an applicant's humor, though. One must keep in mind that humor used as a weapon (sarcasm) may be quite destructive to the bonding process. Those who prepare for endeavors such as expeditions understand the need to anticipate what may come--to plan, to think ahead and to link what one knows to what might happen. Some people borrow energy from one part of their lives to use for another purpose, the process of sublimation. Some are able to suppress unpleasant realities which they cannot control--remember Scarlett O'Hara's "I'll think about it tomorrow." These five coping mechanisms--altruism, anticipation, humor, sublimation and suppression--are discussed by Vaillant in the reference given. They are among the most adaptive used by mature and capable individuals, and, I believe, should be among those used in selection for stressful endeavors.

How might this information be of use to someone selecting members of an expedition? (Remember, we are not addressing physical criteria or technical skills, although the process is probably similar in those areas.) The first tentative selection criteria should come from those who know something firsthand about the expedition and its goals. Intuitively, one might look for someone who's already done it. What problems were encountered, and how were they handled? What sorts of people did well, and did not do so well? Why did they volunteer--what sorts of interest seemed to hold up? What mixes of motivation and personality seemed best? From such data, the initial criteria may be deduced. Selection for such stressful activities as overwintering in the Antarctic have confirmed the usefulness of factors of occupational ability, psychological stability, and social compatibility. (Taylor, 1987). People have to be resilient enough to deal with boredom and social isolation, and these qualities may not be prominent in people who volunteer out of a need for adventure or excitement. There are some very unhealthy reasons for wanting to be alone for six months!

As for the matters of motivation and personality that underlie group cohesion, a number of questions arise: What sort of job is it? How long will it last? What size group will be formed? How closely knit will it be? Will members be able to have privacy? Will success depend more on cooperative efforts or on individual requirements? Will some aspects require leaders and followers to trade roles? If both men and women are chosen, what sorts of problems may arise, and how will they be handled? Will sexual partners be specifically chosen or excluded? As criteria are decided upon, are these technically measurable, or do they depend on interpersonal assessment by leaders or other expedition members?

One factor that is clear from military experience is that the tougher the selection criteria, and the more rigorous the training, the better morale is likely to be. Members will be assured of each others' competence and determination, and be more willing to trust each other. Training itself is a great source of bonding--NASA takes people with various disciplines and turns them into astronauts with various disciplines. Training should be tough and realistic, clearly related to the job to be done. Harassment for its own sake is not as effective. The tougher the training, the better people feel about their ability to deal with tough realities. This is a part of effective leadership, surely a crucial part of group cohesion, and one much too complex to discuss further in this presentation.

Ethics and relevance

To summarize, the more standards used for selection, or the more rigorous those standards are, the smaller the number of people who can meet them all. Medical standards should generally pertain to two factors: 1) competent, dependable and safe accomplishment of the expedition, and 2) maintaining the health and safety of the individual member. Since failing to meet a standard removes a person from a chance to go on the expedition, justifying that standard should also include the concept of fairness, and of equal opportunity. When selecting expedition members, then, the standards themselves must meet the meta-standards of safety, health, dependability, competence and fairness. Of course, the ultimate arbiter of the validity of any such standards is the success of the member and of the expedition, but this may not be known until it's all over.

Validity

Some traditional medical standards have been around for a long time, and the reasons behind them have been lost. Minimum weight standards and automatic chest x-rays are good examples. We may have to decide whether to continue to use such standards, to try to figure out what they're for, or scrap them. Bureaucratic process being what it is, the burden of proof is upon the one who wishes to change accepted standards. This makes it difficult to apply new information or techniques to the dynamic process of selection and health maintenance. Refining standards is hard enough in, say, the technically-oriented field of cardiology. It has been even harder in subjective processes such as evaluating the mental fitness of candidates, or determining how well they should get along together.

Resistance of applicants

Doing clinical tests on people who are not patients may lead to difficulty. Using Magnetic Resonance Imaging (MRI) to look at old head injuries, for example, might show you some unidentified bright objects (UBO) compatible with multiple sclerosis in a person who has never had any symptoms. Now what do you do with this person if she's an astronaut candidate? People may refuse to volunteer for academic studies because they fear that, using new techniques, we might find something which could disqualify them. Lack of assured confidentiality can also lead some not to volunteer as subjects: for example, fliers may fear the possible loss of flying status because of findings incidental to research. This might become an issue in getting the necessary informed consent.

Select-in and select-out

One way to look at selection standards involves two concepts known as *select out* and *select in*. Working with mental health standards has been simplified by the recent development of more objective criteria for psychiatric diagnosis (DSM-III-R, 1987), which allow the examiner reasonable and reproducible means of establishing the major psychiatric diagnoses.

Some mental health standards are fairly self-evident. Most people would consider a person with a psychotic disorder to be neither safe nor dependable on an expedition. Psychotropic medications might render a person unsafe under some conditions, either because of the nature of the condition requiring their use, or because of their effects. Conditions formerly known as neurotic (e.g., phobias, generalized anxiety disorders, functional amnesias) might generally be considered disqualifying on the basis of their interference with safe and effective interpersonal interactions. Drug and alcohol abuse are obviously dangerous. However, diagnosis or rational standards may not be so easily established in other areas, specifically the personality disorders. Some of the most difficult decisions would concern the amount of disturbed behavior (obsessive neatness, passive aggressiveness) necessary to disqualify a person from an expedition.

The NASA experience

Once those with mental problems are selected out, those who remain are presumably qualified. Now the job is to determine which of these mentally fit persons should be selected into the group. What normal qualities are desirable, or undesirable? One of the most rigorous selection processes involves picking astronaut trainees from the huge pool of volunteers. NASA has had 13 selection cycles from 1959-1989: of about 25,000 applicants; about 200 were chosen and about 115 have flown in space. Thus, the chance of selection runs about 1%. Let's look at NASA's methods.

The original 7 Mercury astronauts were chosen in the early 1960s from 508 military pilot applicants.

The 1965 selection included emphasis on academic achievement; 6 were chosen from 909 applicants with doctorates in science, medicine, and engineering.

Today, astronauts are chosen in three categories: pilot astronauts, mission specialist astronauts, and payload specialist astronauts. About 3000 people may respond to an announcement that a new group of astronauts is to be chosen. The selection process goes through four phases:

PHASE 1. The civilian applicants' basic qualifications are evaluated by the Astronaut Selection Office: about 60-90% are "qualified" and move forward.

PHASE 2. Applicants are re-evaluated within their own disciplines by specific boards. [Military applicants enter here, having been screened by their respective services.] Each is scored as Qualified, Well Qualified, or Highly Qualified. Those in the last category move forward (about 13-30% of the group).

PHASE 3. Applicants are rank-ordered within their own specialty group. Medical records are screened, and security clearance is evaluated. The Astronaut Selection Board (comprised mainly of experienced astronauts, with equal opportunity representative and personnel management representatives) pick the top 20-25% (2-5% of the original applicants) to come to Johnson Space Center (JSC) for medical and psychiatric examinations, and interviews. The actual number to be selected is based on the projected needs of the Astronaut Office for the next three years, including attrition of the current astronauts.

PHASE 4. This five-day process at JSC includes extensive medical and psychiatric examinations, orientation to NASA, tours, social events, and an interview.

The medical examination is rigorous, and includes 24 hour Holter cardiac monitoring and 24 hour blood pressure monitoring, as well as cardiac fitness testing. The mental health examination involves a structured diagnostic interview, psychological testing, and a discussion of each applicant within a meeting of all the examiners, where strengths and vulnerabilities are discussed. In a recent selection cycle, 9 of 106 candidates (8.5%) had psychiatric diagnoses (two disqualifying: depression and a personality disorder) and 12 more (11.3%) had "near-diagnoses, meeting at least half of the criteria necessary to make a particular diagnosis. (Santy, personal communication.) Both the medical and mental health selection processes lead to a simple decision--the applicant is qualified, or is not. This is the only information available to the Astronaut Selection Board.

The interview (or, more specifically, ***THE INTERVIEW***) is conducted by the Flight Crew Operations Director, some current astronauts, and equal opportunity and personnel management representatives. Actual selection depends about 60-80% on the interview ranking, although the medical and psychiatric evaluations may disqualify an applicant. About 15% of those seen

at JSC are selected for astronaut training, representing about 1% of the original applicants.

Thus, this most rigorous of selection processes for what some regard as the ultimate expedition considers educational, professional, medical and mental health factors. A number of highly trained experts spend a great deal of time and effort to determine which applicants should be disqualified. However, once an applicant is found fit in all other ways, the human element re-enters, to determine which of these qualified people will be chosen. In the last analysis, a face-to-face interview with current astronauts and a few other specialists is what really counts, an interview which heavily weights interpersonal and other intangible factors. A lot of expertise goes into presenting this board with people who *are qualified* to be astronauts, but the board of peers chooses those who will *become* astronauts. Gideon, Leonidas, Columbus, Shackleton and Jimmy Doolittle would understand.

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EXPEDITION MEDICINE: PRACTICAL CONSIDERATIONS

HOWARD DONNER, MD

OBJECTIVES:

Following this presentation, participants will be able to:

1. Identify skills necessary to practice emergency care in extremely remote wilderness environments.

2. Compare the fundamental philosophical differences between urban and third world medical care.

3. Become familiar with medical problems commonly encountered in the expedition environment.

4. Design and organize the remote expedition medical kit.

Wilderness Medicial Society
July 1991

EXPEDITION MEDICINE: PRACTICAL CONSIDERATIONS

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There's good news and there's bad news . . . The good news is that if you're involved in primary care, you're already equipped to handle the majority of medical problems that come up in most expeditionary medical scenarios. The only bad news is that there are skills which you'll need to bone up on before embarking, as medical officer, to parts unknown. Fortunately the following list of skills can be easily learned within a short period of time by any motivated health care practitioner.

PREREQUISITE SKILLS:

- You are well advised to take an **Advanced Trauma Life Support** (ATLS) course which includes a basic review of the 'ABCDE's, and provides a general approach to dealing with the multiple trauma patient. The skills reviewed in ATLS are key to providing a reasonable level of trauma support on an expedition.
- You should be well versed in **preparation for foreign travel**. You will generally be considered the expert source for any questions regarding appropriate immunizations, malaria prophylaxis, etc.
- Familiarize yourself with the **endemic diseases** common in the area of the expedition, for example the local malaria risk, exotic diseases, local animal and arthropod hazards (e.g. bears, snakes, scorpions, leeches).
- Prepare yourself for the all-pervasive **travelers diarrhea**. Learn how to help prevent this problem with appropriate hygiene. Learn how to differentiate clinically between dysenteric and non dysenteric diarrheas, including when to treat conservatively and when to incorporate antiinfectives. Become expert in **water disinfection** methods for the group.
- **Environmental concerns**, for example, knowledge in the area of cold injury, including prevention and treatment of hypothermia and frostbite for those heading towards cold environs. A comprehensive understanding of altitude problems, such as AMS, HAPE and HACE is requisite for those "going higher".
- You will need to be comfortable **improvising splints** for common **orthopedic injuries** including the use of the 'SAM' splint or other improvised splinting systems. The improvisation and application of **femoral traction** should also be learned. The use of fiberglass splints (such as the 3M 'One Step') is helpful. It is key to develop fundamental skills in the use of **adhesive tape** for stabilizing the inevitable sprains and strains of expedition life (i.e. thumbs, ankles etc.) Learn to recognize and treat common **overuse syndromes** that inevitably present as aches and pains on the trail (i.e. "sahibs knee", tendonitis, etc.).

• Familiarize yourself with basic **patient transportation and evacuation** systems including improvised methods if necessary.
• Develop some **basic dental skills**, for example the use of temporary filling material, i.e. cavit, and perhaps some more sophisticated skills such as simple extraction of abcessed teeth. An excellent book for the dentally naive is Where There Is No Dentist, put out by the Hesperian Foundation.
• Become familiar with the acute management of **epistaxsis**. This should include both posterior and anterior nasal packing (see medical equipment below).
• The medical officer should be familiar with the management of **corneal foreign bodies and corneal abrasions**, as well as the management of **UV keratitis** (snowblindness).
• Wound care management of basic abrasions and lacerations in the backcountry, including the **use of suturing material** (both deep and skin). Optimal treatment of **infected blisters and burns**, as well as surgical management of **ingrown toenails** and **I&D of abcesses.**
• Some basic **nursing skills** are essential. This may frighten the over-pampered physician. You must learn how to administer all of the IM and intravenous medications and fluids carried.

> Remember: a creative and common sense approach to problem solving often proves more useful than pure medical knowledge (which too often is based on sea level dogma).

PRE-TRIP CONSIDERATIONS:

• It is crucial to determine the pre-existing medical problems of each expedition member. A record should be compiled for each person including his/ her blood group (if "inter-group" transfusion capabilities are opted for), chronic illnesses, prior surgery (e.g. appendectomy?), prior injuries, drug allergies and medications. Appropriate vaccinations should be obtained and expedition members should carry documentation on an International Certificate of Vaccination.
• Members should be encouraged to obtain a dental exam prior to leaving as well as treatment of any minor medical or surgical problems (e.g. hemorrhoids, athletes foot, troublesome toenails, etc.)
• The medical expertise of the group should be determined. This information may be very useful when managing a medical emergency or orchestrating a rescue.
• Another consideration is getting the medical kit through customs. This usually presents minimal difficulty if the medical equipment is being carried by a doctor or nurse. Be sure to carry your medical license for verification. Horror stories do exist of medical kits being confiscated by customs. Be forewarned.

• Pre-arrange for professional rescue and become familiar with avenues to take should the need arise. For example, in Nepal all helicopters are operated by the Royal Nepal Army. If a request for rescue is sent, a helicopter typically will not leave Kathmandu until someone has paid in advance for the flight. This can be arranged through a trekking agency or at times the embassy of the country sponsoring the expedition. This requires registering with your embassy on arrival in Kathmandu. The bottom line is . . . pre-arrangement can greatly expedite the rescue process should the need arise.

• Although many high altitude expeditions are getting away from the traditional use of climbing oxygen, it is certainly prudent to bring medical oxygen on most large scale expeditions or treks. Remember that there are stringent regulations involving the shipping of medical oxygen via aircraft. Oxygen is considered a hazardous material. Your alternatives are: 1) Ship your oxygen by surface at least three months in advance, 2) Plan on purchasing or renting your oxygen equipment in the country of your expedition (note: its next to impossible to obtain in many third world countries), 3) Pre-arrange to have the oxygen shipped via air freight utilizing the assistance of a hazardous materials shipper. Inquire well in advance!

ADDITIONAL CONSIDERATIONS:

As medical officer your primary responsibility is your expedition group. However, in most third world countries, you will invariably be called upon to help the locals. Most of us instinctively wish to help, however there are a few concepts you should consider prior to administering to the local sick. In many third world countries, such as Nepal, the government attempts to establish and maintain local health posts. Delivering wholesale medicine on the trail can destoy the confidence of natives in local health care systems. Additionally, inappropriate or inadequate trailside treatment may destroy confidence in western medicine leading villagers away from appropriate care in the future.

Remember that many third world people have little understanding of the basis of western medical practice (i.e. the word for germ in Nepali is the same as that for insect). The use of pills is often misunderstood. More significance may be attributed to the shape or color of the pill than the medicine itself. Patients may attempt to collect medicines which are indiscriminantly distributed to others at a later time.

Consider teaching "self-care" methods, e.g. wound hygiene, hydration with diarrhea, rather than being eager to dispense medication. Be certain that if treatment is provided, that full explanation and written instructions are given (using symbols or via interpreter). Perhaps more importantly, do not indulge in "heroic fantasy". The fact that you are trekking through does not imply that you must take on the responsibility for the village healthcare.

An often neglected aspect of expeditionary medicine is the health care of the porters. A common misconception is that porters are natives and thus immune to the problems that affect westerners. Unfortunately serious illnesses and deaths amongst porters have occurred because they were not observed for symptoms of hypothermia, frostbite, altitude illness, etc. Remember, as medical officer, your responsibilities extend to your ancillary staff as well as your western expedition members!

THE EXPEDITION MEDICAL KIT:

Organizing the medical equipment for an expedition requires an enormous amount of planning and forethought. No matter how much equipment is hauled in, one cannot possibly prepare for every conceivable illness or accident.

What should one take? There is a wide range of opinions ranging from "nothing but a triangular bandage and swiss army knife", to a collection which would stagger the local porters. The surgical types inevitably anticipate trauma, while medical people favor pills and shots of all types. Remember, when medical kits are too bulky and heavy they are likely to be left behind and not be available when needed.

The specific contents of the medical kit clearly depends on many factors, including:

- the environmental extremes of the trip
- endemic diseases
- medical expertise of the medical officer
- medical expertise of the expedition members
- number on trip
- responsibility for local health care
- length of trip
- distance from definitive medical care
- availability of rescue (e.g. helicopter, professional SAR, etc.)

The expedition doctor must recognize that is impossible to anticipate every problem. Furthermore there is a wide variation in approach to most medical problems which generates a healthy requirement for "artistic license".

The medical kit for a climbing expedition is typically divided into two major groups: 1) medical kit-trek, and 2) medical kit-climb. The medical kit for the climb is further divided into 1) personal kit, 2) base camp kit, and 3) a mobile camp kit (one per camp).

The **personal kit** should be carried by each expedition member. It includes the most commonly used items and might contain the following:

1) non-narcotic analgesics
2) anti-inflammatories
3) throat lozenges
4) sunscreen and lip protection
5) water disinfection
6) blister care
7) minor wound care
8) insect repellent
9) malaria prophylaxis (if risk exists)
10) vitamins
11) personal medications (for pre-existing problems)

The contents of the **mobile camp kit** will depend on many factors of bulk and weight constraints and is often loaded into small polyethylene containers (tupperware).

The remainder of this section will concentrate on the supplies for the **main medical kit**. There are infinite methods for organizing this kit into subgroups. Use whatever method seems logical to you.

1. ANTIBIOTICS

Even in a hospital setting two doctors seldom agree on the optimum antibiotic for treating a given disease. Prepare for the common problems:

- Upper and lower respiratory infections- your choice
- Skin and soft tissue infections- consider a first generation oral cephalosporin; **Duricef** (cefadroxil)- is preferable because of the q12-24 hr. dosing schedule (i.e. less bulk)
- Bacterial diarrheas- remember that many enteric pathogens are currently resistant to the old standby **Septra**; therefore a quinalone antibiotic should be included (i.e. **Cipr**
- Giardia- **Tinidazole** or metronidazole
- Rickettsial illness (rare)- **Tetracycline**
- Helminthic infections (hookworm, roundworm and tapeworm)- **Mebendazole** or other antihelminthic
- Malaria (for prophylaxis or presumptive treatment) - **mefloquine** or chloroquine and fansidar

(Note: injectables will be covered later)

2. ANALGESICS

Percocet (or other strong p.o. narcotic)
NSAIDs (non-steroidal anti-inflammatories) e.g. **Naprosyn 500**- should be carried in large 'bucket' to provide for the many aches and pains developed on the trail
Acetominophen- for those wishing to avoid NSAIDs

3. RESPIRATORY

Albuterol metered dose inhaler or spinhaler- commonly overlooked medical item; essential for cold or exercise-induced brochoconstriction, a common malady on mountaineering expeditions at higher altitudes; also serves as an important adjunct in the treatment of lower respiratory tract infections

4. ENT

Rhinorockets- light-weight, simple method for anterior nasal packing
Afrin or other topical nasal decongestant- a coveted item on mountaineering expeditions; essential for upper airway congestion which often occurs; also helpful for eustachian tube dysfunction during altitude changes
Cortisporin otic or other topical anti-infective
Seldane or other non-sedating antihistamine which have a myriad of uses for allergic symptoms
Throat lozenges or hard candy for the omnipresent "altitude throat" (bring lots)
Sudafed or other oral decongestant

5. EYE

Tobrex or other potent topical antibiotic
Cyclogyl (cyclopentolate HCL) or other intermediate-acting cycloplegic for relieving the ciliary spasm of uveitis (i.e secondary to U.V. photokeratitis)
Tetracaine-essential for corneal examination; also useful in extreme conditions for allowing a climber with disabling photokeratitis to get off the mountain (suboptimal treatment!).
Fluorescene strips for staining corneal defects
Eye patches- for corneal abrasions, photokeratitis

6. CNS

Valium
Haldol- note: psychotic breaks are not uncommon under the extremes of expedition life (Imagine seven days in a snow cave with someone you dislike)
Caffeine- useful for a little extra 'get-up-and-go' or to relieve caffeine withdrawal headaches
Dexedrine- very controversial, though may be life-saving under extreme conditions, i.e. 'get off the mountain under your own power or die' (not normally recommended).
Transderm Scopolamine- motion sickness

7. CARDIOVASCULAR

Inderal LA- chest pain or MI
Nitrostat/ Transderm Nitro- chest pain or MI
Nifedipine- HAPE, angina, hypertension, and effective for Raynauds phenomena

8. GYN

Monistat suppositories
Urine pregnancy test (conspicuously absent in most expedition kits)- very important for ruling out ectopic pregnancy in the reproductive age female with pelvic pain and/or vaginal bleeding
Sanitary napkins

9. GI/ PROCTOLOGY

Peptobismol tablets- for symptomatic treatment of non-dysenteric diarrhea
Immodium- irreplaceable for long bus rides or summit bids (avoid indiscriminate use)
Colace and **Metamucil** for "freeze dried megacolon"
Compazine suppositories (melt at high temperatures) and tablets- added benefit of respiratory stimulant at altitude
Antacid tablets- reflux common at altitude
Oral rehydration solution (dehydrated packettes), e.g. "Jeevan jal" or "Infalyte" (homemade ORS can also be prepared)
Annusol HC and **Tucks pads** (containing 50% witchhazel)- hemorrhoids are extrem common on expeditions

10. ALTITUDE

Diamox- usual dose 250mg BID or one 500mg spansule; smaller doses are effective for improved sleep, i.e. 125mg. q HS
Decadron 4mg tablets- for treatment of cerebral edema
Lasix 40mg. tablets- treatment of peripheral edema

11. TOPICALS

Nizoral (or other antifungal)
Kenalog .1% cream (or other intermediate potency steroid)
Insect repellant with DEET
Silvadene cream- for burns
Topical antibiotic (e.g. Bactroban, Polysorin)
Vaseline or other ointment such as Aquaphor or Blistex for treatment of chapped lips or fever blisters (some choose to include Zovirax ointment for this)
Aloe Vera Gel- for superficial burns
Labiosan (or other bomb-proof sun protection for lips)
Betadine or Hibiclens
K-Y Jelly- for rectal or vaginal exams
Footpowder

12. BLISTERS

Moleskin- tons of it!
Adhesive foam- for fashioning donuts and padding for boots
Spenko Second Skin- expensive, but indispensable for painful, ulcereated bases of de-roofed blisters; also helpful for small burns

13. WOUND SUPPLIES

Steristrips (multiple sizes)
Tegaderm or **Opsite**- for abrasions
Sutures- multiple sizes, both nylon and absorbable
Superglue- for instantaneous treatment of painful skin fissures (don't quote us)
Misc. gauze, bandaids, etc.

14. SURGERY

14G. Angiocath- emergency tube thoracostomy
Splinter Froceps (best are Uncle Bill's tweezers)
Disposable skin stapler (Ten Shot Precise, 3M)- especially useful for scalp lacerations
#11 scalpels for I&D
Instruments: bandage scissors, tissue scissors, needle drivers, and **forcep**
10 or 20 cc. syringes with **18 G. angiocaths** for high-pressure wound irrigation
TB syringes- for administration of lidocaine
Paper drapes (optional)
Sterile gloves
Shavers
Safety pins- "16 uses"!

