

Volume 2, Edition 2

Spring 2002

Journal of Special Operations Medicine

A Peer Reviewed Journal for SOF Medical Professionals



Dedicated to the Indomitable Spirit & Sacrifices of the SOF Medic

From the Surgeon



SOF Colleagues,

Recently we had a big success for the soldiers, sailors, airmen and marines we are sworn to serve and dedicated to, and I thought I would pass along the news.

Over the past several military operations (Just Cause, Desert Storm, Somalia) it became apparent that while the body armor we provide our troops does a very good job of protecting them from wounds to the chest and abdomen, our folks still are wounded. Often these wounds are close to the vest (in the groin, the axilla or the neck) and getting good tamponade to bleeding is very difficult under controlled conditions, let alone under the stressors of combat. Recognition by medics of this difficulty pushed us all toward a fix.

Over the past several years our CINC has given the SOCOM Surgeon's office money for investing in medical studies of "stuff" that may make a difference in the care of our forces. A function called the Biomedical Initiatives Steering Committee (BISC) came into existence with the intent to take our CINC's money and invest "seed dollars" in research on medical initiatives that will help save our Quiet Professionals. Several folks, who had experience with wounding and the frustrations at being unable to stop hemorrhage, helped push several initiatives forward. One of these was the hemostatic dressing initiative. It was felt that if we had a dressing that not only covered a wound, but also helped initiate clotting, it would prevent some battlefield losses due to exsanguination. BISC dollars were applied, and several technologies were evaluated. We in SOCOM were most pleased with the "fibrin dressing" initiative where a dressing with impregnated fibrin was applied to significant wounds, arresting bleeding after a short period of time. Several other very promising technological advances of interest to us are also out there, but for brevity sake I will only mention them (Factor VII, Marine Polymer Dressings, Chitosan Dressings).

As one can imagine, this technology has a significant price tag, but that is where the story gets most exciting. The Army was the lead on this research and the funds weren't forthcoming. Due to the medical issues related to Afghanistan, our CINC and the CINCCENT sent a letter to the Chairman of the Joint Chiefs asking for support for this technology and the CJCS concurred. The funding is imminent--actually, the Army research folks are moving fast on the fielding. This fall we should have the first delivery of 1,000 of these potentially life-saving dressings in to the hands of our medics, corpsmen and PJs.

So, I spend this time on the topic to let you know that there are folks working the issues the front line medics bring up, and things are accomplished. It will be a success for them and their requirements, the CINC's BISC initiative, and for all of us in a support role to the front-line medic doing the job.

Finally, I want to briefly state that we are close to resolving other issues for our SOF Medics:

-hemoglobin oxygen carrying fluid is getting lots of attention and we hope to soon have this in place of

crystalloid fluids and Hetastarch as volume expanders.

-Factor VII research goes on.

-A one-handed tourniquet is on the way.

-Interosseous fluid routes in place of fragile intravenous routes are being examined for our use.

So, again, find your colleagues and get information to us that will help you in the medical care and salvage of our SOF Warriors--they are truly national treasures.

dhammer



Dave Hammer (center) ,1st Marines, Fleet Marine Force Pacific, winter 1959.

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USASOC



Rocky Farr, MD
COL, USA
Command Surgeon

In December 2001 at the Special Operations Medical Association meeting in Tampa (www.soma.org), the USASOC surgeon's office presented a day of lectures and discussions on unconventional warfare medicine entitled: "Medical Unconventional Warfare Doctrine for the 21st Century: Guerrilla & Resistance Medicine & Hospitals."

Although we had conceived doing this many months before 11 September 2001, it was right on target and sort of eerie to be talking on this subject at the same time that Special Forces teams were DOING unconventional warfare medicine on the ground for the first time in a long time. I would like to thank all of the staff from my office that helped write and presented the various briefs. The briefings and those who gave them were:

1. Introduction--Mission For the Day: New Ideas, New Medical Doctrine for the 21st Century. COL Warner D. Farr
2. Those Who Cared Before Us: Historical Examples of Guerrilla Medicine in the Unclassified Literature. COL Warner D. Farr
3. U.S. Guerrilla Warfare Doctrine 1941-2000: From FM 31-21 1951 to Draft FM 3.05.200 1999. MAJ Hal Abner
4. U.S. Medical Guerrilla Warfare Doctrine 1941-2000: From "G" Hospitals to Black Helicopters. MAJ Mitch Meyers
5. Emerging 21st Century U.S. Unconventional Warfare Doctrine: Urban, Joint, and Coalition. MAJ Hal Abner
6. Medical Personnel in Unconventional Warfare: What! Who, Me? COL Kevin Keenan
7. Emerging Principles Outside the Ordinary: Surrogates, Allies, and National Infrastructure. Mr. David Passaro

I have always thought it important to know our roots so a recommended reading list of unconventional warfare, most all with an emphasis on its medicine is:

1. Chapman, F. Spencer. The Jungle is Neutral. London. 1949.
2. Conboy, Kenneth and Morrison, James. The CIA's Secret War in Tibet. University Press of Kansas. 2002.
3. Dragic, Djordje Dr. Partisan Hospitals in Yugoslavia. Vojnoizdavacki Zavod.1965.
4. Evanhow, Ed. Dark Moon. Naval Institute Press.1995.
5. Grose, Peter. Operation Rollback. Houghton Mifflin Company. 2000.
6. Hoyt, Edwin P. Guerilla. MacMillan Publishing Co., Inc. 1981.
7. Moss, Robert. The War for the Cities. Coward, McCann & Geoghegan, Inc. 1972.
8. Rogers, Linday. Guerilla Surgeon. Collins Clear-Type Press: London and Glasgow.1957.
9. Stahl, Bob. You're No Good To Me Dead. Naval Institute Press. 1995.
10. Street, Brian Jeffrey. The Parachute Ward. T.H. Best Printing Company Limited.1987.
11. Temchin, Michael. The Witch Doctor. Holocaust Library.1983.