15. ORTHOPEDICS

SAM splints- both full-length and finger sizes (great for improvising just about anything)
Kendrick Traction Device- very light-weight design for femoral traction
Adhesive tape- for splinting ankles, fingers, etc.
Ensolite pads or Thermarest pads- no, you don't stick these in the medical kit, but they're mentioned here because they make excellent improvised universal knee immobilizers, ankle splints, C-collars, etc.
Ace wraps- essentially worthless for stabilizing 'blown-out' joints, however they serve an important function by providing compression; also useful for holding pressure over taped extremities (Coban works well too)
Fiberglass splints (optional) e.g. 3M "One Step"
Stiff Neck Extrication Collar (optional)- if room permits (this can be improvised)
"Air Casts" or "Gel casts"- light-weight splint for sprained ankles which allows near normal ambulation and can be used inside of boot

16. INJECTABLE MEDICATIONS

(Note: Always consider individual drug stability under environmental extremes)
Epinephrine 1:1000- a must!
Antibiotics- multiple alternatives here; these are included to provide temporizing treatment in the unlikely event of meningitis or an intraperitoneal infection, i.e. ruptured appendix or bowel perforation

a. **Primaxin** (imipenem-cilastatin sodium)
b. **Rocephin** (Note: these are very expensive medications; those on a tighter budget may want to consider good old-fashioned chloramphenicol)

Morphine Sulfate
Narcan- where there's MS, there's Narcan
Decadron- essential for HACE
Phenergan
Compazine
Benadryl
Valium- consider rectal administration if difficult IV access
Depo-Medrol- trigger point injection or severe tendonitis
Lidocaine 1-2%
Toradol IM (Ketorolac Tromethamine)- note: current problems with availability

17. IVs

Multiple sizes of **angiocaths** (16, 18, & 20G.)
Intraosseous needle
Blood solution administration sets
Microdrip administration sets
"Heparin" locks (with flush sets)
Bacteriostatic normal saline &/or heparin flushes
Anticoagulated blood collection bags (if group transfusion is considered)
IV fluids- crystalloid for volume resuscitation, e.g. normal saline or lactated ringers (possible role here for hypertonic saline); D5W (or any available IV fluid) for administration of medications (use Heparin lock when possible)

18. AIRWAY

Oral airways
Endotracheal tubes (7.5, 8.0) for blind nasotracheal intubations (note: some expeditions carry laryngoscopes; they're heavy and bulky--disposable plastic units may be an alternative)
V Vac suction device (optional)- somewhat bulky but very effective
Pocket mask
Medical oxygen tanks with regulators, nasal cannulas and 100% non-rebreathing m with reservoir

19. DENTAL KIT

Minimal kit includes:

- **Cavit** (no mix temporary filling)
- **Eugenol** (topical anesthetic, anti-inflammatory)
- **Dental floss**- multiple uses including reinforcing splint for avulsed teeth

More extensive kit to include:

- **Benzocaine in orobase** (topical analgesic)
- **IRM powder** for recementing crowns
- **Dental syringe** with **marcaine** ampules
- **Mouth mirror** and **explorer**
- **Filling instrument**
- **Probe**
- **Universal extractor**
- **Elevator**

20. MISCELLANEOUS MEDICAL

Hypothermia thermometer (Zeal subnormal)
Stethoscope (often ear to chest is adequate)
Snakebite kit (Sawyer extrator works best)
Condoms- for improvising one-way "Heimlich" valve for use with tube thoracostomy or chest tube (rubber glove works too, but is less exotic)
Paper and pencil- essential for communication
Headlamp- essential for any nightime operations or surgical procedures requiring extra lighting (e.g. dental procedures)
Urine Chemstrips (include leukocyte indicator)
Tongue blades
Sterile applicators
Foley catheter- 16 French with 30cc balloon, can be used as urinary catheter, improvised chest tube, posterior nasal pack
Water disinfection system (the choice is yours)
Fluorescent surveyors tape- marking helicopter landing zone, finding your way bac the victim (like Hansel and Gretel), etc.
Spare sunglasses (protective eyewear can also be improvised if necessary)
Optional: (note : rarely will these instruments change the initial impression or plan)

- **oto-opthalmoscope**
- **blood pressure cuff**

TRAVELER'S DIARRHEA

Howard Backer, M.D. 1991

(Adapted from Backer H. Infectious diarrhea from wilderness and foreign travel. In: Eds, Auerbach PA, Geehr EC. *Management of Wilderness and Environmental Emergencies*. Chapter 29, p 759-804, CV Mosby, 1989.)

North American travelers to underdeveloped countries are susceptible to infectious diarrhea. Virtually all travelers eventually will suffer an intestinal illness; most do not have to wait long. Traveler's diarrhea (TD) is a syndrome, not a specific disease, which may be caused by any pathogenic enteric microorganism. The spectrum of illness may vary as much as the etiology. Transmission is by fecal-oral contamination; water and food are the most common vehicles and the level of sanitation is the most important factor determining transmission. Aside from immune responses, natural barriers to enteric bacterial infections include gastric acid, normal gut motility, and normal bacterial flora.

Risk

Attack rates of illness are highest (20-50%) among people from the "industrialized or developed" countries in North America and northern Europe who travel to "undeveloped" countries in Africa, Asia, and South America. Intermediate risk occurs for short-term travelers to resorts in the Caribbean, northern Mediterranean, Pacific, and countries such as Japan and Israel. Susceptible travelers experience highest risk soon after arrival, with peak incidence of diarrhea on days 2-5. Previous travel to an area with high rates of traveler's diarrhea does not decrease risk of illness. Multiple episodes may occur on the same trip. Attack rates remain high for up to one year, then decrease, but not to levels of local inhabitants. Complete immunity is unusual for most infections.

Etiology

Enteric bacteria cause most cases of diarrhea. *E. coli* bacteria that produces an enterotoxin (ETEC) account for 25-75% of illnesses; Shigella and *Campylobacter* bacteria are responsible for 10-20%. A few other bacteria (*Salmonella, Vibrio cholera, Vibrio parahemolyticus*) cause sporadic cases. Viruses may be responsible for 5-10% of cases. Protozoa, mainly *Giardia lamblia* and *Entamoeba coli* account for 2% each--but in certain places this can be much higher. 10-20% of cases are mixed infections. In 25-45% of cases, an etiologic agent cannot be indentified, even in the best laboratories. The list changes as advances in laboratory techniques "discover" new enteric pathogens. One such protozoan organism, *Cryptosporidium*, is the apparent cause of diarrhea in some immunocompetent travelers; but the oocysts are commonly found in stool samples from around the world and serologic evidence of past or recent infection is more common, even in asymptomatic persons.

The pathogenicity of some organisms, such as *Blastocystis hominis*, is still being debated.

Clinical syndrome

The typical clinical syndrome experienced by travelers resulting from infection with ETEC (and many of the other causes) begins abruptly with watery diarrhea and abdominal cramping. Most cases are mild, consisting of 2-4 episodes of diarrhea per day with cramping but no other symptoms. Another 30% experience moderate illness with malaise, nausea, and vomiting accompanying the diarrhea. Only 10-20% experience severe illness with more than 4 stools per day, malaise, myalgias, nausea, vomiting, chills, and fever. While the average duration is 3 days, half of cases resolve within 48 hours, 10% last longer than one week, and 1-2% last one month or longer.

5-10% of victims describe dysenteric symptoms with fever and bloody stools. Fever is common, as well as lower abdominal cramps and tenesmus (rectal urgency). Stools are typically liquid and small volume, containing gross blood and leukocytes. An extensive study of "several hundred thousand" Swiss travelers found no reported deaths from TD. Less than 1% were admitted to a local hospital while traveling, and only 4% consulted a local physician. However, 30-40% were confined to bed or needed to alter their travel plans because of illness.

Epidemiology

The relative importance of food and water is difficult to determine for traveler's diarrhea. However, several investigations have shown high rates and amounts of enteric pathogens in foods tested from markets in Asia and Mexico. Some infections (i.e., *Salmonella* and *Campylobacter*) are definitely associated with meats. Diseases with a small infectious dose (shigellosis, amebiasis, giardiasis, and ascariasis) may also be spread by direct person-to-person contact. Prevention of enteric infections includes adequate hand washing and personal hygiene.

Risk of illness appears to be lowest when the majority of meals are cooked by oneself, intermediate when eaten in private homes or restuarants, and highest from street vendors. Guests staying at four-star hotels have only sightly reduced risk. The cleanliness of the food is no better than that of the hands preparing it.

Except in one instance, most studies evaluating risk have found little correlation between routine precautions and illness. Thus, the following standard dietary recommendations for prevention are based more on known potential vehicles for transmission of illness than strong epidemiologic evidence.

Safe: Bottled and carbonated drinks; commercially bottled beer and wine; boiled or otherwise disinfected water.

Unsafe: tap water and ice made from untreated water; bottled water that is not carbonated should be suspect.

Comment: Bottled carbonated beverages are considered safe due to antibacterial effects of the low acidity. Home-made beverages

cannot be guaranteed. Most enteric organisms can survive freezing and melting in common drinks, so ice is not considered safe unless made from treated water. (Alcohol in mixed drinks does not disinfect.) Ice in block form is often handled with unsanitary methods.

Safe: Pateurized dairy products

Unsafe: unpasteurized dairy products.

Comment: These may be the source of infection with *Salmonella, Campylobacter, Brucella, Listeria monocytogenes, Mycobacterium tuberculosis*, and others.

Safe: peeled fruits and vegetables, and cooked food.

Unsafe: raw vegetables, salads, meats and seafood.

Comment: Raw vegetables in salads may be contaminated by fertilization with human waste or by washing in contaminated water. Anything that can be peeled or have the surface removed is safe. Fruits and leafy vegetables can also be disinfected by immersion and washing in iodinated water or by contact with boiling water for 30 seconds.

Raw sea food has been associated with increased risk of diarrheal illness in travelers and is suspected to be the cause of *Vibrio parahaemolyticus* infection in Asiatic travelers. Shellfish concentrate enteric organisms from contaminated water and can carry hepatitis A, Norwalk virus, *Aeromonas hydrophila, Yersinia enterocolitica, Vibrio cholera*, and *Vibrio parahaemolyticus*. Raw fish can carry parasites such as *Anisakis simplex, Clonorchis sinensis*, and *Metagonimus yokogawai*. Raw crustacea are the source of *Paragonimus westermani*. Raw meat is the source of *Salmonella, Campylobacter, Trichinella, Taenia saginata* and *T. salium* (beef or pork tapeworm), and *Sarcocystis*. Adequate cooking will kill all microorganisms and parasites. However, if food is left at room temperature and contaminated prior to serving, it can incubate *Salmonella, E. coli*, or *Shigella*.

Prophylactic medication for prevention of TD

Vaccines are available for typhoid and cholera, but these offer only limited protection. Vaccines for *Shigella*, ETEC and rotavirus are being developed with strong economic and public health incentive.

10-25% of European travelers to high risk areas and one third of U.S. travelers to Mexico take prophylactic medication to prevent TD. In one survey, 42% took prophylactic or therapeutic intestinal drugs, and 22% took more than one agent.

Lactobacilli was shown in one study to provide a 40% protection rate, but other studies have not substantiated this. Antimotility drugs, such as diphenoxylate, have adverse effects when used for prophylaxis.

Hydroxyquinolones, mainly iodochlorhydroxyquin or clioquinol (available commercially as Entero-vioform) are widely available in many parts of the world. While they are primarily useful as amebicides, they have been widely used for prevention and treatment of TD. This medication is not recommended because of its association in Japan with subacute myelooptic neuropathy leading to

blindness.

Of the non-antibiotic drugs, only bismuth subsalicylate (BSS) has been shown by controlled studies to offer reasonable protection (40-70%) and safety. The currently recommended dose is 2 tablets four times a day (2.1 g/d). The precise mechanism by which BSS prevents diarrhea is not known. Salicylate has some antisecretory effect, and bismuth products have some antimicrobial effect.

Mild side effects include constipation, nausea, blackened tongues or stools. Although it is not known if this salicylate cross reacts with aspirin, BSS should not be used by someone with a history of aspirin allergy. Caution is recommended in small children, patients with gout, renal insufficiency or those taking anticoagulants, probenecid, methotrexate, and other aspirin containing products. Although levels of bismuth have been detected after 2-3 weeks of BSS use in travelers, these were well below the toxic range. BSS concurrent with doxycyline should be avoided, since it appears to chelate this antibiotic, and may decrease absorption.

Antimicrobial prophylaxis of travelers diarrhea

Several antimicrobial agents have been shown highly effective (80-90%) in preventing travelers' diarrhea when given over short periods.

Currently, the most experience has been obtained with doxycycline 100 mg/day and trimethoprim 160 mg with sulfamethoxazole 800 mg (TMP-SMZ) once daily. Both prevented diarrhea from susceptible strains of ETEC and *Shigella*, but protection was higher than could be explained on the basis of these organisms alone, suggesting that other susceptible organisms may be found to cause susceptible diarrhea. With both, the effect lasted only as long as the drug was continued. Subjects who remain in a high risk area, experience an increased incidence of diarrhea during the week following cessation of prophylaxis. In areas with antibiotic-resistant ETEC, protection was considerably less (60-70%).

Several recent studies have shown very good protection rates (90-95%) using quinolone-carboxylic acid derivatives, norfloxacin (400 mg) or ciprofloxacin (500 mg) taken prophylactically *in a single daily dose for one week.*

Despite dramatic protection against diarrhea, investigators do not recommend widespread use of these medications by travelers. Several reasons are cited:

1) Side effects

Doxycycline caused no major problems in these studies, but some persons experienced gastrointestinal side effects. Photosensitivity is potentially a serious problem. Pregnant women and children should not use doxycycline for weeks at a time due to potential for teeth staining. TMP-SMX caused rash requiring discontinuation of drug in 12% of subjects. With larger numbers of people using these drugs, more serious side effects (e.g., Stevens-Johnson syndrome and hemolytic or aplastic anemia) might cause a higher risk of morbidity from prophylaxis than from illness. The

quinolones have few serious side effects, but should not be used in pregnant women or children.

2) Selection of drug resistant bacteria.

Emergence of doxycycline and trimethoprim-resistant ETEC has occurred during prophylactic use. Multiply resistant stains of ETEC and shigella are becoming common. However, it is noteworthy that a recent study in Mexico demonstrated the acquisition of antibiotic resistant *E. coli* in feces of travelers from the U.S. without prophylactic or therapeutic antibiotic use, indicating that resistant strains are very common in underdeveloped parts of the world and travelers become colonized with these strains within a few weeks (not all had diarrhea).

Hopes that norfloxacin and ciprofloxacin, may not promote rapid development of resistant strains may be premature; plasmid-mediated resistance to nalidixic acid in *Shigella dysenteriae* has now been reported.

3) Alteration of normal bacterial flora

Vaginal candidiasis and gastrointestinal side effects, including diarrhea, are common with antibiotic therapy. Severe colitis caused by colonic overgrowth with *Clostridia difficile* has occurred following therapy with many antibiotics, including erythromycin and TMP-SMX. Studies using norfloxacin and ciprofloxacin have shown eradication of most gram negative aerobic gut bacteria during treatment that returns to normal within six weeks. So far no deleterious results have been noted from this dramatic alteration in intestinal flora.

4) Decreased prevention

Travelers taking antibiotics may relax their vigilance for dietary precautions and increase their risk of acquiring other infections, such as hepatitis A or typhoid fever.

Although there is consensus that antibiotic prophylaxis should not be used by all travelers, there is no agreement as to who should use them. The potential candidate would be a resident of a low risk country going to a high risk area for a few weeks who cannot accept the potential disruption of traveler's diarrhea, who may not be able to take reasonable dietary precautions and understands the limitations and potential side effects of antimicrobial prophylaxis (e.g. politicians, business and consultant travelers, athletes, military personnel). Underlying health should be another consideration: those who could not tolerate dehydration and electrolyte loss (patients on diuretic or digoxin therapy, or with renal insufficiency); those at higher risk of infection (patients with achlorhydria, gastric resection or antacid use); patients with inflammatory bowel disease, immunodeficiency, or with a past history of frequent severe TD.

Evaluation and Laboratory Use

In the field, evaluation of the history, clinical exam findings and gross appearance of the stool are sufficient to determine fluid needs and to decide who should receive presumptive antibiotic therapy. In the clinic or hospital, judicious use of laboratory and diagnostic tests may allow a definitive diagnosis,

however, these tests are not necessary in the majority of patients with acute infectious diarrhea.

Exam. Most important is assessment of the patient's general condition. The level of hydration should be clinically evaluated by vital signs with orthostatic pulse and blood pressure, mental status, skin turgor, mucous membranes and urine output. The abdominal exam often shows mild tenderness, but should not demonstrate signs of peritoneal irritation.

Diagnosis by clinical means. Studies have attempted to correlate stool appearance with the presence of bacterial or invasive infection, indicating which patients should be cultured and started on antibiotics. Grossly bloody stools did not correlate as well as the presence of fecal leukocytes. Large number (>25) of polymorphonuclear leukocytes (PMNL) per high power field (hpf) correlate significantly with invasive *Shigella* and *E. histolytica* infection. The test can be simply done in an outpatient clinic setting on a fresh stool sample. A fleck of mucous, or liquid stool is mixed on a slide with a drop of methylene blue and observed under a microscope. Leukocytes are easily seen, although they can be confused with protozoal cysts.

Laboratory. Specific diagnosis of bacterial infection is made by stool culture. However, relatively few pathogens are identified by routine stool testing. It is generally agreed that only one culture is necessary, although this does not pick up all infections.

Diagnosis of protozoan infections is usually made by examination of the stool for cysts and trophozoites. Intermittent shedding and scant numbers of organisms in the stool may make diagnosis difficult. Microscopy of one stool sample identifies only 50% of infections, so three stools at 24-48 hour intervals are considered standard. If these are negative, and suspicion is high, three more are indicated.

Special diagnostic procedures. When dysentery is present, sigmoidoscopy or colonoscopy is the best method for evaluating colonic lesions and collecting samples for culture and microscopy. Barium studies may show nonspecific signs of enteritis or colitis, but are not diagnostic for enteric infection. Blood counts and electrolytes are seldom helpful, except when complications such as anemia, severe dehydration, or underlying disease are present. Common enteric protozoa do not cause eosinophilia. Antibody-specific serologic tests are now widely used for invasive amebiasis, and will become available for other infections in the future.

TREATMENT

Even among the potentially severe infections, almost all are self-limited, requiring nonspecific treatment to prevent dehydration. Culture results are not conclusive for at least 24-48 hours. By this time most patients have begun treatment or spontaneously improved, so therapy is frequently not changed by the culture results. Invasive organisms causing dysentery and systemic toxicity should be empirically treated with antibiotics in the field. If therapy does not lead to rapid improvement, diagnostic

measures are important.

Fluid and dietary treatment

The most significant advance in the past 25 years in the treatment of diarrheal illness has been oral rehydration. Two observations account for the successful use of ORS even with high fluid losses from cholera:

1) glucose enhances absorption of sodium through the intestinal mucosa (other electrolytes are absorbed non-selectively);

2) absorption remains intact despite the presence of enterotoxin and concurrent secretory losses of water and electrolytes.

Watery diarrhea, often caused by production of an enterotoxin, has electrolyte composition similar to plasma, varying somewhat with the type of infection and age of the patient. The formula packaged and promoted by the World Health Organization and UNICEF contains powder to be mixed with one liter of disinfected water: sodium 90 mEq, potassium 20 mEq, chloride 80 mEq, bicarbonate (or trisodium citrate) 30mEq, glucose 111 mmol (2%). If balanced electrolyte salts are not available, similar solutions, can be made from common ingredients.

Fruit juice, coconut milk, simple sugar and salt solutions, or diluted cola drinks are fine for mild dehydration, partial maintainance, supplementation, or if nothing else is available. However, for moderate losses, these do not contain correct electrolyte balance and osmolality. Many glucose electrolyte solutions sold commercially are not ideal for replacement of diarrheal losses: electrolyte contents are often insufficient and high concentrations of glucose may increase diarrheal fluid loss.

ORS does not prolong duration or increase rate of diarrhea compared to intravenous replacement. Repair of fluid deficit is accomplished within 12 hours in 90% of patients.

ORS works well for mild to moderate (5-10%) dehydration. Severe dehyration or shock, obtundation or seizures should be treated first with intravenous fluids with ORS instituted as soon as possible. Protracted vomiting or gastric distention precludes initial use of ORS. However, a few episodes of vomiting during early treatment should not discourage oral therapy. Nasogastic administration of ORS is recommended when intravenous fluids are not available or the patient will not drink enough to replace fluid losses. 90% of patients can be treated with ORS alone. Failure rates are as low as 1%, most often due to stool losses exceeding oral intake.

Most adults and children can ingest ORS *ad libidum*, with extra water or juices provided in addition. In travelers diarrhea, adults requirements will probably be only 3-4 liters in 24 hours. Infants and small children may be given one part water, half strength milk formula, or juice to each two parts ORS.

Adults and children should be discouraged from fasting. Supplemental nutrition is beneficial and can be given as soon as fluid deficit losses are replaced, usually after the first 4 hours.

Breast feeding of infants should be resumed as soon as possible. Except for an occasional patient with carbohydrate intolerance, staple foods such as cereals, bananas, lentils, potatoes, and other cooked vegetables are well tolerated and can be continued during diarrhea. Food high in starch may even decrease diarrhea. Only food and drink that prolong diarrhea or increase intestinal motility should be avoided. These contain lactose, caffeine, alcohol, high-fiber, and fats.

Recent research has centered on food-based oral rehydration salt solution. The electrolyte concentration is the same as standard ORS, but the glucose is replaced with 50-60 gm of cereal powder (rice appears best, but other starches can be used). The starches are digested in the intestinal lumen, generating glucose gradually, without causing an excessive osmolar load that pulls fluid into the gut. Compared to glucose-based formulas, cereal-based formulas decrease diarrhea substantially, so cereal formulas rehydrate more efficiently for a given intake volume. The additional calories and amino acids is an obvious nutritional benefit. Furthermore, these cereals are already a dietary staple in the underedeveloped world so, they are well accepted by the local people. And finally, since the cereal formula is brought to a boil during preparation, the water is disinfected.

Nonspecific therapy

Therapies that alter intestinal flora (lactobacillus) and adsorbents (Kaopectate and activated charcoal--which bind nonspecifically to water and other intraluminal material), are harmless but do not alter the course of illness.

Antimotility agents (narcotic analogues related to opiates: diphenoxylate-Lomotil®, loperamide-Imodium®, tincture of opium, paregoric, codeine) are the most widely prescribed drugs for diarrhea, but there is debate concerning their safety and benefit.

These agents reduce the number of stools and relieve cramps, in illness caused by ETEC and nondysenteric shigellosis. Side effects are infrequent and include mild drowsiness, dizziness, and constipation. There is little evidence that these agents prolong or worsen clinical illness. They may be used in combination with antibiotics (see below).

Currently, narcotic antimotility agents are recommended for symptomatic relief in watery diarrhea, but <u>should be avoided when high fever or dysentery are present</u>. Dosage should be limited to 3-4 doses in one 24 hour period, used only after a loose bowel movement. They should not be given to children under the ages of 2 years due to the danger of central nervous system depression.

Antisecretory therapy is appealing, since increased secretion of water and electrolytes is the major physiologic derangement in acute watery diarrhea. Although aspirin and other nonsteroidal antiinflammatories have been found to inhibit secretion, their usefulness is still limited.

Bismuth subsalicyclate (BSS) is the best studied non-antibiotic drug. When was used to treat TD (30 ml or two tablets every half hour up to a total of eight doses), the number of stools

(but not total weight or water) and subjective complaints of cramping were decreased for at least 48 hours. Taken on two successive days, this same dose decreased the number of stools by 50%. The effect was mainly noted for infections caused by ETEC, with minimal effect in nondysenteric shigellosis. Similar improvement in stool frequency, consistency and diarrhea duration were noted with 1050 mg liquid hourly to a total of four doses in two days. BSS in such high doses should not be given to children, and the same precautions apply for treatment as noted for prophylactic use.

Antimicrobial treatment

Antibiotic treatment with TMP-SXZ or trimethoprim alone can markedly reduce the symptoms and duration of illness in ETEC and shigellosis. Flouroquinolones (norfloxacin and ciprofloxacin) are now becoming the drug of choice in suspected or proven bacterial enteritis, although TMP-SXZ has the advantage of cost and widespread availabilibty. Ciprofloxacin 500 mg bid was equally effective in treating TD compared to TMP-SXZ. It appeared to decrease the duration of fever and diarrhea in campylobacteriosis and salmonellosis as well as shigellosis. Potential advantages include a high degree of in vitro activity against all etiologic agents tested (including *Campylobacter*) and the potential for less bacterial resistance.