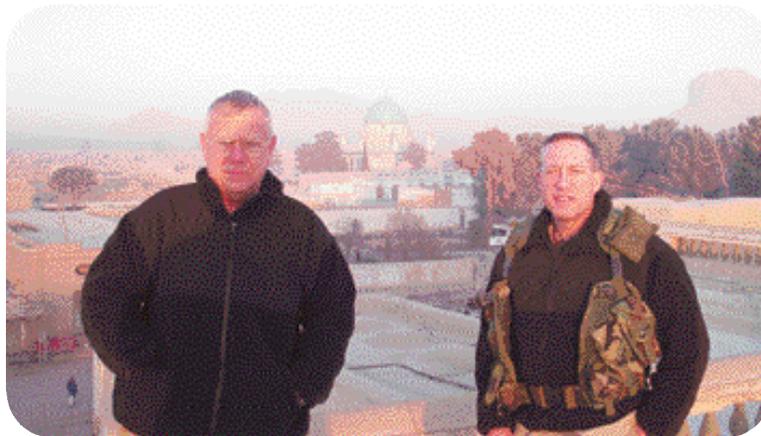
Most of these out of print books can be found by Internet used book searches and usually are only about \$10-\$15 a piece. My office has a CD with all of the Special Forces field manuals back to the 1950s on it and the Tampa unconventional warfare briefings. The old manuals are great. They answer many of the questions that the new manuals leave unanswered.

Less than 2 weeks after this day of talking about unconventional warfare medicine in Florida, I arrived in Afghanistan and was able to see unconventional warfare medicine on the ground, up close and personal. When I ask, "What was your biggest problem?" The first team medic I talked to said, "Nursing care, sir. I treated the trauma just fine, but the next day they were still here!" I have a habit of ranting about the decade of the 1990s being the decade of the "black helicopter that will take away your wounded in 15 minutes." Well, that decade is over. The Special Forces teams saw their wounded, nursed their wounded, healed their wounded and fought with their wounded over the next several months. Colonel Keenan, as the dean of the Joint Special Operations Medical Training Center, is well on his way to enhancing the postoperative nursing care training at the school and was committed to that long before 9/11. To accompany that will also be inhalation anesthesia. Those old medics (me!)

will realize that none of the changes I have just described are new, they are just revolving the wheel back to the way it used to be.

The team medics that I saw were doing very well at both unconventional warfare medicine and in using local health care providers if they had them. I saw a much more "encompassing" picture of using the local assets rather than simply importing western medicine and saying it was the only way. An area that we have far to go in is medical resupply. My office is working with the Army Medical Department's Medical Research and Material Command to field new medical sets. The first units will get them this fall. Pinpoint, directed resupply after infiltration is the problem. In wars in the 1960s, I had the same problem. People tend to send you what they have, and plenty of it, rather than exactly what you ask for. The mechanism to ask for resupply, bundle codes, et cetera, have been ignored for decades.

There will be a formal process of after action reports and other critiques of medicine at the war. My impressions after my December - January deployment is that several of the larger episodes of casualties resulted in a very high number of very seriously wounded soldiers all at the same time and that the medics, physician assistants, and non-medical team members did absolutely wonderful care on the battlefield.



COLs Farr and Burford on vacation



NAVSPECWARCOM



Larry Garsha, MD
CAPT, USN
Command Surgeon

What does it mean to be called Doc?

I observed one of our medical corps officers and a corpsman take charge and take care of one of our shipmates. The individual's names don't matter; what is important is the lesson they provide for all of us.

A senior officer at a local command complained of a funny sensation that he had had for the previous two weeks in his right upper chest. It was not painful; it was more like a tightness. He first experienced the pain when he was getting ready for bed on a vacation trip to Disneyland.

He had been continuing his workouts of pushups and sit-ups. However, with increasing problematic hypertension he was not running as far as usual. He still enjoyed a noontime game of basketball with his teammates. His physician and the IDC corpsman assigned to his unit felt initially that it might just be a muscle strain and asked him to come back after morning PT. He did and complained that the muscle sprain got worse when he was walking. It got better when he stopped and rested. The corpsman and doctor became concerned. They ordered an initial series of tests, accompanying him to the branch medical clinic. When the EKG was not normal, they personally sought out the duty officer of the cardiology department of the local Naval Hospital. Because his vital signs were normal and he was pain free, he was scheduled for a stress test the next day.

The IDC called the officer's wife and relayed the concern about possible heart disease causing the

chest muscle strain. He attempted to be truthful and at the same time not cause undue alarm. He was successful.

Both the IDC and medical officer accompanied the shipmate the next day to the cardiology clinic. They looked resplendent in their uniforms and got the immediate attention of the clinic staff that spent the majority of their time looking after dependents and retirees. As a result, their shipmate got the head of the line privileges.

One thing led to another, a series of phone calls to senior supervising officers and the shipmate's spouse and daughter and, before the day was done, a life was saved. The senior officer ended up having angiography and stent placement to open the left anterior descending.

What's it take to be a Doc?

It takes intelligence to put together clues, it takes intuition to make the guess correctly, it takes caring to offer support when nothing else can be offered, and it takes humility to refer to the "expert" when in doubt or there is a question. Marine corpsman have a creed called "I'm the one called Doc." It is best summed up by the last sentence:

"And if you have to go out there and your life is on the block,

Look at the one right next to you... I am the one called "Doc."

This is what made me proud as I watched our NSW medical officer and IDC. They were standing next to their shipmate.

Remember what it takes to be called Doc. Remember when someone walks in as you are

preparing to leave with what sounds like a minor complaint. Remember when the same person has the same complaint and it grinds on you just to hear it again. Remember that those coming to you think of you as their lifesaver.

Switching to technical / equipment side of the house, who favors the Combitube? I recently attended the Emergency Airway Management Course and came away with a lot of appreciation for the Combitube. The standard of care for intubation in a medical facility is rapid sequence intubation involving pre-treatment with medication in addition to paralyzing and hypnotic agents. In the civilian field, the Combitube is frequently used in head trauma

and other unconscious trauma victims. Endotracheal intubation remains the field standard and most civilian EMT-Ps do them frequently enough to remain competent. But I worry about NSW. Do we do enough intubations to remain current?

Figure that you should do at least 1-3 per week. If you are not doing that, then I would submit that we should have another airway approach for the unconscious patient who may need a reassuring airway. Note I said reassuring, not protected. The Combitube is reassuring in that it provides some protection but it is not a definitive or protective airway.

All for now. Let me know your thoughts at garshal@navsoc.navy.mil.





HANDLING FATIGUE AND FEAR IN COMBAT OPERATIONS

After the controversy over whether the nation's combat forces are "tired and spread thin," and the subsequent response from officialdom that they are not, you might wonder why I would want to engage on this politically sensitive topic. Because it's important! For a number of reasons.

Many of the bad things that happen to people in combat operations can be traced to the effects of chronic fatigue, and the debilitating influence of a constant threat to safety. Whether people think we are, as a whole, spread thin and tired, clearly there are times when on an individual level we have been pushed to the limit, and when we ask ourselves "how much longer do I have to keep looking over my shoulder to make sure that I don't get hurt?" Constant vigilance and long hours are a prescription for mistakes, injury, and loss.

My conversations with our seriously wounded, our returning aviators, wrench benders, and box kickers... and ALL of the folks we need to prosecute the War on Terrorism, indicate to me that there are invisible scars and open wounds that are not healing. Folks who have performed superbly in action are stumped by why they feel isolated, why they are compelled to repeatedly check that doors and windows are locked, why getting up in the morning is such a chore. All of these can be symptoms of the effects of stress and fatigue.

I'm worried that we are not doing enough as medics to fulfill this aspect of our sacred trust: to relieve suffering--not only physical suffering, but also mental suffering.

AFSOC



James Dougherty, MD
Col, USAF
Command Surgeon

Our "one answer fits all situations" approach to combat psychiatry is faulty. The alphabet soup of acronyms, whether you like PIE or BICEPS better, fails to recognize that different stressors lead to different reactions. This is basic medical stuff: different syndromes and different causes mean different therapies. We need to keep the BICEPS principles in mind, but not forget that people have different ways of coping.

From the soldier who is wounded and experiences the loss of close friends to the crew chief who wonders why he or she feels different, possibly unable to sleep at night after returning from theater, there are multiple ways we react.

We need to take a comprehensive look at how we approach (1) supporting the troops while engaged in combat operations, (2) responding to critical incident stress, and (3) what "medicine" we provide to heal our folks upon their return from the theater. Each of these situations requires a different set of tools and strategies, and is best managed by different parts of our medical support system.

In the field the focus is on maintaining combat capability. When one of our soldiers, sailors or airmen falters, the mission is degraded. Under these circumstances, our medics, flight surgeons, and chaplains need to be on the spot, providing the encouragement, emphasizing the individual's value to his or her buddies, and the expectation that they won't let them down.

When major incidents occur, and the horrors of war visit our folks up close and personal, there is a need to intervene IMMEDIATELY to mitigate the potentially harmful long-term consequences of trauma. The focus is on "damage con-

trol" techniques and the resumption of normal activities.

And on their return to home station, there should be an environment that does not stigmatize the individual who is confused by their very normal reaction to extraordinary experiences. Fatigue, fear, and stress slowly break down our strength and stamina. The recovery is just as slow and cannot occur overnight.

NO ONE IS IMMUNE, not even the strongest of us. In fact, our folks who appear to have a spine of steel are particularly vulnerable because they've never been taught how to deal with "internal" injury.

Here's the tasker. For all of our medics in the field, put eyes on everyone in your unit, every day. Constantly "triage" their state of mental health.

Intervene to bolster confidence and ease the load. Take "prevention" into the hearts and minds of the heroes who have agreed to sacrifice their well being on behalf of the nation.

When bad things happen, don't let anyone get lost in the shuffle. If you have folks who appear to be crawling into their hole, pull them out and get them to help.

When those who have performed superbly in combat, adjusting to the death and destruction of warfare, are confronted with the shock of a return to the peaceful day-to-day world, don't forget them. As we know, special operators are not mass produced. Likewise they cannot be "used up," and then expected to perform again. They deserve better, and the nation needs their continued vigilance and spirit.





UNITED STATES SPECIAL
OPERATIONS COMMAND
Office of the Command Surgeon

From the ROAD DOG in the BIG HOUSE,

It's been one year already that I've served you, the Operational Medics (Road Dogs). Just a few notes to let you know where we are and where we are attempting to go. First I would like to share with you what I've been charged with to perform as your SEMA here at USSOCOM:

- * I am the advisor to the USSOCOM SG and key staff on all matters concerning morale, welfare, professional development, and effective use of the enlisted medical force.

- * I coordinate with component SEMAs on all SOF enlisted medical matters.

- * I monitor promotions, assignments, retention, training, and professional development of SOF enlisted medics.

- * I serve on boards and councils relative to medical activities and enlisted affairs.