If a flouroquinolone is not available or is contraindicated, sulfa-allergic patients can use a trimethoprim 200 mg bid or doxycycline 100 mg bid. Ciprofloxacin is not currently recommended in young children due to drug-related arthropathy in laboratory animals. Small children can also be treated with TMP 4mg/kg/day and SMX 20 mg/kg/day in 2 divided doses.

Recent studies on the treatment of traveler's diarrhea compared an antimotility agent (loperamide) with an antibiotic (TMP-SXZ), each given alone or together: the combination was significantly more effective for resolving diarrhea than antibiotics alone. Loperamide is more effective in decreasing symptoms in the first 24 hours, after which antibiotics are more effective in shortening illness. Antibiotics are much more effective in dysentery.

Treatment Recommendations.

Travelers should carry an antimotility agent (loperamide or diphenoxylate) as well as an antibiotic: TMP-SMX, ciprofloxacin, norfloxacin or nalidixic acid (the latter is much less expensive than the newer derivatives). Mild diarrhea requires no treatment, or can be treated with antimotility agents or BSS. In the event of moderately severe illness (more than 6 loose stools in 24 hours or 3 stools in 8 hours associated with other symptoms) or when symptomatic therapy has failed after 48 hours, begin treatment with both an antimotility agent and an antibiotic. BSS is a reasonable alternative. When fever and signs of dysentery are present, begin antibiotics; antimotility agents should be avoided or used with caution. Minimum antibiotic doses and durations have not been

determined. Three days are recommended, but two or less may be sufficient.

Antibiotics will not be effective against diarrhea caused by viruses, *Vibric parahaemolyticus*, *G. lamblia*, *E. histolytica*, and other protozoan, parastic, or non-infectious causes. They have not been shown to shorten the illness associated with uncomplicated *Campylobacter* or *Salmonella* enteritis, although ciprofloxacin may prove to be clinically effective in these illnesses. Therefore, antibiotics should not be continued in the face of persistent or worsening diarrhea.

Giardia is more commonly isolated from stools if diarrhea has persisted more than one or two weeks. When diagnostic facilities are not available, it is reasonable to treat persistent diarrhea that has not responded to antibiotics with metronidazole 250 mg tid for 7-10 days or tinidazole 2 gm in a single dose for 1-3 days. This is effective treatment for giardiasis and at least partial treatment for invasive amebiasis.

Chronic diarrhea in travelers.

Persistent diarrhea in the returned traveler can be a difficult problem. Most of these patients have already tried a course of antibiotic therapy. Several causes must be considered:

1) Persistent infection with a bacterial or protozoal pathogen or an infection with a parasitic roundworm, cestode or nematode. Diarrhea may be a prominent symptom in schistosomiasis, *P falciparum* malaria, leishmaniasis, and African trypanisomiasis.

2) Delayed resolution and malabsorption: Following eradication of microbial pathogens, bowel habits may not return to normal for several weeks. Postinfective malabsorption can persist for weeks to months after acute diarrhea. Finally, acute diarrheal illness may induce temporary or permanent lactose intolerance, characterized by abdominal pain, flatulence, and diarrhea after ingestion of lactose-containing foods.

3) Tropical jejunitis: Also known as tropical sprue, this malabsorption syndrome presents as chronic diarrhea, fatigue, cramps, and weight loss. Onset usually follows an episode of acute enteritis. It is associated with level of hygiene and length of stay, thus is not common in short term travelers.

4) Pre-existing condition: An underlying condition such as inflammatory bowel disease, irritable bowel syndrome, or celiac sprue may become more symptomatic following an episode of acute enteritis.

The approach to chronic diarrhea in travelers should begin with stool culture for all common enteric pathogens, as well as for *Clostridium difficile*. Three stools should be examined for ova and parasites. If all tests to this point are negative, and stools contain leukocytes, sigmoidoscopy and barium enema may be performed, along with empiric treatment for shigellosis. If there are no leukocytes, duodenal mucous is examined for *G. lamblia*, followed by empiric treatment. Dietary modification in all cases should include avoidance of lactose. The next step is to perform tests for malabsorption and biopsy the small bowel mucosa.

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ENVIRONMENTAL HEAT ILLNESS

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JULY, 1991

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CASE PRESENTATION

George is a 49-year-old executive who has driven from Atlanta, Georgia along with three of his friends to raft the Chattooga River in Northern Georgia. They had heard that this was the river where the movie "Deliverance" had been filmed. George had played lineman for the University of Georgia and has maintained a hard-driving attitude in his business. He recently had an EKG stress test which was suggestive of coronary artery disease and he has been advised to see a cardiologist. His past medical history is significant for hypertension. Socially, his wife is filing for divorce because he drinks too much, works most of the time, and prefers to spend whatever free time he has "with the guys." He takes Inderal for his hypertension, Elavil for depression, and Actifed for hay fever. George and his friends arrive at the rafting company at 9 a.m., having already consumed three six-packs of their favorite beverage, "Bud", and are eager to "ride the river." The temperature is 82 degrees Fahrenheit and the relative humidity is 90%.

QUESTIONS TO PONDER

1. What are the environmental factors which predispose someone to heat illness?

2. How does the body compensate for these factors?

3. Can the relative risk of developing heat illness be objectively measured?

4. Does George have any pre-existing medical problems that increase his risk for developing heat illness?

5. Do any of George's medications predispose him to heat stroke?

6. Why is George's raft guide about to have a bad day?

MECHANISMS FOR ACCUMULATING HEAT

1. Basal metabolism alone can create a heat load of 65 to 85 KCAL/hr, which would raise the body temperature 1.1 degrees Centigrade/hr if mechanisms for dissipating heat did not exist.

2. Moderate work could increase this temperature rise by 500% (300-600 KCAL/hr).

3. An individual in bright sun may gain 150 KCAL/hr.

4. Prolonged exposure in hot tubs, saunas, and steam rooms can raise body temperature.

5. Elevated body temperature in turn imposes its own intrinsic additional heat load since cellular metabolism will increase by 13% for every 1-degree Centigrade rise in body temperature.

6. Studies done in runners have shown that dehydration alone is capable of elevating body temperatures. This is probably due to an increased activity of the cellular sodium pump, which accounts for 20-45% of the basal metabolic rate (BMR).

7. When the air temperature is greater than the body temperature, radiant heat gain is possible.

MECHANISMS FOR DISSIPATING HEAT

1. Radiation, the transferring of heat from the body to a cooler environment, accounts for 65% of cooling as long as the air temperature is lower than the body temperature.

2. Normally, 30% of cooling results from evaporation of sweat. For each 1.7 ml of sweat evaporated, the body loses 1 KCAL of heat. When the ambient temperature approaches 95 degrees Fahrenheit, evaporation prevails as the only mechanism that the body has to dissipate heat. If the humidity level should exceed 75%, evaporative heat loss potential will decrease. Sweat that drips from the skin only exacerbates dehydration without providing any cooling benefit.

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- The combination of high temperature and high humidity blocks the two main mechanisms that the body has to dissipate heat. The conditions are right for "heat stroke."

PHYSIOLOGICAL RESPONSES TO HEAT

1. Cutaneous blood vessels dilate to increase the surface cooling area.

2. To maintain blood pressure, in the face of this greatly decreased peripheral vascular resistance, cardiac output may double or quadruple, placing a strenuous load on the heart.

3. There is a compensatory vasoconstriction in the splanchnic vessels.

4. Sweat volume will increase.

5. Acclimatization to heat.

    a. In contrast to hypothermia, physiologic acclimatization to heat is possible. It usually takes one 8-11 days to reach maximum benefit and requires some degree of exercise (at least 1 1/2 to 2 hours) each day.

    b. The mechanisms, although poorly understood, seem to be mediated through activation of the Renin-Angiotensin system with increased production of aldosterone. This results in sodium conservation in both urine and sweat with concomitant losses of potassium.

    c. Sweating is initiated at lower core temperatures and the amount of sweating may more than double.

    d. Three cardiovascular adaptations which result in enhanced delivery of heated blood from the core to the surface have been shown to occur: (1) An increased cardiac output; (2) A decreased peak heart rate; and, (3) An increased stroke volume.

    e. There occurs a marked increase in the density of mitochondria per unit muscle mass. This allows increased potential for oxygen utilization.

INDIVIDUAL CHARACTERISTICS WHICH PREDISPOSE SOMEONE TO HEAT

1. The elderly are less able to increase cardiac output for heat dissipation and are often dehydrated. Intrinsic diseases of the heart such as CAD, CHF, or previous MI would limit the ability to compensate for peripheral vasodilatation.

2. Neonates lack thermoregulatory and sweating capabilities.

3. Obese individuals have more insulation and less surface area-to-volume ratio with which to dissipate heat.

4. Hyperthyroidism can markedly increase metabolic rate with a rise in endogenous heat production.

5. Dermatologic disorders, as well as burns affecting large surface areas, may limit heat dissipation by sweating.

6. Various medications and drugs may predispose one to environmental heat illness.

   a. Beta-blockers will inhibit compensatory increases in cardiac output.

   b. Amphetamines, PCP, cocaine, and other stimulants can increase muscular activity with a resultant increased endogenous heat load. Amphetamines and LSD also act directly on the hypothalamus to produce elevated temperatures.

   c. Anticholinergics such as phenothiazines, lithium, tricyclic antidepressants, antihistamines, and antispasmodics reduce sweating and can disrupt hypothalamic function.

   d. Diuretics may produce dehydration.

HEAT EXHAUSTION VERSUS HEAT STROKE
(See Table I)

Heat exhaustion and heat stroke are often discussed separately, implying that they are two different and distinct pathophysiologic entities. This is misleading because, with few exceptions, they define a continuum of one disease process. Heat exhaustion

often presents with flu-like symptoms, including malaise, headache, anorexia, nausea, vomiting, and muscle cramps. Core temperatures are usually less than 41 degrees Centigrade (106 degrees Fahrenheit) and often are normal. Dehydration is almost always manifest. Clinical signs may include orthostatic hypotension, tachycardia, diaphoresis, and moderate pyrexia. Dehydration, hypokalemia, and relative hypoglycemia have all been implicated as precipitating factors in the development of heat stroke. It represents late stages of heat exhaustion as compensatory mechanisms for dissipating heat are failing. Decreased sweating due to dehydration and high-output cardiac failure contribute to a decompensating system for heat dissipation. Core temperatures may rise rapidly, producing cellular damage. A key point in differentiating heat exhaustion from heat stroke is that CNS function remains essentially intact in heat exhaustion. CNS dysfunction, such as delirium, ataxia, seizures and coma, would suggest heat stroke and mandate aggressive cooling measures. The exact temperatures at which cellular damage starts to occur is not clear, but oxidative phosphorylation becomes uncoupled at temperatures above 42 degrees Centigrade (107.6 degrees Fahrenheit). The resultant damage is both a function of the high temperature as well as the exposure time. Patients with higher temperatures for shorter periods may do better than those individuals who maintain more moderate temperatures for longer periods. Dry, hot skin is not mandatory to make the diagnosis of heat stroke. In one study of military recruits, 50% of the patients with heat stroke maintained their ability to sweat. Anhidrosis may be a late manifestation as a result of profound dehydration and necrotic plugging of sweat gland ductules. Patients with heat stroke almost always manifest signs of tachypnea, hypotension and sinus tachycardia. The cerebellum is most sensitive to heat and ataxia may be an early clue.

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- Heat exhaustion and heat stroke are probably a continuum of heat disorders rather than distinct pathophysiologic entities. In the setting of heat illness, patients with CNS dysfunction should be treated for heat stroke.

- Sweating may still be present in heat stroke.

COMPLICATIONS OF HEAT STROKE

1. Decreased renal perfusion can lead to acute tubular necrosis and renal failure.

2. Damage to muscle and rhabdomyolysis can produce myoglobinuria and exacerbate the nephropathy.

3. Hypoglycemia and hypocalcemia may occur.

4. Although hyperkalemia may be seen initially, total body potassium is usually decreased.

5. Markedly elevated liver enzymes are often seen, suggesting hepatocellular injury. SGOT, SGPT, and LDH values may be in the tens of thousands after 24 hours. Elevations to several thousand are often found even in some patients with heat exhaustion.

6. Bleeding, secondary to a consumptive coagulopathy, may occur. The precipitating factor is thermal damage to vessel endothelial cells with exposure Type III basement membrane collagen. A syndrome of DIC may occur at 1-3 days after onset of heat stroke.

7. The only organ not reported to be directly damaged in patients suffering from heat stroke is the pancreas.

TREATMENT OF HEAT EXHAUSTION

1. Fluids, rest in a cool environment, glucose and conservative measures of cooling are essential treatment modalities. Urine should be monitored for rhabdomyolysis and a thorough neurological exam should be performed.

TREATMENT OF HEAT STROKE

Pre-Hospital Care:

1. Begin cooling immediately at the scene. Remove clothing, spray any available liquid on the patient and fan to promote evaporative cooling. If nothing else is available, even urine can be used as a liquid medium. The fluid does not need to be cold to produce evaporative cooling.

2. Ice bags, chemical ice packs, or cold compresses should be placed in areas where large blood vessels come near the surface such as the neck, axilla, groin, and scalp. Studies using this technique have documented cooling rates of .1 degree Centigrade/minute (5 times that of controls). This technique avoids generalized cutaneous vasoconstriction and shivering.

3. Nothing should be given orally.

4. Aspirin and Tylenol are not effective, and aspirin is contraindicated because of its effect on platelets and clotting. The hypothalamic set-point is not elevated as it is in fever.

5. Patients should be transported as quickly as possible to a medical facility.

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- Heat stroke should be considered a life threatening emergency. Several studies have shown mortality rates from 30 to 80%.

Emergency Room Care: The optimum method for cooling is controversial. Some large centers still use ice baths while others rely more on evaporative techniques. The different methods, along with experimental data on cooling rates, have been listed in Table II.

If the emergency facility is air-conditioned or in a non-humid environment, I prefer the evaporative techniques for cooling. The patient is fully undressed and sprayed with warm water to keep the skin temperature at approximately 32 degrees Centigrade. Fans are used to maximize evaporation. Ice packs can be simultaneously placed along the neck, axilla, and groin. This technique is safe and effective, and requires a minimum of preparation. It is also more practical for managing monitors, I.V.'s, endotracheal tubes and complications that might occur. In addition, it minimizes avoidance behavior and shivering. Most patients can be cooled to temperatures of 101 to 102 degrees Fahrenheit in under 40 minutes using either the ice bath or evaporative techniques. Hyperthermia may recur due to thermoregulatory instability, requiring additional cooling at a later time. Cooling should be discontinued when the temperature falls below 102 degrees Fahrenheit in order to prevent hypothermia from occurring.

Administration of I.V. fluids, consisting of D51/2 normal saline or lactated Ringer's solution, should be guided by urine output, central venous or wedge pressures, and blood pressure. Initial laboratory work should include a blood gas, CBC, electrolytes, BUN, creatinine, liver enzymes, CPK, calcium, platelet count, PT, PTT, and FDP.

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- Heat stroke victims should be cooled as rapidly as possible. The more rapid the cooling, the lower the mortality.

TABLE I

	HEAT EXHAUSTION	HEAT STROKE
SYMPTOMS	Flu-like symptoms (Headache, vomiting, muscle cramps, anorexia)	Same
SWEATING	PRESENT	MAY BE PRESENT OR ABSENT
CNS SIGNS	CNS function unimpaired	CNS Dysfunction: 1. Bizarre behavior 2. Confusion, delirium 3. Ataxia 4. Seizure 5. Coma
TEMPERATURE	Usually less than 41 degrees Centigrade (106 degrees Fahrenheit), often normal	Usually 41 degrees Centigrade or greater

TABLE II

EXPERIMENTAL DATA ON COOLING

Method	Cooling Rate	Advantages	Disadvantages
PERITONEAL LAVAGE (Dialysate at 6-10° C)	0.56° C/Min	FAST!	1. Time consuming to set up. 2. Inherent complications
EVAPORATIVE (15° H_2O spray .4 M/Sec Fan) 45° C Air	0.31° C/Min	1. Less shivering 2. Less vasocontriction 3. Easier to monitor	1. Requires non-humid environment.
ICE WATER BATH	0.1-0.25° C/Min	1. Effective in humid environment.	1. Tub not available 2. Management difficult 3. Shivering 4. Vasoconstriction
ICED GASTRIC LAVAGE (Iced H_2O at 200 Ml/Min	0.2° C/Min	1. Easy and rapid	1. Aspiration
ICE PACKS TO (Neck, Axilla, Groin)	.1° C/Min	1. Easy and rapid	----
COLD INHALED AIR BY IPPB	0.02° C/Min		

WHITEWATER MEDICINE

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INTRODUCTION

Whitewater rafting, kayaking, and canoeing have attracted a growing number of participants in the past ten years. With the present level of 200,000 whitewater enthusiasts growing by an estimated 15% each year, it is not surprising that there has also been an alarming increase in related accidents and deaths.

This article will review both the inherent risks manifest in whitewater sports and its medical ramifications. Recommendations for prevention and treatment will be discussed.

HISTORICAL OVERVIEW

Whitewater boating as recreation began in the late 19th Century, when early guides attempted to emulate Major Wesley Powell's Colorado River expedition by rowing wooden boats down many of the West's large rivers. Inflatable rafts became popular immediately after the Second World War when military-surplus neoprene assault boats and life rafts became available for civilian use. Less than twenty years later, while John F. Kennedy was running the nation, his brother Bobby was running a raft down the Colorado River. At that time, fewer than 500 people per year ran the Colorado River through the Grand Canyon. Now, more than 500 people a day travel the same route.

Rafting did not become popular in the Eastern part of the United States until the early 1960s. In 1968, commercially-guided raft trips were offered for the first time on the New River in West Virginia, while the Chattooga River in Georgia attracted many rafters after the movie Deliverance was filmed there in 1971.

Today, even mountaineering and rock-climbing legends such as Yvon Chouinard and Royal Robbins spend much of their time performing first descents on rivers rather than first ascents on rock faces.

River running has been revolutionized by vast improvements in equipment. Electronically-welded plastic is replacing rubber as the primary material employed in raft construction--making rafts lighter, stronger, and more easily repaired. The advent of the light and maneuverable self-bailing raft has made it common for rafters to run rivers that prior to 1983 had been considered too difficult and dangerous. This greater mobility has been paralleled by a marked increase in the number of accidents far away from modern medical care.

A major innovation in kayaking was the invention of the plastic kayak, first manufactured by the Holloform Company in 1972. Today it is rare to see anything but plastic kayaks except at races, where fiberglass and kevlar boats are still popular. The advent of plastic boats has resulted in a substantial increase in serious injuries and deaths caused by vertical pinning, broaching, and entrapment.

When a kayaker plunges over a drop, the front end of the boat can become trapped between rocks beneath the surface; this results in what is called a vertical pin. Instead of breaking away as

fiberglass boats tended to do in the past, plastic kayaks remain steadfast--or, worse, fold over on themselves, pinning the occupant upside down underwater.

The term broaching refers to the process whereby the unrelenting force of water traps a craft against an obstacle such as a boulder. This can cause a plastic kayak to fold or its front deck to collapse, trapping the paddler's legs and preventing escape. During the last two years, a number of kayakers have suffered serious leg injuries in this way, and at least three have died.

The sport's enormous popularity has led to the exponential growth of professional guide services. In the face of increased competition, guide services have been taking inexperienced clients with little training and few skills down dangerous rivers farther from medical care. Evidence of this alarming trend appeared on the front pages of many newspapers last summer when five major United States business executives died after their raft flipped on the Chilco River in British Columbia. The exact cause of their death is unclear, but major contributing factors must have been their sudden and prolonged immersion in ice-cold glacier runoff water, compounded by a lack of sufficiently insulating clothing. One of the survivors was quoted as saying, "We looked at whitewater as sort of a roller coaster ride."

The dynamic and unpredictable nature of rivers can turn any mishap into a serious disaster with little chance for recovery. For this reason, our initial goal in whitewater medicine must be an ever-present emphasis on safety precautions and accident prevention.

SAFETY EQUIPMENT

According to the United States Coast Guard's boating accident statistics, the three most common contributing factors to whitewater-related deaths are cold water, inadequate swimming ability, and failure to wear a PFD (personal flotation device or life jacket).

The Coast Guard classifies PFDs into five types; of these, only two are commonly used in whitewater sports. Most paddlers wear Type III devices, which permit mobility and comfort but provide the minimum amount of flotation required for safety. Type V jackets are predominantly used by commercial outfitters because they provide more flotation and are asymmetrically constructed with a collar theoretically designed to turn the unconscious wearer face up.

Beyond flotation, life jackets have many other benefits that make them highly useful in wilderness settings. They add extra warmth and insulation to help prevent hypothermia, and they can act as padding in falls on slick rocks. In addition, they are excellent material for improvised splints. They can readily be fashioned into cervical collars for suspected neck injuries, and can easily be converted into cylindrical splints for the knee.

Another vital piece of equipment is a safety rope. This should be accessible in each craft, and secured in a manner which facilitates rapid employment and prevents entanglement. In the past five years, self-contained throw bags have virtually replaced the once-popular coiled ropes. Throw bags, though they take less time to deploy, reduce control over the amount of rope thrown. However, they are less likely to become loose and tangled during a raft flip.

Knives should be readily accessible. A fixed knife is preferable to a folding one, in the event that only one hand is free. Double-edged blades are preferred by many since they can cut in any direction and thus require minimal handling in precarious situations. Many modern knives designed for kayakers feature a serrated edge which can cut through plastic boats during entrapments.

Helmets should be worn by all whitewater boaters. Approximately 10% of all kayaking accidents involve head or facial trauma, as capsized boaters strike the rocks which are an inevitable component of all rivers.

Whistles should also be worn; they enable paddlers to alert others that an accident has occurred. Since paddlers are often spread out over the course of a rapid, attempting to alert someone downstream by yelling over the roar of the rapids can be a frustrating and fruitless endeavor.

Functional, insulated clothing should also be considered a mandatory safety item. Hypothermia is now recognized as a major cause of death among outdoor travelers. More significantly, a growing number of accidents are believed to have resulted from hypothermia-induced impairment of judgement and coordination. Immersion hypothermia is a most serious threat to paddlers during the Spring months when air temperatures are cooler and snow melt-swollen rivers run very cold. Many rivers, especially in the West, are controlled by dams which release water from far beneath the surface and thus remain cold throughout the season.

Studies have determined that water conducts heat away from the body 20 to 25 times faster than does air at the same

temperature. Placing adequate barriers between our bodies and the environment is therefore of paramount importance in aquatic sports. The insulating value of clothing is quantified by a comparative thermal resistance unit termed the CLO factor. The CLO value of cotton, for instance, is three times lower than that of wool or polyester; cotton loses all of its insulating properties when wet. On the other hand, newer synthetics such as polypropylene and polyester pile absorb no more than 1% of their weight in water and maintain their thermal insulating qualities even when wet. When a nylon paddling jacket is added, the combination of materials affords both insulation and wind protection.

Wet suits at one time were considered optimal garments for paddlers in extreme conditions. Today the dry suit has become the gold standard for cold water boating. Dry suits, however, are expensive and must be protected from ripping.

RIVER HAZARDS

The most fundamental hazards found on rivers are hydraulics. Other names for these phenomena are: pour-overs, holes, suck-holes, reversals, and rollers. A hydraulic is created when water flows over an obstacle, causing a depression and producing a vacuum within which the downstream water recirculates. Hydraulics can provide enormous thrills and entertainment; they can also be deathtraps. Rafts and kayaks can be turned upside down by the force of a hydraulic and, if the reversal currents are strong enough, crafts and their passengers can become trapped in the recirculatory flow.

The best way to avoid dangerous hydraulics is to scout the rapid beforehand and to portage when necessary. When proceeding

into a rapid which contains a hazardous hydraulic, always pre-set a rope below the hole to facilitate rescue.