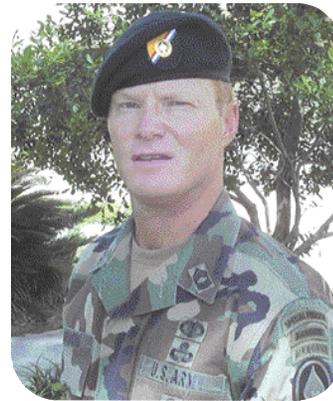
- * I assist SOCS-SG staff in developing SOF medical support concepts and doctrine.

Here is where we are at the publishing of this journal. The USSOCOM Command Surgeon's office has been active fighting the good fight for you the SOF medic. Here is a brief discussion of the top three topics:

- * Health Surveillance- USSOCOM has a practical health surveillance solution which meets operational and statutory requirements. Staff from this office will travel to the component Command Surgeon's offices and provide a block of instruction on the DHSS device and the program that inputs the repository. DHSS devices are almost ready to issue to the component SG for dispersal. More to follow!!

- * Hemostatic Dressing (HD) - DCINC USSOCOM approved ORD 1998 and with the advent of OEF, Medical Research and Materiel Command (MRMC) has been asked to expedite the acquisition process to field the HD. Release of the funds will prompt an FDA approved IND protocol, and production of 5,000 bandages of which 1,000 will be delivered this Fall to SOF units.

- * Organic Surgical Capability- We know that surgical capabilities forward has saved lives, but the SOF community lacks this ability in the second echelon of care



SENIOR ENLISTED MEDICAL ADVISER (SEMA),
MSG Michael A. Brochu

in most cases. Organic surgical assets would mitigate delays associated with the request for forces process for forward surgical units that are embedded in the conventional force structure.

Prior to 9/11, these three topics had met with a lot of resistance. In Jan 02, the USSOCOM Command Surgeon met with and discussed these topics with the Honorable William Winkenwerder - Assistant Secretary of Defense for Health Affairs. He holds the purse strings for all that's health related. This has heightened the awareness and loosed the strings thus moving forward.

The next area I want to cover is the Joint Medical Enlisted Advisory Counsel (JMEAC). This forum is for you, the ROAD DOG! This is what it was made up to do:

- * Provides a forum to address education, training, modernization, R&D, and morale issues within SOF medicine.

- * Chaired by the USSOCOM SEMA.

- * EAC comprised of voting and non-voting members.

- ** Voting SEMAs - USSOCOM, USASOC, NSWC, AFSOC, and JSOC.

- ** Non-voting - BUMED, AMED-DC&S, AETC, ACC, NSHSPORVA, JSOMTC

- ** Council convenes quarterly and the following are the upcoming purposed dates and locations:

- Ø 17-18 July 02: USASOC will host at FT Bragg, NC

- (this timeline coincides with a SOCM graduation on the 19th)

- Ø 6-7 Oct 02: USASOC will host at FT Bragg, NC (this

- time line coincides with a SOCM graduation on the 8th)

- Ø 29 Nov 02: USSOCOM will host an enlisted meeting

- in Tampa before the SOMA Conference.

- Ø 25-26 Feb 03: NAVSPECWARCOM will host in San Diego.

- Ø 27-28 May 03: AFSOC will host at Hurlburt Field, FL.

If you have suggestions, concerns, and/or recommendations for the JMEAC, pass them along to your SEMA and it will be addressed. But you have to..."SEND IT" Remember that

All ground is "LEVEL" at the foot of the cross

ACLS To Go

Disclaimer: The views contained herein are those of the authors and do not necessarily reflect official Department of Defense position. This publication does not supercede any information presented in other Department of Defense publications.

Bruce Voss, MD

Given the necessities of flexibility, mobility and quality in special operations medicine, it is only natural that technology assists us in our endeavors. Another reason to embrace technology is the operational tempo of the last few years. Some programs for continuing medical education are on CD-ROMs. Since access to technology at the team level has increased and the ability to use that technology seems universal, I will review an advancement that can be taken on any deployment.

Manufactured by ANESOFT Corporation, the "ACLS Simulator 2000" is produced by Howard Schwid, MD and Dan O'Donald, PhD. It can be run on Windows 95 or Windows 98 operating systems and it uses 12 megabytes of hard drive, costing about 75 dollars.

The CD-ROM contains 20 possible patient presentations or patient scenarios. Included are patient types that we may come across either with the active, reserve, or deployed forces. An assortment of cases includes witnessed arrest, ventricular fibrillation arrest, elderly man with chest pain, and a variety of trauma cases. Since this CD-ROM is geared more toward civilian medical practice, it also has patient scenarios not truly applicable to our environment, such as a patient with a positive exercise stress test and fluttering in her chest. The CD-ROM can be loaded on to the hard drive of a computer, not necessarily requiring the CD-ROM with each play of the program.

When the program opens, the first screen that you see is an ACLS simulator icon, icons for CME application, a help icon and other proprietary icons. Our interest is in the ACLS simulator icon and highlighting that with your mouse will bring up a picture of two physicians preparing to perform ACLS on a patient.

Above the two physicians are icons for Patient Assessment, CPR, Defibrillation, Medications, Labs, and Record. Above those icons are drop-down menus that correspond to file, airway, breathing, circulation, defibrillation/differential, and help. Once the introduction is open and the physician-initiated CPR has appeared, the case library is superimposed on the physician's screen. At that point you are prompted to select one of the 20 patient scenarios for advanced cardiac life support. Once you select your patient presentation or scenario you are then presented with a synopsis of the patient's condition and pre-morbid state. You are then asked to confirm the scenario and simultaneously there is an EKG monitor below the CPR screen that shows the current heart rate and rhythm as well as a time delay clock that will show you in seconds the time that has passed since patient contact began.