A swimming escape from a keeper hydraulic may necessitate staying submerged and resisting the urge to regain the surface. Surfacing too early can result in recirculation. Hydraulics release water downstream from beneath the surface; often this is the only avenue of escape for the swimmer. Fortunately, most hydraulics eventually spit people out regardless of what action they may take.

Novice paddlers often misjudge the force of hydraulics. It is not the size of the drop which reflects the recirculating potential, but rather the shape and angle of the obstruction causing the drop, combined with water volume and downstream currents.

While hydraulics may be the most obvious hazards, statistically the biggest killers are undercut rocks and strainers.

Undercut rocks are boulders or rock formations which have been eroded away just beneath the surface of the water. They pose a significant risk of entrapment and drowning for both swimmers and kayakers. Unfortunately, they are difficult to recognize from the shore. A number of rafters have died as a result of flipping on undercut rocks and subsequently being pinned between craft and rock. In February of 1986 a kayaker on a commercial trip on the Rio Jatate in southern Mexico died when his upside-down kayak washed onto an undercut rock, pinning him beneath the surface. It took rescuers almost 24 hours to free the body.

Strainers--obstacles such as fallen trees, bridge debris, or driftwood lodged between rocks--will allow water to pass through but will trap the swimmer or boater. Strainers are notorious for

having caused the deaths of some of America's premier paddlers while they were attempting to run rivers during floods. Flooding, of course, increases the likelihood of strainers by washing riverbank debris into the flow.

Yet another kind of entrapment can occur when a swimmer attempts to stand up and walk in swift-moving current. A foot can become wedged between rocks beneath the surface, causing the unfortunate victim to lose his or her balance and fall face down into the river. This type of mishap has caused drownings in water that was less than three feet deep.

Swimmers in rapids should assume a position on their backs, with their feet at the surface pointed downstream to serve as shock absorbers. This minimizes the potential for foot entrapment as well as head and neck trauma.

Entrapment accounted for a threefold increase in whitewater-related deaths from 1985 to 1986. This statistic reflects more than an increase in the numbers of whitewater boaters; it also reflects the 1986 Western drought, which lowered river levels and exposed many more undercut rocks and hazardous areas.

Man-made hazards represent yet another threat to river runners. Low-head dams or weirs form perfect hydraulics with enormous recirculating potential. In the Binghamton Dam disaster of 1975, a 15 1/2-foot Boston whaler with a 20 horsepower engine was sucked into a hydraulic while attempting a rescue; this resulted in the deaths of three firefighters. Other man-made hazards include bridge pilings, submerged automobiles, and low-hanging power lines.

WHITEWATER FATALITIES

Although exact statistics are not readily available due to the inconsistent collection of data by various agencies, we know that most deaths occurring on rivers result from submersion accidents. Each year, accounts of drownings and accidents are compiled by the River Safety Task Force of the American Canoe Association; unfortunately, many accidents go unreported and even some deaths are not recorded.

We have seen that submersion accidents can result from a variety of mechanisms: most commonly, underwater entrapment by strainers, undercut rocks, hydraulics, vertical pins, or wedging of feet. Avoiding entrapment, therefore, must be emphasized as a safety measure, since rescue options are often limited by constraints of time, resources, and ability to reach the victim.

Important preventative measures include:

1. Prior scouting of rapids
2. Pre-setting of safety ropes and rescue boaters
3. Swimming in rapids on one's back with feet downstream and near the surface, thereby using the feet as shock absorbers and minimizing the risk of foot entrapment
4. Questioning local paddlers about hidden hazards or going with a guide who knows the river well
5. Rescuing swimmers before chasing down equipment
6. Avoiding rivers when they are in flood or at an unusually low level
7. Respecting the power of the river.

Regardless of the mechanism of the submersion incident, once extrication has been achieved the gold standard for first aid is

still immediate and aggressive initiation of ventilation and perfusion. Evidence of trauma should be noted and precautions taken in the event of a cervical spine injury. Hypoglycemia and seizures have contributed to drownings during whitewater activities; these must be considered. Because apnea usually precedes cardiac arrest if ventilation is provided, circulatory resumption may occur spontaneously. Supplemental oxygen should be provided as soon as possible.

An important group of submersion accident victims who seem to defy predictions for outcome are those who have experienced prolonged submersion in very cold water. The designation "cold water drowning" has led to some confusion since the mechanisms prolonging survival are not entirely understood. Nor has anyone positively defined "cold water." There have been at least 19 reported cases of submersion estimated at greater than 15 minutes with good neurological outcome. Fourteen of these victims were less than 19 years of age, and of the recorded water temperatures, all were 10° Centigrade or lower.

Acute Submersion Hypothermia--defined as the rapid development of hypothermia during fresh water drowning due to core cooling from pulmonary aspiration and absorption of cold water--has been proposed by Alan Conn, an anaesthesiologist at Toronto Hospital for Sick Children, as an explanation. If enough cold water were aspirated, cerebral cooling might occur prior to circulatory collapse, thus delaying permanent brain injury. Whatever the mechanism behind prolonged survival, the overt implication is the value of prolonged CPR after submersion in very cold water.

There is no logic in attempting to drain water from the lungs in fresh water immersion since the water has rapidly been absorbed into the circulation. The Heimlich maneuver is not recommended unless there is no response to ventilation due to an obstructed airway.

TRAUMA

One of the most common traumatic injuries occurring in whitewater sports is the sprained ankle. Ironically, this usually occurs off the water--while one walks along the banks on loose, wet, slippery rocks during scouting or portaging, or upon entering or leaving the river. Ankle injuries also occur on the river when rafters are thrown about in rapids. Twice I have suffered foot and ankle injuries when the person sitting across from me was tossed into the middle of the raft onto my leg, which was stretched out in a bracing position. Kayakers are prone to ankle injuries from sudden inversion or dorsiflexion when the bow of the boat hits an obstruction such as a submerged rock. The feet are held against the narrow braces while the heels are pushed underneath or the entire ankle is inverted.

Management of foot and ankle injuries encompasses the triad of ice, elevation, and compression to reduce swelling. River water is usually substituted for ice. Splinting is important for reduction of pain and edema and to limit extension of the injury. At one time most rafting companies carried pneumatic splints in their first aid kits. While providing adequate support, protection, and compression, air splints are also liable to overinflate when heated by the sun. Their zippers often malfunction as a result of rusting from moisture or jamming by sand particles. Neurovascular

integrity must be checked frequently with this type of splint. I prefer either to improvise a pneumatic splint from my kayak float bags, or to fashion a "pillow splint" from an extra life jacket or an article of clothing.

Head, facial, and dental trauma are more common in kayakers and decked canoeists than in rafters because of the potential for flipping upside-down while still in the craft. Minor abrasions, lacerations, and contusions are common; serious head injury with loss of consciousness is fortunately rare. Head and facial trauma can be minimized by wearing protective helmets and tucking forward while rolling, instead of leaning backward. Cervical spine injuries accompany 5 to 10% of serious head trauma and should always be considered in the assessment.

Most kayakers suffer frequent abrasions and contusions to the fingers and knuckles of the hand while hanging upside-down after flipping. Oar frames, oars, paddles, and the ammo boxes which are used to keep supplies dry can all inflict injury when rafts are capsized or tossed about in turbulent water. Significant injury can occur when a swimmer is suddenly sandwiched between a downstream boulder or obstruction and the upstream raft, canoe, or kayak that he or she has exited. The rise in popularity of kayak wave-surfing in the ocean has increased the number of incidents in which paddlers and surfboarders alike have been speared by the front of a kayak. Visceral injury is possible and I have seen one case of a pneumothorax by this mechanism. Less serious injuries such as fractured ribs and chest wall contusions frequently result, probably because padded life jackets offer some protection.

Back injuries from strain and overuse are frequently seen among whitewater paddlers. Rafters are prone to such injuries while portaging, pushing stuck rafts off rocks, or carrying crafts to and from the river. Raft guides frequently suffer back strain when pulling capsized customers--who usually weigh more than the guides themselves--back into the rafts. The awkward task of bending over a bulky side compartment and lifting a heavy individual out of the water produces enormous stress on the lumbar musculature. Unfortunately, the construction of rafts tends to limit support from the legs while lifting. Kayakers and canoeists more commonly injure their backs when lifting water-laden boats to empty them, or when loading craft onto automobile roofs. Using the legs for lifting, and sharing the load with another individual, will reduce the likelihood of such injuries.

The repetitive dorsiflexion of the dominant wrist required to operate feathered kayak paddles often produces tendonitis and synovitis. Using a paddle constructed with a 75°-80° offset instead of the traditional 90° can limit stress to this joint. Aspirin or other nonsteroidal anti-inflammatory medications taken 30 minutes prior to paddling have been found to be beneficial. Wrist supports provide limited relief.

The injury most associated with kayaking is the anterior shoulder dislocation. According to one survey of Eastern kayakers, over 10% had suffered at least one shoulder dislocation. The maneuver most notorious for precipitating this injury in kayakers is the high brace. Often used while supporting oneself in hydraulics, surfing on waves, or preventing capsizing, the high brace entails abduction of the humerus with external rotation of

the glenohumeral joint. If the arm should become extended behind the midline plane of the body either by the force of the current or when the paddler lifts the head and turns the body away from the bracing paddle, the triad of forces leading to an anterior dislocation are produced. The humeral head is driven forward and frequently avulses the cartilaginous glenoid labrum and capsule from the anterior aspect of the glenoid cavity. The paddler will usually be aware that something has "gone out of place" and will hold the extremity away from the body, being unable to bring the arm across the chest. The shoulder may appear square due to the anterior, medial, and inferior displacement of the humeral head into a subcoracoid location.

Prevention: The preferred method of bracing which minimizes the risk of shoulder dislocation is the low brace in which the arm is held in internal rotation and close to the body (adduction). This type of brace, while initially more awkward for the novice paddler, is inherently stronger and more versatile as it allows back paddling out of hydraulics.

The shoulder joint itself depends for its stability upon the joint capsule and surrounding musculature. Exercises which strengthen the rotator cuff, deltoids, triceps, and pectorals will inherently reinforce the glenohumeral joint.

Treatment: After the shoulder has been dislocated, the longer it remains out, the harder eventual reduction will be due to increasing muscle spasm. Although on-scene reduction of dislocations is still controversial, the immediate relief of pain, curtailment of ongoing injury to the joint, and subsequent ability of the victim to function more actively in facilitating his or her

own evacuation are all strong reasons in favor of the attempt. Many techniques have been advocated for reduction. The key element is the ability to maintain traction-countertraction while maximizing muscle relaxation in an improvised setting. Always monitor circulation, motor and sensory function to the hand before and after attempting reduction.

Blisters are a ubiquitous problem in paddlers. Among kayakers they develop most often between the metacarpalphalangeal (MCP) joint and the interphalangeal joint of the thumb along the ulnar aspect. Prior taping and eventual callous formation will reduce the incidence of this potentially incapacitating problem. Common sites of blister formation in rafters and canoeists are the proximal palmer surfaces of the MCP joints of the hands.

Infections: Blisters, along with abrasions and lacerations, are always at increased risk of becoming infected in an aquatic environment. Maceration from prolonged immersion in water and exposure to pathogens are predisposing factors. Abscess formation and cellulitis requiring incision, drainage, and antibiotics are common during expedition boating when participants spend extended periods on the river. A recent study of the bacteriology of the freshwater environment published by Paul S. Auerbach, M.D. in The Annals of Emergency Medicine yielded a diverse collection of pathogens including Aeromonas, Pseudomonas, and Vibrio species. Antibiotics that were found to be effective against these gram-negative organisms included: Trimethoprim-Sulfamethoxazole, Tetracycline, Ciprofloxacin, Imipenem, and Ceftazidine.

First-generation Cephalosporins, Ampicillin, and Erythromycin all provided poor coverage[1].

An outbreak of Staphylococcus Aureus skin infections among raft guides in Georgia and South Carolina nearly led to the closing of two rafting companies. Sharp grommets on the thwarts of the rafts had caused repeated lower-extremity abrasions. The causative organism was cultureable from rafts up to 48 hours after use. Daily raft disinfecting became a standard practice which kept the companies from being forced to close.

Otitis externa (swimmer's ear) is a common problem among paddlers. Water exposure to the ear canal both macerates the epithelium and neutralizes the normally acidic pH of the canal, predisposing to infection. The bacteria most commonly cultured are Pseudomonas Aeriginosa, Proteus Vulgaris, and Staphylococci. Purulent discharge from the external auditory canal in a patient, with pain aggravated by traction on the auricle or pressure on the tragus, will confirm the diagnosis. Antibiotic ear drops with or without hydrocortisone are widely available and very useful. Irrigation of the canal with commercially-available solutions containing acetic acid and alcohol will help prevent infection by lowering the pH and drying the canal. The drops should be applied after each outing.

The recent publicity given to water contamination by Giardia is now reinforced by statistics from the Federal Centers for Disease Control which report Giardia Lamblia to be the most common pathogenic intestinal parasite in the United States. Giardia cysts

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[1]Auerbach, P. S. et al. 1987. Bacteriology of the freshwater environment: implications for clinical therapy. Ann. Emerg. Med. 16:1016-1022.

flourish in mountain streams and rivers which were once considered sources of pristine water. They even thrive in very cold water and lend no taste or smell to the water they infest. Rivers are usually contaminated by animals that defecate in or near the water. Studies by the Wild Animal Disease Center at Colorado State University have identified more than 30 species as carriers. Physicians should be aware that onset of symptoms may occur one to three weeks after ingestion, and should not overlook this diagnosis.

Whitewater rafting in foreign countries is becoming increasingly popular as adventure travel companies promote more international trips. Unprepared participants are thus exposed to infectious diseases not encountered in the United States. This was exemplified by a recent report of Schistosomiasis in a group of eleven rafters on a Sobek Expedition in Ethiopia. Schistosomiasis is endemic to large areas of Africa, South America, and the Caribbean, and is transmitted to humans who swim or come into contact with fresh water containing the larval stage. Rafters going to areas in which this disease is endemic should be screened with serologic testing, since up to 50% of infected cases can remain asymptomatic.

ENVIRONMENTAL HAZARDS

Whitewater participants are exposed to an array of environmental hazards, most of which can be readily avoided or prevented. Sunburn and other effects of solar radiation are magnified by the water's ability to reflect up to 100% of the UV light, depending on the time of day. Sand can reflect up to 17% of harmful UV radiation. The "paddler's tan," outlined by the life jacket, is not only painful but can predispose to neoplasms from

repeated exposure. Sunscreens must be applied frequently, as they are prone to washing off in the water. PABA-containing sunblocks, applied a couple of days prior to exposure, will absorb into the epidermis, affording a cumulative and more stable protective barrier. Zinc oxide and other agents more resistant to water are preferable.

Eye protection, often overlooked by paddlers, is important to shield from the chronic effects of UV exposure as well as the more acute problem of photokeratitis. Most rivers are situated in the mountains, where every additional 1000 feet of altitude gained increases the amount of UV exposure by 3 to 4%. Polarizing lenses are helpful in reducing glare from the water but are not by themselves adequate filters of UV and infrared radiation.

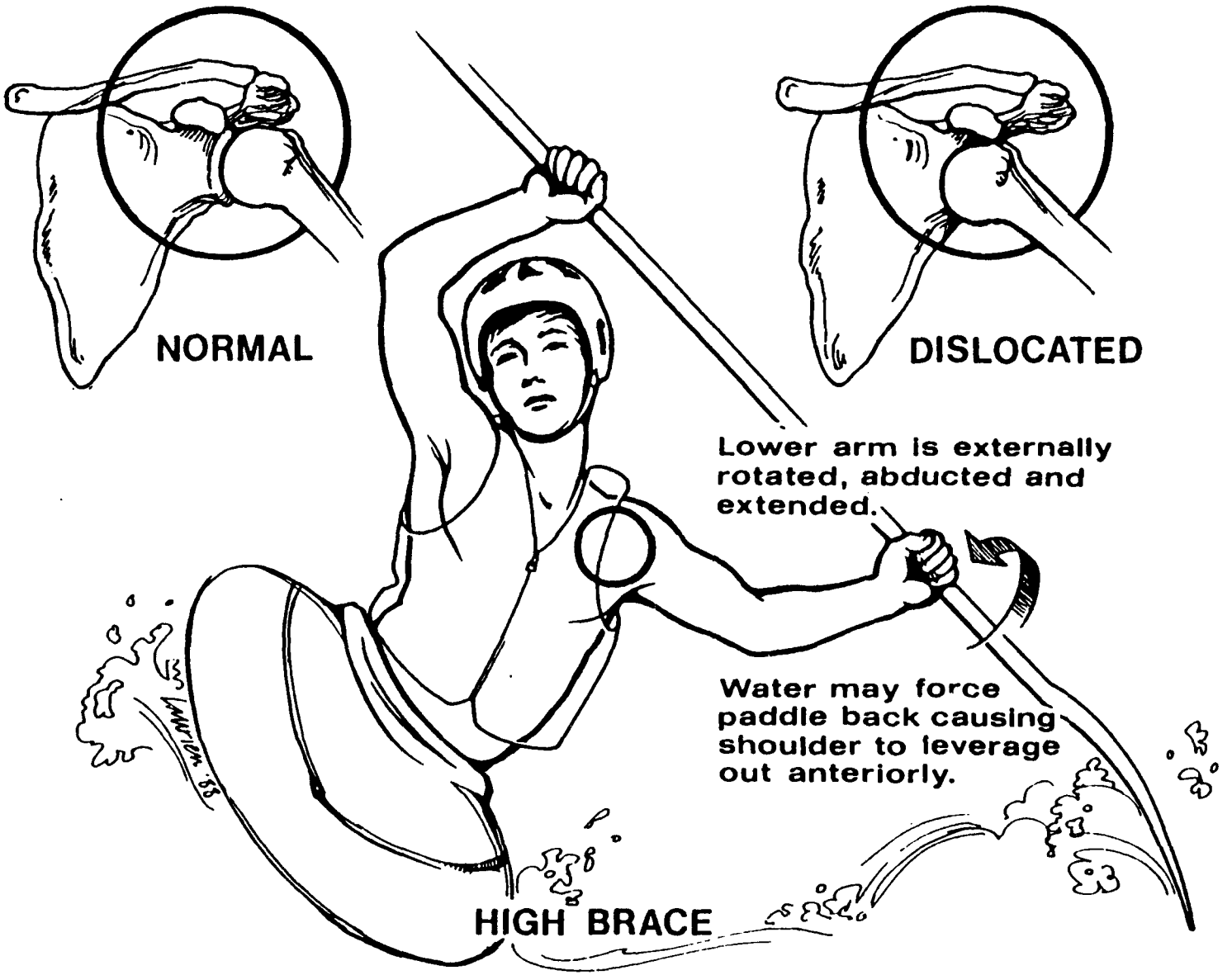
The second most common environmental affliction suffered by paddlers is the dreaded "weed." Rhus dermatitis from poison oak or poison ivy is especially ubiquitous in the Spring, when the vine itself is very potent but the characteristic leaves have not yet appeared--making identification of the plant difficult. The unfortunate paddler is not often aware of his or her exposure until days later when the characteristic dermatitis develops.

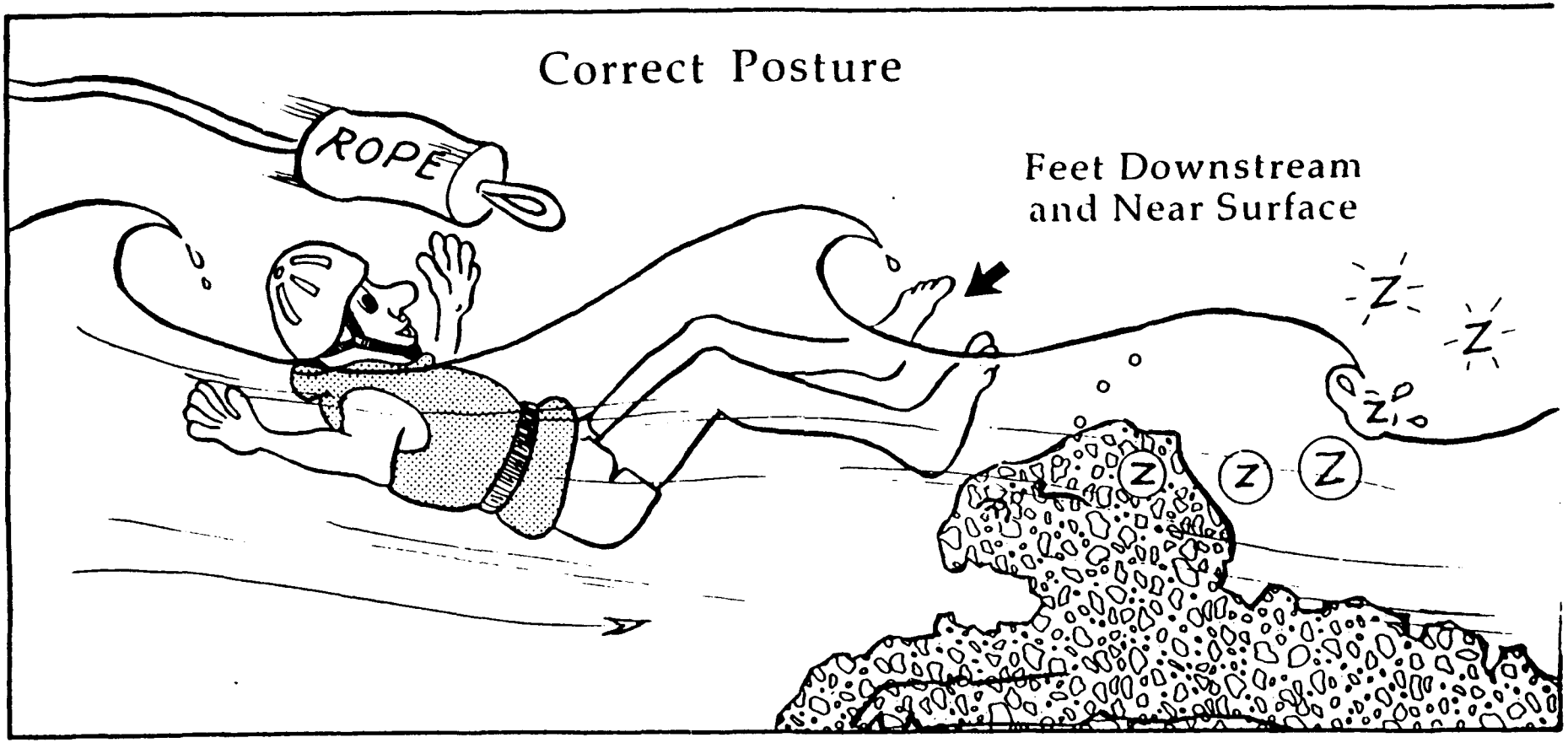
Venomous snakes--most notably the pit vipers--along with scorpions, spiders, and fire ants, are frequently encountered by river runners and should be considered a potential hazard. Reported cases of envenomation, however, are rare.

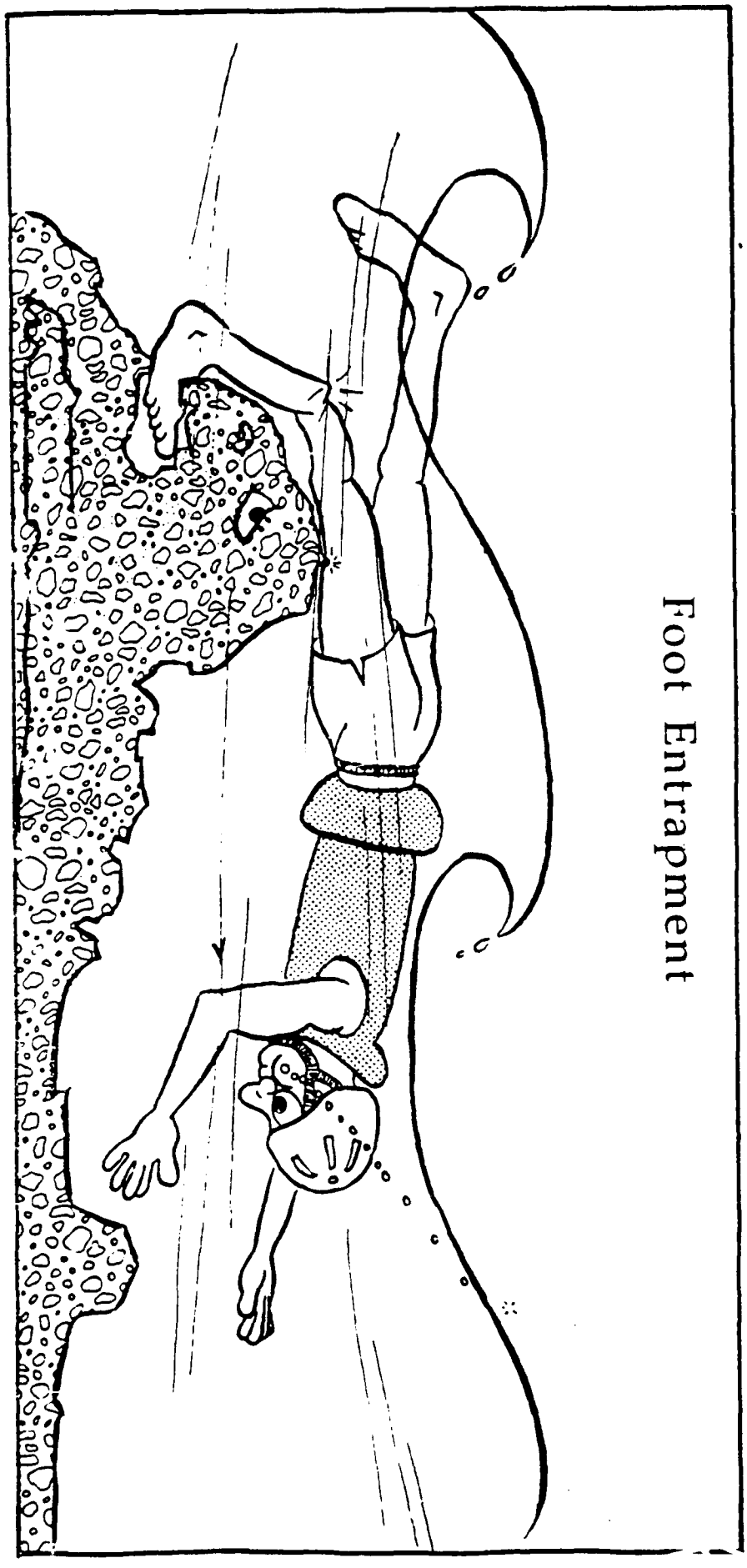
CONCLUSION

The beauty and adventure offered by our nation's rivers, combined with opportunities for spiritual growth, camaraderie, and physical and mental challenge, are luring greater numbers of

people to participate in the sport of whitewater boating. Becoming more familiar with the inherent dangers and medical ramifications of the sport will aid physicians both in advising patients about accident prevention and in treating their injuries.


NORMAL
DISLOCATED
Lower arm is externally rotated, abducted and extended.
Water may force paddle back causing shoulder to leverage out anteriorly.
HIGH BRACE


Correct Posture
ROPE
Feet Downstream
and Near Surface
Z
Z
Z
Z
Z
Z



Foot Entrapment

AVALANCHE RESCUE

Search Dog, Probe and Beacon Techniques

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INTRODUCTION

Avalanche rescues, for the most part are associated with body recovery. Many buried victims do not survive, but rescue attempts must be performed, for there is always hope!

The primary cause of death is suffocation, followed by collision with obstacles, then hypothermia and shock. Chances of a live recovery diminish rapidly with time and depth of burial. Fifty percent of buried victims perish within thirty minutes. This figure includes persons buried in buildings and vehicles.

A quote form the book Kampf uber die Gletschern (Battle over the Glaciers) by W. Schmidkunz says all, "The Snowy Torrents are like the deep sea; they seldom return their victims alive."

Avalanche rescue operations are only effective with a written plan, adequate equipment, and organized trained rescurers. Persons pursuing backcountry travel in avalanche prone areas should be equipped for self rescue. Organized rescue groups are usually too late for a live recovery. The following tables from the Avalanche Handbook summarize survival and causes of death in an avalanche.

TABLE I

Percentage Survival of People Caught in Avalanches	
80 %	Survive if they remain on the surface.
40-45%	Survive if partially or completely buried.
55-60%	Survive if protected by buildings or vehicles.

TABLE II

Causes of Death in Avalanches	
65%	Suffocation
25%	Collision with Obstacles
10%	Hypothermia and Shock

Table III

Probability of Live Recovery	
12%	Live longer than 30 minutes
6%	Live longer than 1 hour
4%	Live longer than 2 hours

AVALANCHE RESCUE

The topic is on three means used in avalanche rescue, each one will be broken down and discused but at this point the amount of time involved in each process will be outlined. The area is 100 meters by 100 meters and the procedures in each, conducted by trained personnel.

Avalanche Beacon	
Course Searching Time (on skis)	3 min/hectare
Fine Searching Time	5 min/hectare

Avalanche Dogs	
Course Searching Time	30 min/hectare
Fine Searching Time	1-2 hrs/hectare

Probes

Course Searching Time (20 men)	4 hrs/hectare
Fine Searching Time (20 men)	20 hrs/hectare

With trained personnel, equipped with avalanche beacons, search times are within a reasonable recovery time. As long as the victim is equipped with a beacon and rescue is initiated as quickly as possible, there is a very good chance of a live recovery.

Most North American organizations have agreed with The International Commission of Alpine Rescue (IKAR) to adopt transceivers with the 457 KHZ frequency. There are many models available to the consumer and product advancements have been made. The transceivers are only as effective as the user, practice is essential, time is the factor.

Avalanche dogs are more effective than probing but in North America there is the problem of these dogs being spread out and the terrain being vast. There have been no live recoveries by dogs in Canada. In Europe however, the avalanche dog organizations have had many live recoveries, due to their extensive network of well trained handlers situated in alpine environments.

In recent years, The Banff National Park wardens and dog handlers in the Whistler area have used the Helicopter Flight Rescue System (HFRS) to deploy dog and handler onto slide paths. The HFRS system uses a fixed line attached to the under carriage of a helicopter to transport the rescue party. This procedure is timely and reduces the risk of exposing other rescuers to further hazards.
It is important to remember that burial sites should be kept as scent free as possible if a dog is being used.

Probing is the least desirable means of recovery. It requires 20 rescurers to be effective and is a slow and tedious procedure. Probe lines must be managed in an orderly and systematic fashion. For further information consult the Avalanche Handbook.

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Field Recognition and Management of Exotic Snake Envenomation

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OBJECTIVES

Following this presentation, participants will be able to:

1. Describe the taxonomy and natural history of venomous snakes.
2. State factors that increase the risk of human-snake encounters.
3. Delineate methods for preventing snakebite in the wilderness.
4. Describe appropriate treatment for the envenomated patient in the wilderness setting.

Introduction;
Snake venom poisoning is a medical emergency that requires sound judgement, accurate assessment and definitive treatment with appropriate antivenin. Managing these injuries in austere and wilderness environments is, with few exceptions, limited to first aid measures that vary with the type of snake. Many first aid measures previously recommended are now known to be useless or even detrimental to the patient. In all cases of confirmed snake envenomation in the backcountry, the priorities are basic life support, restriction of systemic spread of venom, and expedient evacuation of the patient to a hospital for immunotherapy with appropriate antivenin.

Epidemiology,
Venomous snakebite continues to be an important medical and economic problem for developing countries. Underreporting of snakebite occurs in many countries due to dependence on hospital admission reports (which are biased in favor of severe cases). In one survey in northern Nigeria only 8.5% of people bitten by cobras sought medical care[1]. Mortality due to snakebite is similarly underestimated due to reliance on hospital records. In India the reported number of annual fatalities from all provinces has exceeded 20,000 over the last number of years[2]. Brazil reports over 2000 deaths per year from all species[3]. Snakebite is responsible for 5% of deaths among the Waorani tribes of eastern Ecuador and 2% of the Yanomamo of the southern Amazon basin[4]. Snakebite is the fifth most common cause of death in Burma[5]. In the United States there are over 7000 venomous snakebites recorded each year with 9-14 deaths[6].

Natural History;
An understanding of taxonomy is important for the treatment of reptile envenomation because venoms from reptiles of the same genus often produce similar clinical effects. Secondly, scientific names for species are important because they are employed internationally to describe specificity of many antivenins.

Approximately 550 of the 2787 currently known species of snakes, are known to be venomous. The venomous snake families are the Elapidae, including the cobras, mambas, and kraits; Hydrophobidae, including Australian terrestrial snakes and seasnakes; Viperidae, the vipers and pit vipers; and Atractaspididae or "asps", which were previously classed with the Viperids. Over 200 species belonging to these families have caused human deaths. Over 45 species of a fifth family, the Colubridae, are now known to be capable of human envenomation.

Venomous reptiles are distributed throughout the world. Venomous snakes are found as far north as the arctic circle (*Vipera amalon, V. b. beres*) and at altitudes exceeding 5100 m (*Agkistrodon himalayanus*). There are no venomous snakes in New Zealand, Ireland, Madagascar, Atlantic, many Carribean and Pacific islands and polar regions.
Reptile activity patterns are dictated by the behavior of each species, temperature, circadian and diurnal rhymes. All venomous snakes are thigmotrophic to some extent, preferring to conceal themselves from predators through protective coloration and hiding. Prey animals are caught by questing (sea snakes, certain elapids) or by ambushing food species from a secure hiding place (certain viperids). Changes in populations of venomous snakes occur naturally with changes in prey animal populations and climate, or through human encroachment and development of wilderness areas. In particular, flooding, and sudden changes in ecotone (e.g. deforestation) have been attributed with displacing venomous snakes-often with increases in the number of snake bite. In Burma peak incidence of snake bites occur in June when rice is planted and in November when it is harvested[5]. The aggressiveness of each species also plays a role in the incidence of snakebite. Rattlesnakes, cobras, and mambas and members of the Atricaspididae strike readily when cornered,

while coral snakes, kraits, sea snakes and the Gaboon Viper and it's relatives (*Bitis*) appear reluctant to strike. In developing countries the majority of snakebites result from inadvertently disturbing the snake, usually by stepping on the animal in darkness. Encounters such as these, where the victim is ignorant of the snake's presence are referred to as legitimate bites. In contrast, 30% of venomous snakebites in Alabama are illegitimate bites, that is, the snake is known to be venomous prior to the bite, and over 50% of snakebites in adults are associated with alcohol consumption[7].

Prevention;
Prevention of snakebite is insured through common sense and simple precautions (resources that may be in short supply among certain wilderness travellers). Snakes should not be disturbed or handled without first determining that they are nonvenomous. Individuals insistent on approaching snakes in the wild should first learn about the natural history of local species from a responsible source. Never grab exposed tails in rock scree or leaf litter without first determining where the animal's head is located. Many allegedly "dead" snakes have regained their senses while being handled, or in the emergency department. "Rules" for identifying venomous snakes in one region may prove inaccurate in other regions. Appropriate clothing should be worn in snake infested areas, including baggy trousers, stout boots and socks. Flashlights should be used after dark, and when collecting firewood in dense cover. A long walking stick can be used to probe heavy undergrowth and loose sand prior to passage. When travelling through low canopy, look ahead for the dangling vine-like representatives of the genus *Dispholidus* (Boomslang) and *Thelotornis* (vine snakes). Rock climbers should be cautious of the presence of snakes sunning themselves on overhead ledges. Fisherman should wear stout rubber gloves and boots when emptying nets. Swimmers should avoid overgrown beaches and bodies of water with floating aquatic plants. Undercut rocks and logs should first be inspected before dislodging them. Particular care should be taken in the days following heavy rain, and earthquakes as snakes may have been driven from their hiding places.

Envenomation kinematics;
Snakes do not have to be coiled to strike. Lateral strikes by extended snakes and bites from "cold" or torporous snakes have resulted in envenomation. Given sufficient warning, elapids (mambas, kraits, cobras) rear up to 1/3 of their length above the ground. This can prove to be exciting when the serpent is a 5m cobra (*Ophiophagus hannah*). Elapid strikes start high and end low, resulting in a high percentage of bites to the lower extremity. Defensively posturing Viperidae display lateral coiling of the forward body, often with the head and neck held off the ground. This defensive position produces faster strikes and greater range. Certain species of pit vipers are particularly pugnacious, striking aggressively and repeatedly over half their body length.

Venom is introduced through wounds made by chewing, or directly through specialized fangs, canaliculated or grooved teeth. Members of the Viperidae family, subfamilies Crotalinae (e.g. rattlesnakes) and Viperinae (e.g. old world vipers), have hinged fangs. At rest the fangs are covered by a protective sheath and lie flat against the divided mixilla. The Elapidae, including the cobras, kraits, mambas and seasnakes of the genus Laticauda, and the Hydrophobidae, including venomous Australian snakes and other sea snakes, all possess short, non-retractable front fangs. Evolution of fangs varies from enlarged canaliculated posterior maxillary teeth in the genus *Rhabdophis* (yamakagashi), to fixed rear fangs in *Dispholidus* (boomslang) to hollow retractable fangs of the viperidae. Viperidae and Atractaspididae are constantly replacing fangs. Often one to as many as four fangs are functionally positioned. The presence of more than two fang marks at the bite site does not necessarily indicate multiple bites.

With the exception of colubrids, all venomous snakes produce venom in specialized glands located below the eye and spanning posterior to the mandibular joint. Venom is forced from the glands by compressor muscles through specialized ducts which transverse the mucosa and terminate at the anterior base of the fangs. Venom travels the length of the fang in partially or completely enclosed canals, exiting through an opening near the tip. In certain species of cobras, the canal opens into a groove which allow these species to spray a fine mist of venom during exhalation. Venom can be sprayed or "spat" up to 2.5m, and is used for defensive purposes. Rear fanged representatives of the Colubridae produce venom toxins in a modified region of the superior labial gland known as the Duvernoy's gland. During envenomation venom is discharged and transmitted to the bite wound through grooved teeth. In order to introduce sufficient venom Colubrids must hold on and chew the tissue.

Not all bites by venomous snakes are accompanied by envenomation. The bite and the delivery of venom are independently innervated events. This may explain the high percentage of trivial or asymptomatic bites from certain species. Approximately 20% of *Crotalus* and *Bothrops* bites[6] , 50% of *C. rhodostoma* and *V. russellii bites*[5] and over 80% of sea snake bites are accompanied by trivial or no envenomation[8] .

Snake venom biochemistry;
Snake venom, as with snake behavior and environmental adaptation, attests to the remarkable evolution of these animals. The eastern diamondback rattlesnake (*C. adamantus*) for example, possesses a venom particularly toxic to rabbits, the preferred prey. Similarities between snake venoms, and therefore certain clinical effects produced by these venoms, correlate reliably with genera level of classification. However, the age, geographic location, season and even the sex of snakes are known to influence the toxicity of venom.

All snake venoms are comprised of numerous enzymes, polypeptide toxins, and glycoproteins Many venoms contain biogenic amines such as 5-hydroxytryptamine and histamine. These small molecular weight agents are believed responsible for the pain immediately following envenomation by viperidae and certain elapids. Snake venoms are often described as either neurotoxic or hemotoxic; a oversimplification that neglects the multiple targets and synergy of the venoms. For example, venom from cobras and Australian snakes are known for their neurotoxicity, yet bites from these species also cause devastating tissue necrosis rivaling that observed in viper bites. Conversely, several crotalids such as *C. durissus terrificus* , *C. scutulatus* and *V. russelli* spp. possess potent neurotoxins. Several medically significant toxins found in snake venoms are listed in Table I.

Pathophysiology of snake venoms;
Envenomation in humans is an aberrancy of the effects observed in small prey. In general, the greater mass and blood volume of humans seems to be beneficial for surviving the acute effects of the venom. For example, untreated human victims of *C. adamantus* (E. diamondback) envenomation may survive the massive intravascular coagulation that kills the rabbit, but may die days later from hemmorhagic shock or cerebral hemorrhage. Pathophysiology of envenomation may be abstracted into three concurrent, consecutive and/or synergistic actions; These are

1.) injury caused by the direct effects of venom
2.) autopharmacologic effects due to the release of inflammatory mediators and vasoactive compounds.
3.) secondary complications such as infection and disabling sequelae.

I. Viperidae venoms;
Approximately 85% of Viperidae venom by weight is water. Viperidae venoms also contain 7 - 9 enzymes , 2-16 non-enymatic peptides, and low molecular weight substances including amines[9,10]. Viperidae toxins show no particular affinity for plasma proteins and in the case of subcutaneous bites, tend to be spread by lymphatics. There is considerable variation in tissue affinity and eventual elimination of viperidae venom components. *Agkistrodon* (cottonmouth) venom is bound to lung tissue and several *Crotalus* (rattlesnake) venoms have been shown to be concentrated in the liver and excreted in the bile[9].

Many viperidae peptides lyse cell membranes and damage the vascular endothelium permitting vascular fluid exudation into surrounding tissue. Progressive plasma extravasation into tissues and vasodilation induced by venom kininogenases, histamine and probably cytokines, lead to hypotension, hemoconcentration, and acidosis. In unpublished observations, our laboratory has measured high levels of the "shock cytokines" TNF-α and IL-1β in rabbits artificially envenomated with *Crotalus* venoms, and in plasma samples from patients envenomated by pit vipers. Viperid venoms also contain significant amounts of phospholipase A_2 enzymes. which induce hemolysis through the release of lysolecithin.

Viperidae venoms also induce coagulopathies. These clotting aberrancies are the result of multiple sites of enzyme activity and include platelet aggregation, thrombocytopenia, hypofibrinogenemia, DIC, and myriad DIC-like syndromes [11]. Several species of viperidae also possess neurotoxic peptides such as crotoxin of *C. durissus terrificus* (So. American rattlesnake) and Mohave venoms A and B of *C. s. scutulatus* (Mohave rattlesnake).

II. Elapidae and Hydrophobidae venom;
The toxic polypeptides of the Elapidae (cobras, mambas, kraits) and Hydrophobidae (seasnakes and Australian snakes) are small molecules that are rapidly absorbed into the blood stream. The principle toxins of medical importance are small molecular weight polypeptide neurotoxins. Elapidae neurotoxins can be divided into two groups: 1.) the disulfide bridged peptides which include the cell membrane toxins and postsynaptic α-neurotoxins, and 2.) the phospholipase A_2 toxins, including presynaptic β-neurotoxins and myotoxins[12,13]. Many elapid and hydrophobid venoms also contain acetylcholinesterase activity.

Several Elapidae and Hydrophobidae venoms induce tissue necrosis. In particular, bites from *Naja n. nigricollis* (Black-necked Spitting cobra), and Hydrophobidae (Australian snakes and beaked sea snakes) cause extensive tissue injury. Venom from Hydrophobidae and many species of elapidae induce generalized rhabdomyolysis, resulting in myoglobinurea and danger of renal failure. Several important Elapidae and Hydrophobidae toxins, and the species that produce them are listed in Table II

Clinical presentation;
The victim of snakebite may present with symptoms resulting from damaging "first aid" measures, from emotional anxiety, or from the actual effects of the venom. Anxiety and fear can smoke screen accurate assessment and interfere with management. Tourniquets can occlude blood flow to a bitten extremity resulting in numbness, tingling, pain, congestion, swelling, and loss of distal pulses. Cruciate incisions, scarification and currettage can cause

structural damage to underlying nerves and vessels leading to uncontrolled bleeding, loss of function, secondary infection. Cryotherapy may provide pain relief, but often at the expense of increased tissue damage. Electrical shock therapy for snakebite has no scientific or medical basis, but has rekindled the depressed "stun gun" market in the United States.

Unabating pain , edema and the presence of one or more fang marks provide the most reliable indication of Viperid envenomation. Cobra and mamba envenomation (Elapidae) also produces pain and variable edema, but fang marks may not be obvious. Local *erythema* and *swelling* become noticeable within minutes following bites by the Viperids, Australian snakes, cobras (*Naja*) and mambas (*Dendroaspis*). Care should be taken concerning bites by PanAmerican coral snakes (*Micrurus* and *Microroides*) and African and Asian kraits (*Bungarus*) because of the localized anaesthetic effects of the venom. Krait and coral snake venoms produce little local change, and the first indication of envenomation could be a systemic symptom. Blood may be totally defibrinogenated in 15 minutes following bites by the Malayan Pit Viper *C. rhodostoma* [14] and respiratory paralysis present 30 minutes following bites by mambas and cobras. There are recorded deaths within 20 minutes following bites by cobras, mambas and Australian snakes. These cases are, however, exceptional, the usual time for death being hours following Elapidae and Hydrophobidae envenomation, and days following Viperidae envenomation[2,15]. Multiple assessment schemes are used to grade the severity of envenomation. The simplest and most applicable scheme was described by Russell (way back when) for pit viper envenomation. This scheme seperates the severity of envenomation into trivial; minimal, involving swelling and echymosis at the bite sight; moderate, involving the extremity; and severe, the presence of any systemic symptom (e.g.perioral paresthesias, changes in clotting values)

Death from Viperid venoms is usually due to circulatory collapse associated with a drop in blood volume due to extravasation through capillary endothelium and pooling of blood in microvasculature. Death from Hydrophobidae and Elapidae envenomation is usually due to respiratory paralysis induced by curariform effects on neuromuscular junctions.

Wilderness Management

First Aid:

Get the victim away from the snake. Attempt snake identification in regions where species-specific (monovalent) antivenin is used, and where Viperid and Elapid species overlap. Sacrifice the snake only when I.D. is questionable and when it can be accomplished safely.

Manage ABCs. Management of airway, breathing and circulation takes priority. Obstruction of the upper airway by the tongue or inhaled vomitous occurs in envenomation by Australian snakes, mambas, kraits and several species of cobras, and can precipitate respiratory arrest. Breathing difficulty has been recorded in less than 1 hr following envenomation by a cobra[16]. In unresponsive patients, airway can be managed by proper positioning of the head, and positioning the patient on their side. In anaphylactic reactions, and bites that cause swelling of the upper airway, an oralpharyngeal airway (if available) or airway improvised from available materials can be used. In elapid and hydrophobid envenomation, respiratory paralysis may develop with intercostal muscles affected first. In the absence of antivenin and anticholinesterases, patients supported by manual ventilation or ventilator have recovered sufficient diaphramatic movement to breath on their own in 1 to 4 days[15,20]. Several ambubags (BVMs) such as the Svenchen (Zurich, Switzerland) have been developed for use in extended field care . The Svenchen BVM weighs .4 kg and has sufficient volume for hyperventilation of adults.

Assess the patient. Calming the patient reduces faintness and hyperventilation which hinder accurate assessment. Fang marks, pain, swelling, erythema, or in elapids, tingling at the bite sight, or the presence of any systemic symptoms not attributed to excitation, indicate envenomation. Assuring the patient may also decrease heart rate, with the potential benefit of slowing the spread of venom.

Immobilize the bitten extremity. Animal experiments have demonstrated that immobilization of the envenomated extremity delays systemic spread of crotalid and elapid venom[16,17]. Immobilization is effectively accomplished in the wilderness by improvisation. Excellent immobilization is accomplished using SAM splints™, internal frame stays from packs and crevat sling and swaths. EVA pads can also be tailored to immobilize extremities. Patients with bites to the abdomen, neck and face should be backboarded if possible. Spine immobilizers improvised from external pack frames or KED™ extrication devices are suitable for this purpose.

Initiate mechanical suction over the fang marks. The Extractor™ (Sawyer Products, Safety Harbor, FL, USA) is a portable pump that generates up to one atmosphere of negative pressure. Animal studies using the Extractor have demonstrated up to 30% removal of crotalid venom proteins if used within three minutes following the bite[18]. In other animal studies using *C. horridus horridus* and *C. horridus atricaudatus* venom, suction continued to remove radiolabelled venom until swelling of tissues prevented an effective seal between the pump bell and skin[19]. Rubber suction cups such as those included in the Cutter Snakebite Kit (Cutter Industries, Newark, NJ) are compact alternatives but lack the high suction pressures produced by the Extractor. Suction units can be improvised in the wilderness by cutting a large irrigation syringe (60 cc) with a heated knife. The suction seal is improved by shaving the area around the bite and coating the suction cup with a small amount of petroleum-based ointment. Viperid bites leak blood and serosanguinous from the fang marks. Be prepared to empty fluid from the suction bell and reapply suction regularly. The Extractor may prove effective for the removal of elapid and Australian snake venoms as well. The shorter fangs of these snakes result in the superficial deposition of venom-potentially resulting in a more efficient retrieval of venom with negative pressures. There are numerous cases of oral suction being used on snakebite as are there cases where the individual providing the suction experienced mild symptoms of venom poisoning. The risk-benefit associated with oral suction remains controversial .