At this point, you employ your ACLS and medical management skills and assess the patient, administer CPR, initiate defibrillation or apply what interventions you think are appropriate. At any point in the presentation or scenario you can ask for a consultant by way of the drop-down menus. As you progress through these drop-down menus you can either assess the rhythm or pause the patient scenario.

The next step in management allows you to assess the airway, breathing and circulation. This constitutes the ABCs of ACLS and allows you to progress in invasiveness from, for example, simple facemask ventilation to intubation and from spontaneous breathing to ventilation, with assessment of pulse oximetry and circulation i.e. heart, blood pressure, etc. Also, you are prompted for chest compression, IV access, and EKG as well as a precordial thump.

Another aspect of this program is the ability to either defibrillate the patient or, if in doubt, go through the differential diagnosis, which will take you step by step through how the patient presented or what you should be thinking or checking as the scenario unfolds. Finally, you can turn to the Help drop-down menu, where the objectives will be explained for the current patient scenario, with algorithms and drug administration tutorials. It should be noted also that the drug tutorial under the

Help drop-down menu includes the latest guidelines for amiodarone and vasopressin administration.

Also under the Help button you will find the objectives for this scenario. Included here will be the objectives that should have been achieved or will be achieved in the management of this patient. For scenarios that continue without patient improvement, under the Help menu you can go to What Next and that will focus on resolution and patient resuscitation.

Last but not least, under the Help drop-down menu is the squib for instructions. Under that bar lies the current ACLS-suggested patient management for a specific case. The guidelines of the Year 2000 Cardio-Pulmonary Resuscitation and Emergency Cardiovascular Care Symposium are utilized in the scenario development and the treatment protocols are based on recommendations of that international consensus.

Note that the simulator is divided into two modules. First is a rhythm module and second is the pulse module. The rhythm module covers basic EKG rhythm recognition and the pulse module is a real-time simulator providing a scenario for the practitioner to manage, including megacode situations. Even though the simulator program has a multitude of interventions by way of the drop-down menus, the majority of the patient management will come from the icons above the CPR screen. Under the patient assessment icon for example, is the current patient status, to include a quick "head to toe" scan of vital signs, patient appearance, hemodynamics and ventilation ("breath sounds are clear bilaterally and pulse is strong"). At that point you can intervene in treatment of the patient according to what you see on the EKG monitor and the time delay clock. So, for example, if you think that it is time to intubate the patient and administer drugs, but you must establish intravenous access. Also at any time during the management of the patient, if there is a question as to the next step you can always go to the Help button drop-down menu and look at the algorithm to see the next step or what medication should be administered at that time. During the patient management, under the Record icon you can find not only the patient history, but, more importantly, you can review your management of the patient as you progressed from your signing on to signing off.

In summary, as the patient scenario devel-

ops you either have successful or unsuccessful patient scenario resolution. The real key to this program is not mere repetition or text knowledge but application of ACLS principles founded on classroom and didactic work, applied in a setting similar to a flight simulator. Not only does this program allow the individual operator to refresh and challenge his skills and knowledge base in ACLS but also can serve a small or large audience through a projector.



Dr. Bruce Voss entered active duty 8/70 and attended basic, infantry and jump school. He was assigned to the 1st 505th 3rd Bgd 82nd ABN until 1971. In 1971 he went to SF training group with Phase I, II (weapons) and III completed, then went to the 5th SFG. From there he went to 300F1 at Ft Sam, OJT at Ft. Hood and med lab at Bragg. He went back to B/2/5 on a team as 11B4S/91B4S which then became 18B and 18D. He left active duty in 1974 and attended the University of Minnesota with a double major of microbiology and psychology. He graduated Mt Sinai School of Medicine in 1985. Dr. Voss then began 2 years of training in general surgery at Rutgers University Hospital in New Jersey. While in medical school, he drilled with the 11th SFG in NJ as an 18D/18B, E-5/6. He then went to the University of Texas at Houston for a residency in Anesthesiology followed by a fellowship in cardio-thoracic and vascular anesthesiology at Texas Heart Institute and Baylor College of Medicine in Houston. He later did a fellowship in critical care medicine in the University of Texas (Hermann Hospital) Shock Trauma Unit. He was commissioned as a captain and attached to the 11th SFG and later was attached to the 20th SFG (NG) (1996) where he has remained. He attended the USN Hyperbaric Medicine (R&T) course at Panama City, the NOAA DMO course at Ft. Eustis and Virginia Institute for Marine Science, the Hyperbaric Med Course at Key Largo, FL. He received the basic PADI and basic free-fall certifications when on active duty and after the NOAA course attended the IANTD course in North Miami for Nitrox, Technical Nitrox, Deep Air, Trimix and decompression diving. He is board certified in anesthesiology and critical care medicine and will take boards in hyperbaric medicine next year along with boards in trans-esophageal echocardiography (tee). Currently, he works as an anesthesiologist, medical director of an ICU, recently appointed medical director of ACLS training, and is a visiting instructor in critical care medicine in a trauma ICU and hyperbaric physician. Dr. Voss recently started his second year of law school at Concord University School of Law.



Biomedical Research and Development Update:

Bob Clayton

The USSOCOM Biomedical Initiatives Steering Committee (BISC) meets quarterly to discuss projects and programs that support Special Operations Force medical issues. At times the BISC will provide "seed" money to start a program that can transition into a Department of Defense (DOD) laboratory for research, development and acquisition. Each Component Surgeon has a vote in the BISC process and has a representative that provides operational concerns or mission needs of their respective organization. The last meeting was held in April at AFSOC and the next meeting will be in July at USASOC.

The *hemostatic dressing* fielding had been delayed due to funding issues. Recently, the funds became available and the anticipated fielding date is now scheduled for late August 2002. The *chitosan dressing* is in the Advanced TechBase development program, and if all testing goes well and if the FDA approves the application, these bandages may be available in about six months. More information will be available in the next JSOM.

Recently a DOD panel met to discuss the use of *hemoglobin based oxygen carriers* (HBOC) in far forward locations. There are several agencies doing investigative research on HBOC. Most of the data that is available is not applicable to a military setting. This panel is making recommendations to their respective Surgeon Generals as to what courses of action should be taken before HBOC is used in far forward trauma situations. Once the panel report is final, it will be included in an update.

The BISC has been working with the Army Medical Department in the development of a one-handed tourniquet. After four years of "negotiating" one candidate has been selected. It is a rather simple device which may not be the 100% solution but will suffice while the search goes on.

Special Operations Forces Medical Handbook (SOFMHB): Change 2: On page 7-21, figure 7-2, after "perform escharotomies along the lines shown". Strike out the sentence: " Avoid making incisions across any involved joints, as

indicated by bold lines". Change the sentence to read: "Bold lines indicate the importance of incisions crossing any involved joints".

USSOCOM MEDTECH FY02 FUNDED PROJECT DESCRIPTIONS

CONTINUING PROJECTS

Advanced SEAL Delivery System/Underwater Breathing Apparatus ASDS/UBA

The lack of a DMO, dive supervisor, or master diver in the pressurized compartment of the ASDS will not allow the use of the NSW Dive Planner in its current format, so alternative means of calculating decompression obligation must be explored. The use of higher PPO₂s in the breathing mix should be considered. The lack of a boat air option makes the presence of an emergency open-circuit air capability in the UBA an important feature. This study will formally address these issues and new diving UBAs and techniques will be established as necessary.

BREATHING GAS MIXTURES EFFECTS ON DCS WITH AC-130H AND CV-22

Documents the impact on SOF mission performance due to DCS symptoms resulting from oxygen regulator "normal" settings on extended AC-130H/MV-22 operational missions. The study recommends DCS protection strategies for SOF aircrew.

CAPRINE ANALGESIA STUDY

Extensive soft tissue combat trauma injuries in remote locations may require a regimen that keeps wounded individuals pain free, or experiencing no more than minimal pain for five to seven days while allowing for second intention healing following extensive soft tissue trauma. Ideally, the pharmacological agents used will be shelf stable and can be administered in a field environment with a minimal amount of specialized equipment. This study will develop an animal model (caprines) to determine recommended protocols.

LASIK IN SPECIAL OPERATIONS BUD/S

Determines the effects of laser in-situ keratomileusis (LASIK) on SOF visual performance and determines the integrity of LASIK-treated corneas. This study will develop recommendations regarding LASIK and relative effects on SOF mis-

sion performance for BUD/S prospective candidates.

ORAL FLUOROQUINOLONE PROPHYLAXIS IN COMBAT TRAUMA

A number of potential issues for improving battlefield care for combat casualties were raised following Mogadishu. One of these issues was the need for antibiotics to be administered as soon as possible after wounding. This was not done in Mogadishu and there was a high incidence of wound infection that followed the prolonged evacuation time for the casualties in this engagement. The fluoroquinolone class of antibiotics offers the advantage of good bioavailability following oral administration combined with excellent spectrum of action. This study will make specific recommendations regarding the use of oral fluoroquinolones in both penetrating abdominal trauma and penetrating extremity trauma with associated fracture.

PROTECTIVE BARRIER SUBSTANCES FOR COELENTERATE ENVENOMATION

Coelenterate envenomation is a frequent hazard during Special Operations combat swimmer missions. The injury to an unprotected diver may range from minor skin irritation to cardiovascular collapse and death depending on the species of coelenterate involved and the extent of the envenomation. Wet suits and dive skins offer substantial protection against envenomation, but there have been recent concerns about heat injury from diving in very warm water in the Persian Gulf. This study will address the option of using a barrier substance that would protect the skin from nematocyst contact.

SPECIAL OPERATIONS COMPUTER ASSISTED MEDICAL REFERENCE SYSTEM (SOCAMRS)

Converts medical reference data pertinent to Special Operation Force (SOF) missions from hard copy to a self-contained, user-friendly multiple CD-ROM set. Updates to the SOCAMRS are through the research transformation of hard-copy biomedical data to optical media for USASOC, AFSOC, and NAVSOC. SOCAMRS provides a method for Special Operations medical personnel and medics to access relevant biomedical reference documentation during remote deployments.

WARM WATER DIVING STUDIES

Collects and analyzes data selected from the SOF community norm to assure accuracy in future studies in comparisons between performance of research subjects and the community norm, as well as between pre- and post-intervention scores in the Mission Related Performance Battery. Provides recommendations regarding thermal exposure limits, appropriate hydration/rehydration strategies, and clarification of storage limits of Sofnolime for SOF divers in warm water and hot air environments. This study determines relative effects of SOF diver dress in relation to warm water and extreme heat environments.

USSOCOM MEDTECH FY02 FUNDED PROJECT DESCRIPTIONS

2001-1 IMMERSION PULMONARY EDEMA

Studies incidents of immersion pulmonary edema (IPE) that are reported sporadically in the medical literature, but are being seen with increasing frequency in SEAL training. Medical personnel at the Naval Special Warfare Center estimate the current incidence rate at approximately 30 cases per year, with the preponderance of cases occurring in first phase. This incidence exceeds any other series reported in the medical literature. Affected individuals present with dyspnea, cough, rales, and hemoptysis. Chest radiography shows the classical picture of pulmonary edema and pulse oximetry demonstrates decreased hemoglobin oxygen saturation. The condition typically resolves without sequelae but may progress, especially during Hell Week. Some individuals have been dropped from BUD/S training because of recurrent episodes and the possibility of permanent injury to the students must be considered.