Pressure wraps and lymphatic-occlusive dressings;
A circumferential pressure wrap has been shown to delay the spread of hydrophobid (Australian snake) venom from the bite site. This technique involves the application of crepe or elastic bandage directly over the bite, and continuing the wrap proximally up the extremity. The extremity is then splinted as described above. The principle for this technique is attributed to compression of subcoutaneous microvasculature and lymphatics, thereby compartmentalizing venom from spreading[16]. The pressure wrap was also found to be effective at reducing the spread of crotalid venom (C. adamantus) in animals. Unfortunately, following removal of the pressure wrap, the animals developed sudden elevations in prothrombin and partial thromboplastin time and the affected extremity developed massive edema, ecchymosis and discoloration [17]. Currently the pressure wrap is not recommended for Viperid envenomation[17].

For Viperid venoms (pit vipers, old world vipers), the spread of venoms can be reduced by the application of a partially-occlusive dressing that impedes lymphatic flow. The lymphatic "tourniquet" should be placed immediately proximal to the fang marks. The dressing should be wide enough to distribute pressure evenly, and should not interfere with venous return. The tightness of the lymphatic dressing should be monitored as tissues

swell. If the dressing becomes too tight, a second dressing should be applied proximal and superior, prior to removal of the original dressing. Removal of rings, watches, and bracelets prevent loss of perfusion as tissues swell. Determine progression of injury by comparing cicumferential measurements at the bite site with measurements on the unaffected extremity, and recording changes over time.

Snake venom ophthalmia; care for patients sprayed or "spat" at by cobras require aggressive irrigation of the eye and mucous membranes with water or other safe fluid. The eye should be closed with a soft dressing and a topical antimicrobial should be applied. Patients should be quickly transported to a hospital and the eye should be examined using fluorescent stain.

Supportive Care

Nephrotoxic and myotoxic venoms; patients bitten by certain rattlesnakes (*C. horridus, C.d. terrificus*) several vipers (V. russellii spp), cobras, sea snakes, and Australian snakes may develop rhabdomyolysis. Early symptoms include headache, fever, vomiting and thirst. Myoglobinuria appears between 2-8 hr of the bite. Complications include renal failure and cardiac arrest from hyperkaelemia. Some venoms (*C. d. terrificus, V. russellii, Bothrops jararaca*) can induce renal failure directly or through vasoconstriction, DIC, and ischemia to the kidneys. Forced drinking (up to 1 lt/hr) and parenteral solutions have been recommended to maintain renal function. Suspected rhabdomyolysis and the presence of myoglobin in urine has been treated with forced alkaline diuresis.

Circulatory Collapse; envenomated patients who are hypotensiven should be treated for hypovolemia if possible. Hypotensive patients envenomated by *Vipera russelli* and have been successfully treated with dopamine alone.

Paralytic snakebite; due to the low avidity of snake neurotoxins to targets, *all paralytic effects of snake venom are reversible with time*. Patients have recovered after manual ventilatation (by relays) for 30 days. Neurotoxic symptoms from a variety of elapid venoms (cobra, krait, mamba) have improved with Neostigmine and related anticholinesterases. The current recommendation for neurotoxic envenomation is edrophonium chloride (10 mg IV) after atropine (.6 mg) [20].

Antivenin;

Definitive treatment of envenomation requires rapid administration of the appropriate antivenin. Unfortunately, the majority of antivenins are of equine origin, and therefore prone to induce hypersensitivity reactions. Lyophilized antivenins are also dilute, sometimes requiring tens of vials to reverse systemic symptoms. The use of a single vial incurs all the risk of dangerous side effects with marginal benefit and has been likened to "a sniff of penicillin". Effective immunotherapy also requires that the antivenin be administered in high bulk intravenous diluent. The effectiveness of lyophilized antivenins is short lived without continuous cold chain. These shortcomings to their use in the field, and the effectiveness of prevention, together preclude the addition of antivenin in all but the most elaborate field kits. Paramedical personnel belonging to select Indian and Thai military patrols at increased risk of snakebite carry a antivenin kit containing 6-10 vials of polyvalent antivenin against local species, I.V. kits and supportive medication. The Thai kit weighs approximately 2 kg, and 1 kit is supplied for every 70 soldiers. More kits are carried if the risk of snakebite is felt to be greater. Several groups are currently working to develop antivenins of greater stability, fabulated (Fab'_2 and Fab') antivenins, and human monoclonal antivenins.

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Table I
Snake Venom Enzymes

enzyme	effect
phospholipase A_2	presynaptic neurotoxic activity (inhibit release of acetycholine at neuromuscular junctions) damages nerve fibers releases histamine damages vascular endothelium lyses rbcs, leukocytes activates platelets myonecrotic (skeletal)
hyaluronidase	"spreading factor"
L-amino acid oxidase	proteolytic
phosphomono and phosphodiesterases	inhibit protein synthesis cytotoxic via accumulation of toxic fatty acids

Table II

Elapidae & Hydrophobidae toxins

cobras, kraits, mambas, coral snakes, Australian snakes and Laticauda sea snakes

I. phospholipase A_2 toxins :

a. presynaptic β-neurotoxins including;

β-bungarotoxin	Bugarus spp. (kraits)
crototoxin	C. durissus terrificus (So. Am. rattlesnake)
notexin	Notechis scutatis (tiger snake)
notechis II-5	
Scutoxins A and B	
taipoxin	Oxyuranus scutellatus (taipan)

b. myonecrotic toxins

II. disulfide bridged (peptide) toxins (comprised of peptides with 60-62 or 66-74 amino acid residues linked by 3-5 disulfide bridges) :

a. the postsynaptic α-neurotoxins including;

α-bungarotoxin	Bungarus multicinctus (Chinese krait)
erubotoxin	Laticauda spp. (sea snake)
cobrotoxin	Naja naja spp. (monocellate cobra, chinese cobra)
notechis III-4	Notechis

b. membrane toxins

WILDERNESS MEDICAL LIABILITY

LEARNING OBJECTIVES

Following this presentation, participants will have:

1. Acquired a basic understanding of the general legal principles of medical malpractice claims and their application to expedition settings.

2. Become aware of the areas in which a physician is exposed to a medical malpractice claim in an expedition setting.

3. Obtained suggestions and recommendations to help lessen the physician's exposure to liability.

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WILDERNESS MEDICAL LIABILITY

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I. Introduction

Participation by a physician in an expedition or other wilderness outing unquestionably exposes the physician to the risk of liability for professional negligence. The probability of a claim being made appears to be small, probably because the type of people who participate in such activities know they are assuming some risk and are more likely than the general public to accept responsibility for the consequences. The lack of cases reported in United States courts supports this conclusion. While the probability of a claim may be small, the financial consequences of a successfully prosecuted claim could be devastating. If the claim is covered by your professional malpractice insurance, the insurer will pay to defend the claim as well as to indemnify you for any resulting judgment or settlement, up to your limit of liability. If your insurance does not cover the claim, you will bear the costs of defense and be personally liable for any resulting judgment or settlement.

Many of you may recall that there was a death on the 1986 SINO-USA Upper Yangtze River Expedition. The expedition still photographer died at approximately 14,500 feet on the Tibetan Plateau from the combined effects of high altitude pulmonary edema and cerebral edema. His widow sued the expedition leaders, the documentary film company and the film company's president. The cost of successfully defending the expedition leaders alone was approximately $175,000 and that amount did not include some substantial costs assumed by the film company, such as expert witness fees. The expedition doctor was not sued, but his conduct was a central issue in the case.

The purpose of this paper is to highlight the general concepts of medical malpractice liability and apply those concepts to the activities of a physician who participates in an expedition or other wilderness outing. For ease of presentation,

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[1] John W. Knottnerus, an attorney employed in the author's firm, provided substantial assistance in the preparation of this paper.

the example of an expedition physician will be employed. The principles are applicable to other less organized situations as well.

This paper is not a comprehensive review of malpractice liability, which varies from state to state and country to country. The purpose of this paper is to make you think about your exposure to claims and provide suggestions on how to avoid them.

II. Basics of Medical Malpractice Liability

A brief explanation of the general legal principles involved in a generic medical malpractice claim is necessary in order to appreciate and understand the specific issues raised by a physician's involvement in an expedition.

A. Elements of a Claim

A professional malpractice case involves elements generally applicable in any tort or negligence action. In the normal tort action, the plaintiff[2] must prove: (1) a duty on the part of the defendant to conform to a specific standard of conduct for the protection of the plaintiff; (2) breach of that duty by the defendant; (3) the breach of the duty was the actual and proximate cause of plaintiff's injury; and, (4) damages.[3] The factor that distinguishes medical malpractice cases (as well as other professional malpractice cases) from most tort actions is the "standard of care" to which the defendant's conduct is compared.

A general duty of care is imposed, in tort law, on all human activity. However, if a defendant has knowledge, skill, or even intelligence superior to that of the ordinary person, the law will demand of that defendant conduct consistent with such attributes.[4] Professional persons (doctors, lawyers, engineers, etc.) in general, and those who undertake any task calling for

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[2]Note that the "plaintiff" is not always the person to whom the professional provided service. For example, a spouse, children, parents, or a decedent's estate may be the ones bringing an action against a doctor for the actions the doctor took in diagnosing and treating the patient. Therefore, even if the participant was willing to take the risk and was satisfied with your actions, the family left behind may have different ideas.

[3] Prosser & Keeton on Torts § 30 (5th Ed. 1984).

[4] Restatement (Second) of Torts § 289, comment m.

special skill, are required not only to exercise reasonable care in what they do, but also to possess a standard minimum of special knowledge and ability.

B. Physician's Standard of Care

The standard of reasonable conduct for medical professionals is the range of accepted responses to a given set of circumstances by others with a similar practice in the same or similar community--commonly known as the "community standard."[5] With respect to "the range of responses," the law contemplates that in any particular set of circumstances there may be more than one approach that would meet the physician's standard of care. For example, even if another physician would not respond to a given set of circumstances in the same manner, neither physician is negligent if both responses are within the range of accepted responses in same or similar community.[6] An example would be surgery to treat a thyroid problem instead of drug therapy.

The physician's community and his or her practice can be critical to a determination of liability. One who holds him- or herself out to the public as a specialist in a particular field of medicine is held to a higher standard of care than a general practitioner who renders the same type of service as a part of his or her practice.[7] The specialist must conform to the standard of that segment of the profession in which the physician specializes.

The place where the physician practices is also relevant to the community standard. In some courts, a physician is entitled to have his or her conduct tested by how others in the same or similar places would conduct themselves.[8] In other words, the law recognizes that a general practitioner in New York City may respond differently than a general practitioner in Two Dot, Montana (U.S.A.).

The physician's medical community, geographic community, and specialty (if any) define the standard of care to which the physician will be held. The law sets the standard of care and

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[5] McCoid, The Care Requirement of Medical Practitioners, 12 Vand. L. Rev. 549 (1959).

[6] See e.g., Willard v. Hutson, 234 Or. 148, 378 P.2d 966 (1963).

[7] Prosser & Keeton on Torts § 32 (5th Ed. 1984).

[8] See e.g., Reeg v. Shaughnessy, 570 F.2d 309 (10th Cir. 1978).

negligence is determined by whether the standard is met as the result of what one does or does not do, rather than by what one knows or does not know.

C. Informed Consent

Aside from liability arising from actions that may fall outside the "community standard," there is another area of potential liability called, "informed consent." As a general rule, a physician has a legal duty to warn a patient of the risks of a procedure and advise of alternative approaches if the evidence discloses that: (1) the risk of injury inherent in the procedure is material; (2) there are feasible alternative courses of action available; and, (3) the plaintiff can be advised of the risks and alternatives without detriment to his or her well-being.[9]

The patient can bring an action against the physician if the physician did not obtain informed consent. Implicit in the cases involving informed consent is the requirement that the patient establish that he or she would not have consented to the procedure if adequately advised.[10]

III. Areas of Exposure in Wilderness Medicine

With some of the basic legal principles applicable to medical malpractice cases in mind, the circumstances unique to expedition medicine, under which these principles will be applied, can be outlined. The potential theories of negligence that might be advanced by a patient's attorney are endless. However, experience from the Yangtze River case and common sense reveal several likely areas of exposure.

A. Pre-Expedition Screening & Examination

A physician's exposure to a medical malpractice claim may arise even before the expedition departs. Most prudent expedition leaders will require that all participants, as a condition to participation, obtain a physical and provide a medical history. As the expedition physician, you may be asked to conduct the physicals, which would include a review of the participants' medical histories. More likely, the expedition physician will be responsible for reviewing the results of the physicals and the medical histories. In most cases, prudence

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[9] 2 Louisell & Williams, Medical Malpractice § 22.01 et. seq. (1989).

[10] Waltz and Scheuneman, Informed Consent to Therapy, 64 NW U. L. Rev. 638 (1969).

will require that the expedition physician, who is fully aware of the specific health risks of the expedition, prepare a detailed questionnaire for each participant that seeks information pertinent to the specific risks.

If a participant suffers serious injury, illness or death from the complication of a pre-existing condition, which should have been a contraindication to participation, the physician who performed the physical and the reviewing or expedition physician are exposed to a claim. Likely theories are failure to inquire about health problems later deemed relevant to the known risks of the expedition, failure to recognize the risks, failure to report contraindications to the expedition leaders and failure to advise the participant of contraindications.

B. Expedition Diagnosis & Treatment

A physician's exposure to malpractice claims for misdiagnosis and treatment during the expedition encompasses many of the same areas for which the physician would be exposed to a malpractice claim in his or her everyday practice. Basically, anything that went "wrong" in the diagnosis and treatment phases could be a potential malpractice claim. The specific legal issues raised by diagnosis and treatment during the expedition are discussed below.

C. Equipment & Supply Deficiencies

The expedition physician is customarily called upon to assemble, sometimes at his or her own expense, the medical "kit" for the expedition. While the expedition leaders or other participants may have some suggestions based on past experience, the physician, for the most part, exercises unreviewed discretion.

If a participant is injured, or succumbs to an illness, and the right type or quantity of supplies or drugs are not available, the physician is exposed to a claim. Because many expeditions today are equipped with sophisticated equipment, the expectations of participants and their families are that the medical supplies will likewise be "state of the art." Remember that in the case of a death, the claim will likely come from a spouse or parent. Their expectations are frequently higher, usually due to assurances made by the now-deceased participant, and they will not understand or accept the practical expedition reality that there are limits on what can be carried, preserved or possibly even taken into a foreign country.

The standard of care imposed on the physician assembling the medical kit is more likely to be based on what physicians have taken on similar expeditions to similar locales than on a survey of physicians in the community where the expedition physician

practices. Consultation with those who have gone before you and equipping accordingly will lessen your exposure. Document your communications with those physicians.

D. Informed Consent

The second major branch of medical malpractice liability is informed consent. Under certain circumstances, an expedition physician may have a duty to advise an injured or ill participant of the risks and alternatives of a proposed treatment or procedure, before administering it. There is no logical impediment to applying this concept to treatment in the expedition setting in exactly the same form it is applied in a hospital before surgery.

IV. Legal Issues Raised by Expedition Medicine

We have not found any reported cases involving claims of medical malpractice on expeditions, so there is not a great deal of guidance on how the courts would deal with such a case. Although there are no definite answers, in the legal sense, the following are some of the legal issues raised by expedition medical care.

A. Standard of Care

The physician's general duty of care, under the community standard, takes into account the physician's area of practice, including, in some states, the geographical location of his practice. With respect to the physician who performs the pre-expedition physical, there should not be an issue with respect to the applicable community, particularly if it is the participant's personal physician. The standard of care applied to the expedition doctor is more difficult. The standard could be drawn from the community where the physician practices, from the conduct of expedition physicians on similar expeditions to similar locales or from a specialized practice, such as high altitude medicine or the treatment of hypothermia.

Advances in the field of wilderness medicine, including the existence of physicians whose entire "practice" is limited to wilderness and expedition medicine, raises the issue of whether the physician is a specialist, which is a familiar concept in United States law relating to the standard of care. One who holds him or herself out to the public as a specialist in a particular field of medicine is held to a higher standard of care than a general practitioner who renders the same type of service as a part of his or her practice. Therefore, the expedition physician may be subject to the standards, knowledge and skill of other physicians specializing in wilderness medicine, or certain discrete areas, such as high altitude medicine, whether or not

the particular physician actually has that knowledge or skill. If you participate in a climbing expedition, as the expedition physician, you may be judged by a standard of care developed from the work of people like Dr. Hackett, if the work is published and a reasonable inquiry would have discovered it. In other words, you should attempt to familiarize yourself with the state of the art on medical issues likely to arise, given the specific nature of the expedition.

B. Good Samaritan Statutes

Good Samaritan statutes offer total immunity from liability--even if the physician fails the community standard. The theory of Good Samaritan immunity has been accepted in all of the United States.[11] All states have legislatively adopted some type of statute exempting certain bystanders or "rescuers" from all liability for any negligent act or omission arising out of their rescue attempts. Many, if not all, of these statutes were passed for the express purpose of protecting physicians who altruistically render services in emergency situations. Some states also extend the protection to other medical and emergency personnel (nurses, EMTs, paramedics, firemen, etc.)[12] and some to anyone rendering emergency assistance.

Every state's statute is different and the applicable statute must be consulted for its exact language and requirements. The statute most likely to apply is that of the state where you practice, the state where the participant resides (or resided) or the state where the injury occurred, and not the law of a foreign country. Even though the statutes differ, some broad statements with regard to the conditions placed on a physician's immunity under most Good Samaritan statutes can be made.[13] First, many statutes require a good faith state of mind on the part of the rescuer rendering emergency assistance. Second, many statutes require that the care be rendered "gratuitously" which has been interpreted to mean that there is no pre-existing duty to render such service. Third, some

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[11] See, Good Samaritan Laws--The Legal Placebo: A Current Analysis, 17 Akron L. Rev. 303 (1983).

[12] For an example of an statute whose coverage extends to nurses, see Cal. Bus.&Prof. Code § 2727.5 (West 1974). For a complete outline of every state's statute, see the Akron Law Review article cited in the prior footnote.

[13] These generalizations are drawn from: Annotation, Construction and Application of "Good Samaritan" Statutes, 68 ALR4th 294 (1989); 2 Louisell & Williams, Medical Malpractice, chptr 21 (1989); and, the Akron law review article cited above.

statutes specify or limit the places in which the emergency aid must be given (i.e. "at the scene") for immunity to attach. Fourth, the statutes will not afford immunity to a physician who has been grossly negligent, reckless, or has engaged in willful or wanton misconduct. Finally, the emergency situation must not have been created by the person rendering assistance.

For the most part, it seems that the conditions imposed by most Good Samaritan statutes could be met by the expedition physician. This is particularly true in situations where an unexpected accident or injury occurs. The most apparent reason to prevent immunity from attaching is the requirement that the physician perform the service gratuitously with no pre-existing duty.

Even though the physician may not have been "hired" as the expedition physician and receives no compensation, the presence of a physician on most expeditions is no accident. Participants are told, or may assume, that you are there to provide them medical services. Expedition leaders and participants expect that if something happens the physician will act. The purpose of the physician's participation and the expectations of the expedition members may give rise to a pre-existing duty to render services. If a court or jury found that duty to exist, the immunity offered by a Good Samaritan statute might not apply.

C. **Informed Consent**

As described earlier, the informed consent doctrine centers around the physician's duty to advise the patient of the nature and risks of a proposed medical treatment, in order to enable the patient to make an intelligent and informed decision as to the treatment.

Two exceptions to the physician's duty to inform and obtain consent are applicable to the expedition setting. First, the duty is not applicable in an emergency situation where the patient is unconscious or otherwise incapable of consenting and the harm from a failure to treat is imminent and outweighs any harm threatened by the proposed treatment.[14]

The second exception, often termed the "therapeutic privilege," eliminates the duty to inform and obtain consent in situations where the physician believes that the patient's emotional state is so fragile that the very act of revealing the

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[14] 2 Louisell & Williams, Medical Malpractice § 22.06 (1989).

risks may: (1) cause the patient to refuse the treatment that the physician thinks is needed; or, (2) create physiological reactions that would increase the dangers of the procedure.[15]

The usual situation for application of this doctrine, the severely emotional patient, is not likely to present itself on an expedition. It is more likely that a physician will be presented with the risk of harm to other members of the expedition, and to himself or herself, than a risk to the "patient" if treatment is refused. This problem is very real in the expedition setting, has not been discussed as a consideration in the reported cases, and raises ethical problems which are beyond the scope of this paper.

E. **Insurance Coverage**

As with any other situation, the expedition physician should be concerned with whether his or her medical malpractice insurance policy will cover a claim arising from acts or omissions on the expedition. Your policy should be carefully examined before participating in any expedition. The first thing to look for is a geographical limitation. The second is language in the policy suggesting that only activities normally associated with your stated practice are covered. Careful attention should be paid to all exclusions and endorsements, which frequently modify the terms of coverage otherwise extended in the main insuring agreements.

If you have doubts about coverage, consult a lawyer before you consult your insurance agent. Disclosure to the agent may result in issuance of an exclusion defeating any coverage for the expedition. This will eliminate a later argument that an ambiguous policy does provide coverage.

You should also find out if the expedition leader has obtained insurance specifically for the expedition. If so, obtain a copy and determine, with the help of a lawyer if needed, if the policy covers you. If it does not, request to be added as an additional insured.

D. **Releases of liability**

The Yangtze River case was won on the strength of an assumption of risk, or release, document signed by the deceased participant. The expedition leaders retained a lawyer to prepare the release and required each participant to sign one as a

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[15] 2 Louisell & Williams, Medical Malpractice § 22.06 (1989).

condition of participation. The Yangtze River release did not expressly release the expedition physician, but it could have been drafted to do so.

You should discuss releases with the expedition leader and require, as a condition of your participation, that each participant execute a release that covers you as well as the leader. There is no standard form release. Be aware that releases from liability, especially for physicians, are not favored in the law and will not automatically apply to protect you from a claim in every case. You and the leader should retain a lawyer to assist you in drafting the release.

V. Conclusion

The threat of liability should not chill your participation in expeditions or other wilderness activities. Rather, if you keep the issues and suggestions presented in this paper in mind when you are preparing for an expedition or other activity, you will lessen, or perhaps eliminate, your exposure to claims.

DEALING WITH DEATH: THE PSYCHOLOGY OF RESCUE

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INTRODUCTION:

This presentation assumes that the people involved in deadly situations are generally in good health, both physically and mentally, before the mishap. It will deal with the victims--those saving themselves, or needing rescue--and with the rescuers themselves.

THE WILL TO SURVIVE

Survival is one of the basic instincts in every animal, including humans. If we are physically threatened, we respond instinctively in order to survive, by fight, flight, or fright (freezing). If the thing we do (our behavior) helps us survive, we remember it and tend to use it if the same situation threatens us again. This is learning by experience.

Another way animals acquire survival behavior is through simulation; we learn through planning and practicing. Much of the playing that young animals do is for this purpose.

One of the unique things humans do is to learn survival behavior before the event by mental rehearsal--learning through imagination. This includes listening to how others have dealt successfully with potentially deadly experiences, or imagining how we would deal with such a situation. Thus, we can "remember" how to deal with things that we have never actually faced, because we have thought them through ahead of time. Aviators do a lot of this.

However, one element which is impossible to simulate, rehearse or plan for is the emotional component of danger, fear. Fear is to the mind as pain is to the body; it repels us from danger. Our natural instinct is to avoid things that hurt us or frighten us. Fear changes the way we think, feel and act, acting partly through the mind, but mostly through the autonomic nervous system (ANS). Of course, we can use will power to overcome these natural aversions, but doing so takes a certain amount of psychological energy.