2001-2 PULMONARY O₂ LIMITS

Studies the feasibility of extending the daily maximum oxygen exposure limit of SDV Teams from four hours to six hours. This request stems from their desire to conduct two 3-hour dives per day during the Combat Swimmer portion of SEAL Tactical Training. This extension would significantly enhance the quality of Combat Swimmer training.

2001-3 TREATMENT STANDARDS FOR Decompression Sickness/Arterial Gas Embolism DCS/AGE

Special Operations are often conducted in remote areas where there may be a significant delay in access to recompression facilities for the victims of decompression sickness and gas embolism, with an accordingly higher probability of severe or refractory disease as a result. This study forms a standing UHMS committee to review the literature on treatment of decompression sickness and gas embolism and make recommendations for therapy based on the best clinical series, case reports, and animal studies available. Special emphasis in this review will be placed on the pre-recompression phase of treatment, which may be prolonged in Special Operations and recommendations for specific animal trials that will study the most promising new treatment modalities or otherwise enhance SOF ability to treat dysbaric disorders will be provided.

2001-4 MISSION RELATED PERFORMANCE MEASURES UPGRADE

A microprocessor supported standard battery of tests to measure these factors was constructed and named the SOF Mission-Related Performance Measures (MRMP). The SOF MRMP was then validated in field-testing. It has since been used to measure changes in performance resulting from a variety of physical stressors and physiological enhancements. The equipment and software used in the SOF MRMP is now in need of upgrading. There is also a need to enhance the exportability of this system to other laboratories that are performing studies in support of SOF. This study upgrades the marksmanship weapons and controlling system, the central data acquisition software, the individual cognitive battery laptops, the cognitive battery software and employs an individual who will be available to take the MRMP battery to other labs and field locations where required.

2001-6 IMPROVING SOF MISSION PERFORMANCE (MSN COMMANDER TRAINING PACKAGE)

Conducts a review of the medical literature and material in the Special Operations Computer-

Assisted Medical Reference System (SOCAMRS) and prepares a concise set of specific recommendations regarding techniques for SOF units during operations. The recommendations address a number of SOF specific operations, and for each type of SOF operation, provide a list of recommendations regarding strategies to optimize SOF operator performance. The results will then be made available to SOF mission commanders in a format suitable for training at the unit level.

2001-8 TELECONSULTATION IN SOF

With SOF units deployed worldwide, SOF physicians, PAs, and combat medical personnel often find themselves in remote locations with little medical support. Should a SOF operator become critically ill or injured, difficult decisions regarding diagnosis, treatment, and evacuation of the casualty must at present be made without access to medical specialists and subspecialists who could provide valuable guidance concerning the management of the patient. The presence of worldwide satellite communications and DOD medical specialists who can be reached through a pager make it possible to overcome this shortfall. This study provides specific recommendations on the feasibility and desirability of establishing this capability for SOF and develops a presentation suitable for briefing SOF Commanders on the results of this project.

2001-10 OPERATIONAL USE OF ADRAC (ALTITUDE DECOMPRESSION SICKNESS RISK ASSESSMENT COMPUTER) FOR CV-22

Alternative methods to prevent DCS, other than pre-oxygenation at ground level have been published, but lack a body of experience to validate proposed procedures. A means to record, organize, and accumulate this experience is needed, especially in SOF aircraft which expose aircrew and passengers to high altitude. To fully exploit the operational capabilities of these aircraft, the capability is needed to document and assess DCS events while in the operational setting in order to refine the efficacy of planned DCS prevention strategies. In addition, the capability is needed to enable operators to predict DCS risk in order to permit enroute mission planning. This study develops methodologies of

using ADRAC to record the flight exposures experienced by SOF aircrew and passengers, index when DCS symptoms appear and resolve, and

sense/record the percent oxygen presented in the breathing gas on a real-time basis to develop treatment protocols for DCS for SOF.



Dive Medical Brief: A Comprehensive Review for the Special Forces Dive Medical Technician

Part Two

Eric D. Martin, DO

Abstract

Part Two is continued from the Winter Edition and continues to provide an overview/review of dive medicine for the Special Forces dive medical technician with a body of information organized in a concise format that addresses the prevention and treatment of various diving medical injuries. Mechanisms, treatments, etc. are abbreviated with deference to the US Navy Dive Manual.

The dive medical brief focuses on five areas: 1) Dive physics and gas laws, 2) Barotrauma, 3) Pulmonary over inflation injuries, 4) Decompression sickness, and 5) Gas related injuries unique to closed circuit diving.

The positive response from combat divers and dive medical technicians who have been trained suggests that this medical brief could serve as a standard review and recertification program for all Special Forces dive medical technicians and would serve to benefit sister services as well as a review.

OBJECTIVES-

1. Describe how to recognize the signs and symptoms of in-water diving injuries.
2. Explain how to render the appropriate medical treatment, including recompression therapy.

Complete Test on Page 49--Answer sheet on Page 47

Completion of this article and test offers 1.25 CME and 1.50 CNE/CEH.

Disclosure:

Dr. Eric D. Martin has indicated that his presentation will include discussion of commercial products or services. However, he has no significant financial relationship with a commercial entity whose products or services are related to the subject matter of the topic he will be addressing.