Some people seem to have what Tom Wolfe called "the right stuff," the ability to continue to think clearly and act correctly during a severely threatening situation, even one which is new to them. No one knows how much of this ability is inborn, and how much is learned. This quality is difficult to predict or to identify, except by observation. At any rate, these people can think under stress and control their behavior in situations where most people might stop thinking clearly and, instead, freeze up or panic. Panicky behavior involves thoughtless and possibly counterproductive behavior that may actually increase the danger. For example, a person may panic under fire in combat and stand up to run away, thus causing needless exposure to enemy bullets.

Danger may not only be immediately threatening, but it may also involve a longer lasting situation which requires a more enduring response, as was the case with the woman with the broken leg who was rescued from deep in a cave in April 1991. After dealing with the initial mishap, the person may be physically exhausted, injured, and utterly alone in a hostile environment. Instinct and immediate response have worked so far, but now a new set of skills is called for, involving very clear thinking: assessment of the situation, control of emotions and actions, and planning a series of moves as in a chess game. The person's status is far from the usual, familiar situation, and the person may feel overwhelmed by the need for innovation and flexibility, and may be tempted to give up or to wait passively for whatever happens next. Indeed, "lying low" may be the best course of action in such a situation, but, once rested, the person must start thinking ahead of the situation again, planning moves, considering "What will I do if.such-and-such happens?"

In this situation, prior planning, listening to others' experiences, mental or actual rehearsing, and other such preparation may pay off with cool, correct action in the long-term survival situation. The will to survive has involved both instinctive and cognitive mental processes.

Once the person is safe, through his or her own actions or through the efforts of rescuers, some psychological reactions may occur. One common reaction is to deny the emotional component, to "get right back on the horse that threw you" and prove that the individual, no longer a victim, is back in control. This may well be effective, and is much used by aviators who fly again as soon as possible after a crash or other brush with death. The problem with such denial, however, is that the individual may feel that any emotional reaction is somehow unworthy, and that one should be cool and emotionless in every situation. Thus, one may actually repress emotions, such as survival guilt, which are so strong that they will affect other areas of life, causing later

problems such as irrational fears (phobias), acute stress reactions, or psychosomatic symptoms.

How should one deal with an individual who has survived a potentially lethal situation? Using an outline form, we will consider three elements: Early Detection of Problems, Rapid Treatment, and Rehabilitation.

I. EARLY DETECTION OF PROBLEMS IN VICTIMS

A. Understand that fear is normal, and serves the same purpose for the mind that pain does for the body. A fearful response to a real threat is not the same as acute anxiety or mental illness.

B. Reactions may be adaptive or maladaptive, immediate or delayed, and acute or chronic. Maladaptive behavior in otherwise uninjured victims may require some therapeutic response.

C. Reactions may include anxiety, disorientation, misunderstanding, confusion, , withdrawal, irritability, increased startle reaction, difficulties with memory, concentration or sleep; also psychophysiological symptoms. These may simply represent normal responses to an abnormal situation, and usually require merely supportive care.

D. Mishap victims may have brief maladaptive reactions of psychotic proportions, but these are usually transient.

II. RAPID TREATMENT

A. Avoid creating an unnecessary "patient" role for victims.

B. Provide basic needs: shelter, protection, warmth, food, comfort and support. Allow regressive dependency to run its course. Use short-acting sedation (temazepam, for example) only if necessary, since medication reinforces the "patient" role.

C. Provide accurate information about the situation.

D. Ventilate emotions--describing what happened helps victims get their own thoughts in order.

E. Allow or encourage victims to express their fears ("I don't know how you feel, but I would be really thinking hard about almost dying. What are you telling yourself about the accident?"). Reassure that such thoughts and feelings are normal. Explore the realities of what happened, and how they survived.

F. If psychological defenses were overwhelmed, expect to see either denial, depression, loss of sense of humor, withdrawal and psychological numbing ("I don't feel anything") or intrusive symptoms such as reliving the experience, flashbacks, nightmares, or agitation. These polar reactions, withdrawal and agitation, may alternate for a while and

then begin to fade away as the individual comes to grip with what happened. Professional help may be useful during this period.

G. In multiple-victim mishaps, let victims help each other as they are able.
   1. This establishes reassuring adult roles in place of dependent ones.
   2. The volunteer effort saved may be better used elsewhere.

H. Brief reactive psychosis.
   1. Produced by an overwhelming stressor.
   2. Symptoms include emotional turmoil, and at least one psychotic symptom: incoherence or loosened associations, grossly disorganized behavior, delusions and/or hallucinations.
   3. Again, avoid use of psychotropic medications if possible, since they reinforce the "patient" role, and may mask neurologic symptoms. Use short-acting medications such as haloperidol if necessary.

I. Physically injured patients should be followed psychologically in a consultation/liaison model with their primary physicians, since they will have had the same stressors and losses.
   1. Psychotropic medication may mask psychological symptoms for a while, and may interfere with the normal grieving process. It may also mask head injury symptoms.
   2. Injured patients need ventilation too, but not excessively. They may need to be buffered from the press, dignitaries, and curious staff.
   3. Bad news about family members or others involved in the mishap should be handled carefully and thoughtfully.

III. REHABILITATION

A. Mental health consultants may switch from crisis intervention to treatment of post-traumatic stress disorder as time passes.

B. Although careful early follow-up may diminish the incidence of Post Traumatic Stress Disorders, case-finding efforts by the Red Cross and similar agencies may be useful in reaching victims who were not primarily involved, such as family members of actual disaster victims.

C. All victims may have troubling symptoms for a while, but many will handle these through normal grieving processes. Symptoms may alternate between periods of psychic numbing, which protects from overwhelming emotion, and intense emotional pain, which may feel intolerably threatening, as if the victims were "losing their minds" or "going crazy."

D. A stable environment, allowing the processing of the situation in small emotional increments and accepting the ups and downs of the recovery period, helps people in processing the disaster and the losses. Expect some inappropriate anger, often directed at rescuers or helping agencies.

1. Therapy should be aimed at re-establishing control over one's own life, and dealing effectively with the post-disaster realities.
2. Tearfulness, both expected and unexpected, is part of the healing, and will probably continue for up to a year. Frequency and intensity will diminish with time.
3. Through talking about their losses, victims may relieve some of the feeling of isolation in grief. Like tears, the need to ventilate and thus seek support will diminish after a year or so.
4. Dreams about what happened may represent one means of natural desensitization to the event.
5. Psychosomatic symptoms, including appetite, libido, or sleep patterns, may be disturbed, but should gradually return to normal. Memory and concentration may go through similar disturbances. Intrusive thoughts should also gradually lessen, although all these symptoms may be exacerbated by anniversaries, sights, sounds, and other associations. Victims should be made aware of these and similar patterns of reactions as normal phenomena, similar to somatic healing processes, and should be reassured that they are not signs of mental illness, weakness, or lack of moral fiber.

E. During this interval of recovery, victims should be made aware of a need not to self-impose additional stresses. For example:
1. Respect emotional waves. If one occurs, for example, while driving, pull over and stop as soon as safety allows, and wait until it passes before resuming driving. Allow a little extra time. Travel with a friend, if possible.
2. Be especially careful if work requires alertness; e.g., around machinery.
3. Watch out for physical health; stress can affect resistance to disease. Consult a physician for somatic symptoms. However, keep in mind that grief can also be a factor in such symptoms. Beware of overmedication.
4. Delay making decisions which will cause added stress, if possible. If not, separate such decisions in time. (Therapists should keep the Holmes-Rahe Life Change Events tables in mind.)
5. Share problems, concerns and cares with responsible family members, trusted friends, and/or mental health professionals.

RESCUERS

A considerable literature exists on the care of rescuers, and others who may have been only peripherally involved in the disaster, such as reporters, morgue attendants, and medical staff. A sensitive and professionally moderated post-incident debriefing procedure may help to elicit troubling thoughts and emotions which otherwise would not be recognized for what they are. This process should not be undertaken without prior specific

training, and should be carried out by persons who were not involved in the primary disaster or response. See the articles by Jones and by Mitchell in the bibliography for further details.

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INSTITUTIONAL RESOURCES

Center for Mental Health Studies of Emergencies
National Institute of Mental Health
Parklawn Building, Room 6C 12
5600 Fishers Lane
Rockville, MD 20857
Tel. 301-443-1910

National Organization for Victim Assistance
717 D. Street, N.W.
Washington, DC 20004
Tel. 202-393-6682

The Society for Traumatic Stress Studies
P.O. Box 1564
Lancaster, PA 17603-1564
Tel. 717-396-8877

Disaster Preparedness in the Americas
Pan American Health Organization
525 23rd Street, N.W.
Washington, DC 20037

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WILDERNESS MEDICINE -- JULY 1991

VENOMOUS ARTHROPODS

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BIOLOGY

The phylum Arthropoda is the largest phylum in the animal kingdom. It contains the spiders, scorpions, insects, ticks, kissing bugs, water bugs, caterpillars, moths, butterflies, grasshoppers, centipedes, and millipedes, among others. Some arthropods sting (bees, ants, scorpions, etc.), others bite (spiders, centipedes, kissing bugs, etc.), while still others discharge a secretion that is toxic (millipedes, caterpillars, etc.). Although the number of arthropods that are sufficiently venomous to be of a potential danger to humans is not known, these animals are implicated in far more bites and stings than all other phyla combined. Almost all of the 20,000 species of spiders are venomous, but luckily for man, only relatively small numbers have fangs long and strong enough to penetrate the human skin. There are some 500 species of scorpions and all are venomous, although only a small number are of a danger to humans. In the order Hymenoptera, the bees, wasps, yellow jackets and ants, numerous species are involved in serious envenomations. Among the other arthropods are numerous animals of medical importance.

The number of deaths from arthropod stings and bites is not known, nor do most countries keep records of the incidence of such injuries. In Mexico, parts of Central and South America, and in North Africa, deaths from scorpion stings may exceed several thousand a year. Spider bites probably account for no more than 200 deaths a year, worldwide. The number of deaths from arthropod bites or stings in the temperate countries is far greater than the number of deaths from snakebite. Almost all of these deaths, however, are anaphylactic in nature. In the underdeveloped countries of the tropics, a far greater number of deaths from arthropod bites or stings are due to the direct effects of the venom.

CHEMISTRY AND PHARMACOLOGY

The venoms of the various arthropods are very different, and, if the spider venoms so far studied are an indication, these poisons may prove to be more complex than originally suspected. For instance, the venom of the black widow spider (*Latrodectus mactans*) contains at least 15 proteins, and preliminary work on the brown spider (*Loxosceles*) as well as other spider venoms, indicates that they may all be equally as complex. Like the snake venoms, the arthropod poisons exert their deleterious effects at the cellular level, and since some arthropods bite while others sting, the passage of the toxin across membranes, its absorption at specific receptor sites, metabolism and excretion are quite different, reflecting the teleology and evolution of the poison. The interested reader will find the texts by Bettini [1] and Maretic and Lebez [2] particularly important with respect to the composition and modes of action of arthropod venoms.

SPIDERS

There are at least 200 species of spiders that have been implicated in significant bites on humans [3]. Unfortunately, spiders often get the "bum rap" for the bites or stings of other arthropods, or for dermatological diseases traceable to etiologies other than arthropods. Of approximately 600 suspected spider bites seen in one series of cases, excluding those caused by the widow spiders, 80% were found to be caused by arthropods other than spiders, 10% were probably caused by *Loxosceles* sp., and 10% by other disease states. The arthropods most frequently involved in the misdiagnoses were ticks (including their embedded mouth parts), kissing bugs, mites, bedbugs, fleas (infected flea bites), lepidopterous insects (lepidopterous blisters), flies, beetles (vesicating beetles), water bugs, and various stinging Hymenoptera. Reviews on spider venom poisoning will be found in Bettini [1], Maretic and Lebez [2] Southcott [4], Russell [5], and Gertsch [6].

Lactrodectus Species (Widow Spiders)

These spiders are commonly known as the black widow, brown widow, or red-legged spider, depending on the species. They have many other common names in English: hourglass, poison lady, deadly spider, red-bottom spider, T-spider, gray lady spider, or shoe-button spider. The eastern black widow spider is *L. mactans*, whereas the western is *L. hesperus*.

Although both male and female widow spiders are venomous, only the latter have fangs large and strong enough to penetrate the human skin. Mature females range in body length from 10 to 18 mm, whereas males are from 3 to 5 mm.

In most patients, there is a history of having received a sharp, pinprick-like bite, but in some cases the incident is so minor that it goes unnoticed. The initial pain is sometimes followed by a dull, occasionally numbing pain in the affected extremity, and by pain and some cramps in one or several of the large muscle masses. Muscle fasciculations can sometimes be seen within 30 minutes of the bite. Sweating is common and the patient may complain of weakness, as well as pain in the regional lymph nodes. Lymphadenitis is frequently observed. Pain in the low back, thighs, or abdomen is common and rigidity of the abdominal muscles is seen in most cases in which envenomation has been severe. Severe paroxysmal muscle cramps may occur, and arthralgia has been reported.

The extent of the local tissue reaction around the bite may vary, depending on the species involved. In most cases in the United States, there is no reaction at the bite site and the puncture wounds may be impossible to find. In some cases, there is an immediate 2 to 4 mm blanched area around the puncture wounds, surrounded by a slightly erythematous area, which in time becomes pallid. This may persist for 20-30 minutes. In children, there may be localized diffuse edema.

Local sweating may be evident, although the erythematous plaque usually remains dry. Piloerection around the bite area and local changes in the sensitivity of the skin have been reported following the bites of some species. Opisthotonos, bizarre head and neck movements, elevation of body temperature, mydriasis, increased salivation, anorexia, oliguria, and convulsions have all been reported but are not common. Hypertension is a characteristic finding in moderate to severe poisonings. It may not be evident during the first hour following the bite in untreated cases; it is often present between the second and fourth hours. In some cases, it may develop rapidly and become acute, warranting immediate attention. There may also be

changes in the electrocardiogram, varying from simple ST segment depression and prolongation of the QT interval to a complete block.

There are no first-aid measures of value for the widow spider bites. If the pain is intense, ice can be placed over the wound until the patient arrives at a medical facility. If the symptoms and signs are trivial, no specific treatment is indicated. In most cases, 10 mg calcium gluconate intravenously, or a relaxant such as methocarbomal, by slow push, will relieve the muscle cramps and pain. The calcium gluconate will often need to be repeated at four-hour intervals for optimum effect. Diazepam can be used in the less severe poisonings. Meperidine hydrochloride or morphine sulfate have been used where respiratory depression was not a problem. Simple hot baths may afford relief in some cases. Mild sedation is sometimes indicated. Hypertensive crises are treated with nitroprusside. Atropine, ergot preparations, and physostigmine have all been tried in black widow poisoning but with indifferent results.

The use of antivenin is usually restricted to the more severe poisonings that do not respond to the above measures or when pregnancy is present. Antivenin for *L. mactans* is prepared in Argentina and the United States and is said to mitigate the effects produced by all North, Central, and South American *Latrodectus* species. The author advises one vial of antivenin, given intravenously in 10 to 50 ml of dilutant over a five-minute period.

Loxosceles Species (Violin Spiders)

These spiders are variously known in North America as the fiddle-back, brown, or brown recluse spiders. There are over 100 species of *Loxosceles*. The most widely distributed, *L. rufescens*, is cosmopolitan, and has been imported into North America. *Loxosceles laeta* is chiefly a South American species, but it has been introduced into Central America as well as small areas in Cambridge, Massachusetts, and Sierra Madre, and Alhambra, California. The abdomen of these spiders varies in color from gravish through orange and reddish-brown to dark brown. The violin on the cepnalothorax is brown to blackish and distinct from the pale yellow to reddish-brown background of the cephalothorax. This spider has six eyes grouped in three diads, forming a recurved row. Females average 8 to 12 mm in body length whereas males average 6 to 10 mm. Both male and female spiders are venomous

The bite of this spider produces about the same degree of pain as that of an ant sting, but patients may be completely unaware of the bite. In most cases, a localized burning sensation develops about the injury. This may last for 30 to 60 minutes. Pruritus is often present, and the area begins to appear red with a small blanched area around the immediate bite site. The reddened area enlarges during the subsequent 1-8 hours. It often becomes irregular in shape and as time passes, hemorrhages may develop throughout the area. A small bleb or vesicle forms at the bite site and increases in size. It subsequently ruptures and a pustule forms. The red, hemorrhagic area continues to enlarge, as does the pustule. The whole area becomes swollen and painful, and lymphadenopathy may develop. During the early stages, the lesion often takes on a bull's-eye appearance, with a central white vesicle surrounded by the reddened area, ringed by the whitish or bluish border. The central pustule ruptures and necrosis to various depths sometimes develops. The necrosis can invade the underlying muscle. Systemic symptoms and signs include fever, malaise, stomach cramps, nausea and vomiting, jaundice, spleen enlargement, hemolysis, hematuria, and thrombocytopenia. Fatal cases, while rare, are usually preceded by intravascular hemolysis, hemolytic anemia, thrombocytopenia, hemoglobinuria, and renal failure.

There are no first aid measures of demonstrated value. In fact, all first-aid procedures should be avoided, because the natural appearance of the lesion is most important in determining the diagnosis. An ice pack, however, may be placed over the wound if the pain is severe. Some physicians excise the bite area if the patient is seen within several hours of the bite and there is no doubt of a *Loxosceles* spider being involved. This procedure, however, is becoming less and less practiced in the United States. If a child presents with a significant lesion and/or systemic manifestations, one of several courses can be followed. The patient may be placed on a corticosteroid, such as prednisone 1 mg/kg daily for five days, during the acute phase. If the poisoning is severe, dexamethasone 0.4 mg/kg/day in four divided doses may be given. King and Reese [7] have suggested the use of dapsone for the systemic manifestations of *L. reclusa* bites, whereas Russell and Hansen [8] have had some success with colchicine. The former drug must be used with extreme care. Antihistamines are of questionable value. The antivenins prepared in Peru and Brazil for *Loxosceles* envenomation have not been used to any extent in the United States. In the author's experience, they have not proved of value in *L. arizonica* bites. A species-specific antivenin is currently being evaluated by King and Reese, and early experience indicates its efficacy.

Steatoda Species (Cobweb Spiders)

These spiders, variously called the false black window, combfooted, or cupboard spiders, are abundant in the Old World and reached the Americas through trade sources. These spiders are often mistaken for black widow spiders. The female of *S. grossa* differs from that of *Latrodectus mactans* and *L. hesperus* in that it has a purplish-brown abdomen rather than a black one, and its abdomen is less shiny and more oval than the round abdomen of *Latrodectus*.

The literature on poisoning by these species is scanty. According to Maretic and Lebez [2], bites on humans by *S. grossa* or *S. fulva* could be quite serious. In the United States, however, bites by the former may produce no more than local pain, induration, pruritus, and the near breakdown of tissue at the bite site.

Phidippus Species (Jumping Spiders)

These spiders, variously known as crab spiders or eyebrow spiders, are large eyed jumping spiders, usually less than 20 mm in length, and have a somewhat elevated, rectangular cephalothorax that tends to be blunt anteriorly. In some species, the cephalothorax is larger than the abdomen whereas in the others, the two are about equal. The abdomen is often oval or elongated. There is a great deal of variation in the color of these spiders. This genus includes the heaviest and hairiest of the American jumping spiders.

The bite of this spider produces a sharp pinprick pain, and the area immediately around the wound may become painful and tender. The pain usually lasts 5 to 10 minutes. An erythematous wheal slowly develops. A dull, sometimes throbbing pain may subsequently develop over the injured part but it rarely requires attention. A small vesicle may form at the bite site. Around this is an irregular, slightly hyperemic area,

which in turn may be surrounded by a blanched area tender to touch and pressure. Generally, there is only mild lymphadenitis. Swelling of the part may be diffuse and is usually accompanied by some pruritus. The symptoms and signs usually abate within 48 hours.

Chiracanthium Species (Running Spiders)

The 160 species of this genus enjoy an almost circumglobal distribution, although only four or five species have been implicated in bites on humans. Maretic and Lebez name *C. punctorium*, *C. inclusum*, *C. mildei*, and *C. diversum* as the species most often implicated in envenomations. The abdomen is convex and egg-shaped and varies in color from yellow, green, or greenish-white to reddish-brown, and the cephalothorax is usually slightly darker than the abdomen. The chelicerae are strong and the legs are long, hairy, and delicate. The spider ranges in length from 7 to 16 mm.

The author's experiences with nine bites by *C.* species have been very similar. Like *Phidippus*, but even more so, *Chiracanthium* tends to be tenacious and must sometimes be removed from the bite area. For this reason, there is a high degree of identification of these spiders. The patient usually describes the bite as sharp and painful, with the pain increasing during the first 30-45 minutes. The patient complains of some restlessness, and a dull pain over the injured part, often accentuated by movement. A reddened wheal with a hyperemic border develops. Small petechiae may appear near the center of the wheal. The injured area is tender to touch and pressure. Skin temperature over the lesion is often elevated. Lymphadenitis and lymphadenopathy may develop. Vital signs are normal, as are blood findings. Abdominal cramps may develop in patients in whom the bite has been on the lower extremity, and there may be some diarrhea. Pruritus is common.

Lycosa Species (Wolf Spiders)

These relatively large spiders are variously known as hunting spiders or wolf spiders. They vary in size from the 5 mm *Piratas* to the 5 cm *Lycosa*. They have an oval abdomen and a large elongated cephalothorax, which is often elevated and narrow anteriorly. On the lower part of the face, set close together, is a row of four eyes pointing forward and slightly to each side. Above these are two very large eyes that point forward, and further back are two large eyes pointing upward. The legs and chelicerae are robust. These spiders vary in color from an abdomen that may be yellow to brown with a dark brown to dark-brown cephalothorax and a sternum that is often black.

A considerable number of animal studies have been done on the venoms of the different species of lycosids, and along with some human cases, it has become apparent that there can be a considerable difference in the clinical picture of lycosid venom poisoning. Most bites, however, result in little more than localized pain, swelling about the macula area, and some itching.

Other Spider Envenomations

There are no specific therapeutic measures for these bites. They should be debrided if necessary, cleansed and covered with a sterile dressing. Methdilazine HCL 8 mg *tid* may be effective. Where no skin break occurs, apply Itch Balm Plus®,[a] an analgesic/antihistamine/corticosteroid cream that is appropriate for these and all arthropod bites and stings where there is no skin break or infection.

[a] Itch Balm Plus® is marketed by Sawyer Products, Box 188, Safety Harbor, Florida 34695.

SCORPIONS

Approximately 75 of the 800 species of scorpions can be considered of sufficient importance to warrant medical attention. In the United States, members of the genera *Hadrurus*, *Vejovis*, and *Uroctonus* are capable of inflicting a painful and often erythematous lesion. In the United States, only species of the genus *Centruroides* are sufficiently dangerous to warrant definitive medical care. Most stings, however, are inflicted by members of the genus *Vejovis*. The problems of scorpion stings have been reviewed by Mello-Leitao [9], Vachon [10], Balozet [11], and Keegan [12].

Centruroides Species

There are approximately 30 species of this genus confined to the New World. Of these, about seven are of considerable medical importance, and most of these species are found in Mexico. In the United States, they are commonly referred to as "bark scorpions" because of the preference for hiding under the loose bark of trees, in dead trees, or logs. Their general color is straw to yellowish-brown or reddish-brown, and they are often easily distinguishable from other scorpions in the same habitat by their long thin telson, or tail, the pedipalps, or pincer-like claws, and the subaculear tooth. Adults of this genus show a considerable difference in length. *C. exilicauda* (*sculpturatus*) in the southwestern United States and adjacent Mexico reaches a length of approximately 55 mm.

In children, there is often an initial complaint of some pain, although it rarely appears to be severe. Some children, however, do not complain of pain and are unaware of the injury. The area becomes sensitive to touch and merely pressing lightly over the injury will elicit an immediate reaction. Usually there is little local swelling or erythema. Children become tense, restless, and display abnormal and random head and neck movements, as well as roving eye movements. Tachycardia may often be evident within 45 minutes, and hypertension, although it is not seen in children as early or as severe as in adults, may be present one hour after the sting. Respiratory rate is increased and, by 90 minutes postbite, the child may appear quite ill. Fasciculations may be seen over the face or large muscle masses, and the child may complain of generalized weakness and display some ataxia or motor weakness. The respiratory distress may proceed to respiratory paralysis. Excessive salivation is often present and may further embarrass respiratory function. Slurring of speech may be present, and convulsions have been reported.

The history of the treatment for scorpion stings will provide the reader with a fascinating touch of mythology, folklore, hunches (educated and otherwise), and a listing of all sorts of therapeutic devices from electroshock to mechanical compression bandages. The list of drugs that have been advised for scorpion stings include atropine, barium, digitalis, epinephrine, heparin, hyoscyamine, iodine, procaine, morphine, physostigmine, reserpine, steroids, antivenins (snake, spider, and scorpion), vitamin C, ice, hot water, cold water, and all manner of fruit juices, papain, and petroleum products. Other than the scorpion antivenins, there is no clinical evidence that any of these substances is of specific value.

There are no first-aid measures of value, although there is no harm in placing a piece of ice over the sting area to reduce pain. In any severe scorpion sting by one of the species known to be dangerous, the specific or suggested antivenin should be used. The author

generally gives twice the recommended dose and gives it intravenously and slowly, unless otherwise indicated. The antivenin should be diluted with 100-200 ml of 5% dextrose in water or physiological saline. Mild sedation is often indicated. Valium is the drug of choice for convulsions. Intravenous phenobarbital should be used but only with the greatest of care. Nitroprusside is the drug of choice in the acute hypertensive cases. Assisted ventilation may be necessary. Digitalis and diuretics have been used in the rare decompensated infant or child. Atropine and steroids are of questionable value and do not seem indicated.

Vejovis Species

This genus is closely related to *Uroctonus*, with which it is sometimes confused. Both genera are ground scorpions and whereas *Uroctonus* usually inhabits mountain habitats from southern California into Oregon, *Vejovis* has a wide distribution from the southern portions of Canada, south though Wyoming and Colorado to Texas, and west to California. Some species may measure up to 85 mm but most are from 30-55 mm. *Vejovis spinigerus*, medically the most important species, is often called the striped-tail scorpion.

Hadrurus Species

This genus contains seven or eight species and is native to North America. These are the longest and most heavy-bodied of our scorpions, generally termed the giant hairy scorpions because of their size and the conspicuous bristles on their legs, pedipalps, and cauda. Adults of some species may measure up to 135 mm. These are burrowing species and may be found as deep as two feet in sandy soil. These are native to Arizona, California, and parts of Utah, Nevada, and Idaho, as well as Mexico.

The stings of these and related species can be quite painful in children, leading to anxiety and restlessness. Often there is some diffuse swelling and erythema around the sting but, in general, this is not severe. In several children seen by the author, there has been a marked increase in salivation following *Vejovis* stings, as well as marked and painful swelling of the part. There may be some pruritus and few other significant findings. The local swelling may persist for several days.

Stings of all scorpions in the United States, other than *Centruroides* species, are not sufficiently dangerous to warrant definitive medical care, but all such stings in children should be watched carefully during the first four hours, for an occasional child will react adversely. In such cases, the treatment is symptomatic. Itch Balm Plus® can be applied to the bitten area in the event of pruritus, pain, and swelling.

BEES, WASPS, HORNETS, AND ANTS

The venoms of these insects (order *Hymenoptera*) contain, among other components, peptides and nonenzymatic proteins (e.g., apamin and melittin and/or kinins), enzymes (e.g., phospholipase A and B, and hyaluronidase), and amines (e.g., histamine and 5-hydroxytryptamine). While it may take over 100 bees to inflict a lethal dose of venom in most adults, one sting can cause a fatal anaphylactic reaction in a hypersensitive person. There are three to four times more deaths in the United States from bee stings than from snakebites. In a few fatalities that have resulted from multiple bee stings, death has been attributed to acute cardiovascular collapse.

The stings of many *Hymenoptera* may remain in the skin and should be removed by teasing or scraping rather than pulling. An ice cube placed over the sting will reduce pain. Persons with known hypersensitivity to such stings should carry a kit containing an antihistamine and epinephrine when in endemic areas. Desensitization can be carried out using whole-body antigens or, preferably, whole-venom antigens.

TICKS AND MITES

Ticks are vectors of many diseases. In North America, some species of *Dermacentor* and *Amblyomma* cause tick paralysis. Symptoms and signs include anorexia, lethargy, muscle weakness, incoordination, nystagmus, and ascending flaccid paralysis. Bulbar or respiratory paralysis may develop. The bite of some *Ornithodorus* ticks ("pajaroello"), found in Mexico and the southwestern United States, cause a local vesiculation, pustulation, rupture, ulceration and eschar, with varying degrees of local swelling and pain. Similar reactions have been observed following the bites of other ticks.

Treatment of tick paralysis is symptomatic. Oxygen and respiratory assistance may be needed. An antitoxin is presently under study. Pajaroello tick lesions should be cleansed, soaked in 1:20 Burow's solution, debrided, and painted with the aqueous triple dye used for pit viper bites. Corticosteroids are of value in severe reactions. Infections are common during the ulcer stage, but rarely require more than local antiseptic measures.

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GRIZZLY BEAR ATTACKS: PREVENTION AND SURVIVAL

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LEARNING OBJECTIVES

Following this presentation, participants will be able to:

1. Reduce the chances of having a close encounter with a grizzly bear
2. Reduce the chances of being attacked if you have a close encounter
3. Reduce the severity and type of injuries if you are attacked by a grizzly bear
4. Reduce the chances of being preyed upon by a grizzly bear
5. Understand the principles of treating victims of grizzly bear attacks

GRIZZLY BEAR ATTACKS: PREVENTION AND SURVIVAL

Steven P. French, M.D.

I. PERSPECTIVE

Approximate Number of Human Deaths from Animal Attacks Worldwide Annually

Man	100,000's
Snakes	60,000
Tigers, Crocodiles	1,000 (each)
Domestic Livestock	1,000
Hippos, Elephants	500 (each)
Leopards, Lions	500 (each)
Grizzly Bears	< 1

Grizzly Bear Attacks Resulting in Injury

Grizzly bear attacks are relatively rare and sporadic. A total of only 126 attacks were reported from 1900-1979 in all national parks in North America. From 1970-1979 there were 16 grizzly attacks reported in Yellowstone National Park.

Injury Rates

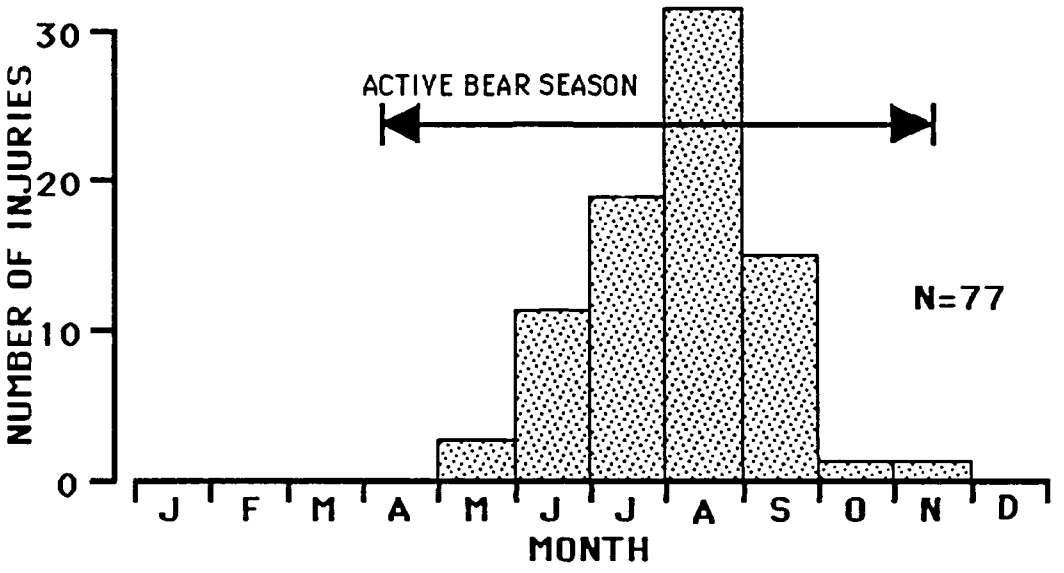
Calculation of an accurate injury rate remains elusive. Not only were earlier records incomplete, but it has always been difficult to define and quantify those at risk. The following are estimates of injury rates for all visitors to these national parks in the 1970's:

Glacier National Park-1 injury/1.3 million visitors
Yellowstone National Park-1 injury/1.5 million visitors

The injury rate for registered backcountry users in Yellowstone during the same period was one per 59,300 backcountry use days.

Seasonal Variations

There is a predictable seasonal variation in bear attacks (both black and grizzly) that correlates with human use of bear habitat during the active (non denning) period for bears. The following graph demonstrates this seasonal variation for 77 bear attacks that occurred in Yellowstone National Park from 1970-1990:



Although grizzly attacks are rare, the psychological impact of widespread media attention appear to inflate their frequency and significance, particularly when they sensationalize the horror and gore. Every attack, regardless of the extent of injuries, is traditionally referred to as a "mauling" and this term itself supports the "grizzly bearanoia" that many people feel about grizzlies, particularly when they visit grizzly country. Being injured and sometimes killed by a wild animal is not considered to be a socially acceptable form on injury by most members of our society today.

II. CIRCUMSTANCES of GRIZZLY BEAR ATTACKS

A. Surprise Encounters

1. Terrain and the environment: can limit the sensory detection of one another until they are in close proximity.

2. Hunters of other game: tend to move around quietly thereby not allowing a bear detect them readily.

3. Sows with young: only 20% of a population is made up of sows with young but they account for up to 2/3 of grizzly bear attacks. Defense of young, or just a greater opportunity for human encounters since sows with young are more frequently active when humans are during the daytime?

4. Carcass: a significant number of injuries have occurred when people have come across a grizzly either feeding on, or situated nearby, a carcass. Is this a defense of carcass, or simply a close encounter when a bear is anchored and preoccupied with a food item?

B. Intentional Encounters

1. Hunters: they obviously violate basic safety rules for the chance to hunt bears. Wounded bears represent an additional risk.

2. Harassment: this particularly applies to the modern increase in photographers, both commercial and amateur.

3. Handling: research and management requires trapping, anesthetizing, and releasing of grizzlies with a significant risk of morbidity and mortality to both handlers and bears.

4. Predation (the intent is by the bear): there is little evidence to suggest that grizzlies in North America stalk humans as prey during the daytime, although there are several documented cases of polar and black bears stalking and preying on humans as a daytime event.

C. Predation

It is important to understand that most people attacked by grizzly bears are not killed because the intent of the bear is simply to remove a perceived threat, not to prey on the individual. From 1900-1979 there were 19 human deaths from grizzly attacks documented in the national parks in North America and an additional 22 deaths in Alaska outside the parks. Some were victims of defensive attacks and probably would have survived if current medical management techniques had been available, such as ATLS. However, some of these deaths occurred as a result of predatory attacks. The point that has puzzled me the most is not that grizzlies occasionally prey on humans, but why they don't do it more often. As a potential prey species, humans are predictable and abundance, are easy to catch and easy to kill, and are easy to consume for a grizzly. So why don't they prey on us as part of their routine feeding behavior?

There is little historical evidence to suggest that grizzly bears preyed upon humans routinely, except in unusual circumstances. In 1860 a smallpox epidemic struck a small band of Stonie Indians (Assiniboin Tribe) who were camped in the Yarrow Creek drainage in Alberta, Canada. Grizzlies began scavenging on the dead who were left on the ground as the tribe moved to the next drainage. Grizzlies followed them to their next encampment and began preying on those who had survived. For several years thereafter, the Indians avoided this area for fear of being eaten by grizzlies who had "learned to prey" on humans.

Since reasonably accurate records have been kept (roughly 1900), predatory attacks on humans by grizzly bears has been generally rare, sporadic, and isolated events. However a disturbing trend appears to have begun in recent times. Since 1967 there have been 8 cases of human predation in or adjacent to Glacier and Yellowstone National Parks in the U.S. and Banff National Park in Canada. In all cases the grizzly involved was judged to be either habituated to the presence of humans and/or conditioned to human foods. It should be pointed out that during this same time period there have been perhaps thousands of bears with these behavioral traits who did not prey on humans. Nonetheless it is possible that conditioned and/or habituated behavior appears to predispose some grizzlies to prey on humans under certain circumstances.

III. CASE HISTORIES

What can we learn from other's mistakes? It is important to realize that the information is gathered from victims who generally are inexperienced with respect to bear behavior and whose interpretations of the events reflect their cultural biases. Also, victims often become instant media celebrities and I know of several cases where the circumstances surrounding the attack "changed" significantly with each telling, generally reducing their own culpability. And because of potential litigation, some victims have only told their stories through an attorney.

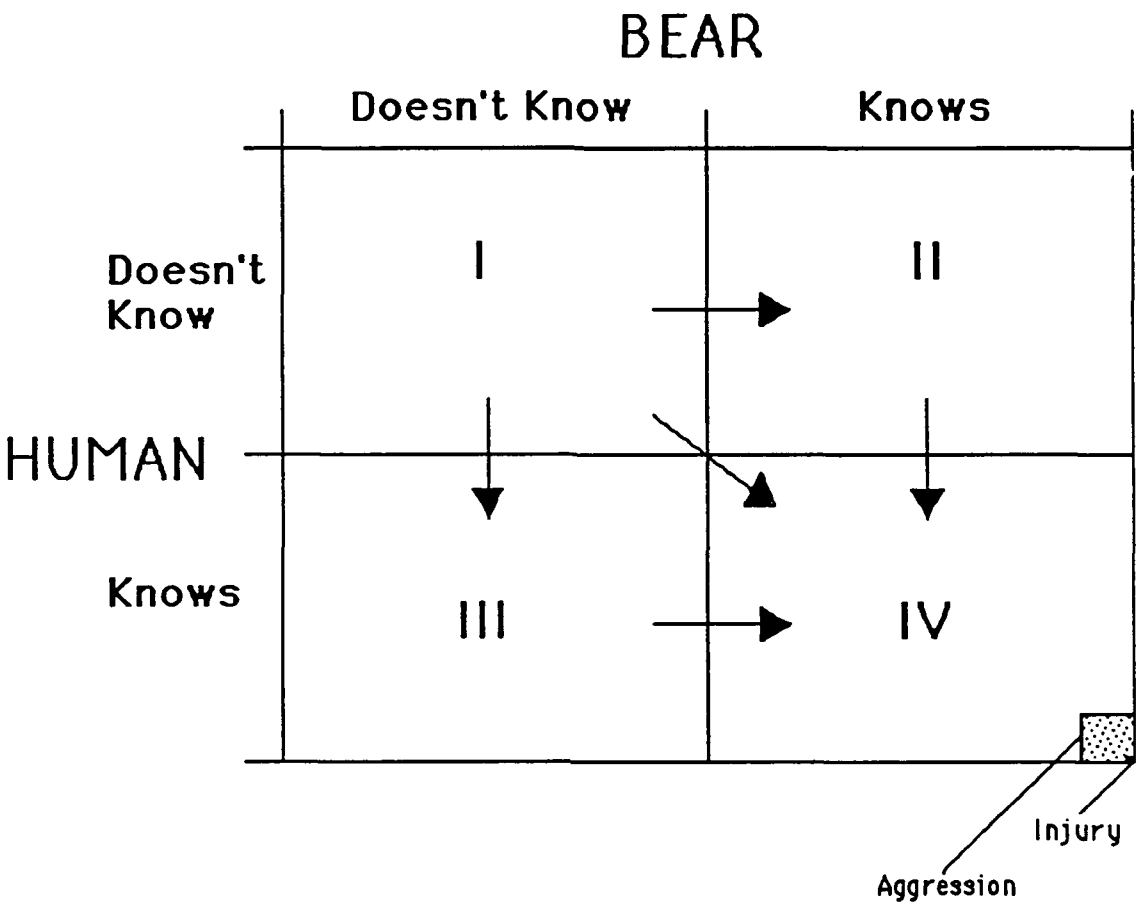
Despite these potential limitations in the history given by the victim, there is often valuable information that can be gathered by investigating the indirect evidence at the scene and along with the victim's account, especially their first one.

IV. CLASSIFICATION OF GRIZZLY-HUMAN INTERACTIONS

Our notions of how best to avoid grizzly bear attacks have have previously been drawn primarily from what attack victims did "wrong". But since most people who live, work and recreate in grizzly country for a significant part of their lives never get injured, it is equally important to understand what it is they have done "right". Unlike bear attack victims, these people have successfully navigated throughout grizzly country without being injured. How have they done it? Unfortunately this information is not as readily available as attack records, but it nonetheless remains critical to our knowledge of grizzly-human interactions.

As an example, from 1900-1985 there were 115 human injuries from combined black, polar and grizzly bear attacks in Alaska, but only 2 victims were natives. This strongly suggest that the behavior of people is very important in determining the manner in which they coexist with bears. The following model was developed to illustrate the relationship between people and bears who occupy the same habitat.

MODEL OF GRIZZLY-HUMAN INTERACTIONS



Class I: People and grizzly bears using the same habitat but are not aware of each other's specific presence at a given time.

Class II: The grizzly bear knows that a person(s) is nearby, either by sight, sound, smell, or a combination. Since successful bears have avoided humans most will retreat if given the opportunity and the person(s) never knows they had an encounter!

Class III: A person(s) typically sees a grizzly but the bear is unaware of their presence. If the person(s) leave the area without disturbing the bear, a potentially aggressive encounter is avoided.

Class IV: Now the person(s) and the grizzly are aware of each other. Even at this stage, bears generally retreat without causing injury. Nonetheless, circumstances that lead to entry into this class of interaction should be avoided when possible.

V. SAFETY IN GRIZZLY COUNTRY

Concerning safety in grizzly country, there are four areas that warrant special consideration:

A. Avoidance

You can significantly reduce your chances of having a close encounter by doing three simple things:

1. Make noise
2. Remain alert
3. Use good judgement

B. Don't provoke an attack

Even if you have a close encounter (class IV), there are still several things you should and should not do that can reduce your chances of being injured:

1. Identify yourself
2. No sudden movements
3. Don't rivet
4. Attempting to climb a tree is generally inappropriate
5. Animal math-safety in numbers
6. If the bear charges, stand your ground
7. Prepare mentally for an attack
8. Submission versus vulnerability

C. If you are attacked-"Damage Control"

If an attack is imminent, you still have important actions to consider. What you do immediately before, during and after an attack will influence the type and severity of your injuries.

This is where the data from attack victims is very useful. Humans are rarely killed during an attack precipitated by a surprise, close encounter even though they are physically capable of easily and quickly doing so. Why? My interpretation of the attack data is that during these type of attacks, grizzlies are only trying to remove a perceived threat and their intent is only to use as much force as is necessary. It is also very important to understand that grizzly bears are head-oriented during agonistic intraspecific social behavior and

they commonly direct their aggression towards humans in the same manner, i.e. towards the head Therefore the general rules to follow during an attack is to "help" the bear remove the perceived threat and to protect your vital areas:

1. Don't run, try to climb a tree, fight or scream
2. Protect the head and neck by interlocking your hands behind your head (ear level) and flexing your head forward, either in the fetal position or flat on the ground face down. Use elbows to cover the face if the bear turns you over.
3. Never look at the bear during an attack
4. After the attack, stay down until you are sure the bear has completely left the area- this is an extremely important point. People who have gotten up before the bear has left generally get significantly more severe injuries during the second attack.
5. When you believe the bear has left the area, peek around while moving as little as possible, try to determine which way the bear left, evaluate your options and leave the area.

Special Considerations

1. Companions-all stay down during the attack unless you are absolutely sure you can get safely away.
2. Bear spray- we do not recommend its use for several reasons. It is usually spayed into the wind during a surprise encounter, limiting its range and most likely spraying yourself. Using it during a charge exposes you to significant injury. It is not likely to be available when you really need it. It gives a false sense of security which can alter the good judgement needed to avoid the close encounter to begin with. It is not aerosol brains in a can!
3. Guns- many of the same arguments can be made against its use as was made for bear spray but there are two additional concerns. First is the kill target for a grizzly is small. The brain is narrow and long and the bear is a quickly accelerating moving target. And secondly is that if you injure the bear you now have an enraged wounded bear that is likely to deliver a more severe if not fatal injury. Most attacks are so sudden that people relying on guns don't have a chance to fire them. If you decide to use a gun, make sure you are well experienced and make sure you kill the bear before it gets to you- it is a very big gamble, and your life is at stake.

D. REDUCE THE CHANCES OF PREDATION

Don't Attract Bears to Your Campsite

1. Avoid travel corridors/seasonal feed sites
2. Avoid sites that have been "trashed"
3. Proper food storage
4. Reduce odors
5. Don't leave a hidden mess for the next campers:

Have a contingency plan

1. Become familiar with the area
2. Sleep in a tent, don't zip up bag
3. Flashlight; bear spray; gun?
4. Group effort- explain

Menstruation

In August 1967, two women were killed in separate events on the same night by different grizzly bears in Glacier National Park. On postmortem exam it was discovered that one had been menstruating. That menstruation may be a precipitating factor in attacks and/or predation has unfortunately become solidly ingrained into the opinions of most people who ponder the question. It has been a complex journey to answer the question with any scientific basis, burdened with strong cultural biases, enforced with hysterical mass media coverage, and finally left dangling by both scientists and bureaucrats alike.

In 1985 B.S. Cushing presented the results of his master's degree study "Response of Polar Bears to Human Menstrual Odors" to the International Bear Association. Although the study was designed inadequately to test the hypothesis that menstruating women were more likely to be either attacked or preyed upon by grizzly bears, the press nonetheless came to this conclusion. The Interagency Grizzly Bear Committee then gave the ambivalent caution in the government's official grizzly bear pamphlet (***Bear Us In Mind***)that said "Women may choose to stay out of bear country during their menstrual period". There remains <u>no</u> scientific evidence that even suggests that menstrual odors precipitate grizzly bear attacks. Keep in mind that there has been only one documented serious attack on a known menstruating woman (August, 1967) in North America, and even the official investigating team concluded that menstruation did not appear to have play a major role in that attack!

VI. INJURIES FROM GRIZZLY BEAR ATTACKS

Severity: Ranges from trivial, typically treated on an outpatient basis, to severe, requiring hospitalization, surgery, and resulting in significant cosmetic and/or functional disability.

Character		Sources
1. Tearing	6. Shearing	1. Teeth
2. Crushing	7. Punctures	2. Claws
3. Blunt	8. Occult	3. Paws
4. Dirty	9. Old	4. Trees
5. Gravity	10. Ballistics	5. Guns

Wound management

The specifics of initial wound treatment will be determined in part by the available medical equipment and location in which you first receive the victim. Stabilization of the patient remains the primary objective using the principles of ATLS. Consider all grizzly bear attacks as major trauma and proceed with appropriate care for the facility, advancing upwards as indicated. Injuries can often be occult. Internal injuries are not uncommon, either from direct penetration (claws, teeth) or blunt trauma. Neuro-vascular injuries must be considered with trauma to the extremities, and neuro-sensory and cosmetic injuries are very common with trauma to the face. Remember that wounds are usually old and contaminated.

Antibiotics

The awesome mystic of the grizzly's power seems to have invaded our sense of the bacteria they must harbor, as if to assume that "you gotta be one bad dude (bacteria) to live on a griz!"

There are no studies of the normal grizzly flora and only limited data for black bears. Nonetheless there does not appear to be any anecdotal evidence that attack victims develop unusual or rare septic complications from unknown pathogens.

It is important to remember that the blunt trauma, deep punctures, and shearing/tearing forces create significant tissue ischemia and necrosis that is not apparent initially.

Rabies immunization

There is no documented case of rabies in a grizzly bear. However the CDC recommends rabies immunization to victims attacked by wild carnivores. Therefore informed consent of the risks vs benefits for rabies immunization is in order.

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