OVERVIEW OF DECOMPRESSION SICKNESS (DCS)

Nitrogen (N₂) and oxygen (O₂) in the compressed air breathed by scuba divers manifest effects because of the increased tissue partial pressure of the inert nitrogen gas. In addition, there are direct effects of bubble evolution caused by liberation of nitrogen from tissues that have become supersaturated during the dive.^{19, 20} This results in some nitrogen coming out of solution as bubbles. These bubbles can be interstitial, intralymphatic, or intravascular.

Decompression sickness, also known as “the bends,” is the result of a series of pathophysiologic responses to the evolution of dissolved tissue gases precipitated by changes in the ambient pressure. Bubbles released from solution by the too

rapid reduction in ambient pressure either impede blood flow due to direct vascular occlusion, cause blood chemistry changes, or stretch and damage tissues. The symptoms can range from innocuous skin itching to central nervous system compromise.

PATHOPHYSIOLOGY OF DCS

Following Henry’s Law, the amount of gas dissolved in tissues is proportional to partial pressure. When a mixture of gases is inspired, the amount of each gas that becomes dissolved in tissues is proportional to the partial pressure of each gas and the rate at which the gases are either removed or metabolized within the tissue.² Ambient pressure governs the rate and total amount of gas dissolved in tissues. Two factors determine

the amount of nitrogen gas dissolved in tissues with changes in ambient pressure: 1) the depth of the dive, 2) the duration of the dive.

Studies indicate that bubble formation primarily occurs in the post-capillary venules because they comprise the lowest pressure system in the vascular network. Arterial bubbles do occur in right-to-left shunts such as a patent foramen ovale (PFO) or arteriovenous malformations in which venous blood mixes with arterial blood (bypassing the lungs). The lungs are very good filters of bubbles; however, they may be overwhelmed by massive amounts of bubbles, leading to pulmonary DCS (chokes). Clinically this looks like a pulmonary embolism and may result in bronchiolar constriction, peribronchiolar edema, hypoxia, and adult respiratory distress syndrome (ARDS).²¹

Tissue metabolism removes oxygen, but nitrogen remains.² Factors that affect nitrogen uptake are exercise, temperature of water, hydration, alcohol, smoking, and body fat percentage.^{22,23} Exercise increases gas uptake and elimination by increasing the respiratory rate, cardiac output, and tissue perfusion. Gas temperature and the diver's state of hydration also affect the rates of gas uptake and elimination. Under increased ambient pressure, gases become dissolved in different tissues at varying rates because each tissue has its characteristic rate of gas uptake and perfusion. For example, gases become rapidly dissolved in muscle but are dissolved very slowly in bone and fat. Fat can take on a vast amount of nitrogen because nitrogen is five times more soluble in fat than in water. Because gas elimination follows a similar time course, nitrogen is eliminated from fat tissues very slowly, and supersaturation with subsequent bubble formation may occur if the ambient pressure is reduced rapidly.

Complications of supersaturation that result in the liberation of inert nitrogen from tissues lead to bubble formation causing direct mechanical and non-mechanical effects. The direct mechanical effects result from intravascular occlusion causing ischemia distal to the site of obstruction. Also an intravascular bubble acts as a nidus for platelet adherence and aggregation, further obstructing blood flow.

The non-mechanical effects result from an inflammatory cascade mediated by platelet aggregation and endothelial cell damage that releases

collagen and tissue factor activating the coagulation cascade. This activation leads to fibrin clot formation. In addition, thrombus formation occurs *de novo* as a result of platelets and fibrin amalgamating. Moreover, cytokines, which include interleukins, are chemo attractants for leukocytes. This activates the complement pathways both classical and alternate. Capillary damage and leakage are due to histamine, bradykinin, prostglandin, leukotriene, and interleukin release. Thromboxane production, a by-product of arachidonic acid metabolism, causes local vasoconstriction.

Tissue hypoxia and injury are direct manifestations of both the direct mechanical and non-mechanical effects of bubble evolution in the body. Bubbles are "bullets to the body" causing damage everywhere they go.^{24, 25}

Several factors contribute to delayed biochemical effects. Proteins may be denatured and enzymes activated. The denaturation of lipoproteins leads to free fatty acid release from cell membranes, releasing fat emboli into the bloodstream. Inflammation, sludging/clotting of blood, and edema may also occur.^{24, 25} Coating of red blood cells with denatured protein can cause red blood cell clumping, thereby increasing blood viscosity and promoting stasis. Because venous blood flow is already slow, further slowing can result in clotting with an increase in capillary filtration pressure leading to interstitial edema and loss of plasma volume.

CLINICAL MANIFESTATIONS OF DCS

Symptoms are evident within an hour of surfacing in approximately 80% of patients and within four hours in more than 95%. In contrast, the occurrence of a gas embolism is presumed when the symptoms arise within ten minutes after surfacing. Decompression sickness has customarily been divided into two categories (See Decompression Sickness Chart). Type I (musculoskeletal) manifests as limb pain or with skin or lymphatic involvement. Type II (neurologic, spinal, pulmonary, or inner ear), also known as serious decompression sickness, encompasses all other symptoms. The diagnosis of any type of DCS mandates immediate transport to a recompression facility.

DECOMPRESSION SICKNESS

CLASSIFICATION & COMPARISON WITH GAS EMBOLISM

Disorder	Symptom Complex	Pathology	Treatment
Type I "Pain-only bends"	Pain confined to an extremity, usually peri-articular, aggravated by movement, often relieved by direct pressure (ie, with sphygmomanometer cuff)	Bubble evolution within a non-distensible tissue (ligaments, tendons, bone)	Table V recompression; Observe for serious symptoms
Skin pruritis	Intense itching, begins during decompression or shortly thereafter, primarily with air dives in chamber, direct pressure may relieve sickness)	Gas forced into sweat and sebaceous glands during dive evolves as bubbles on ascent (not true decompression)	No treatment
Skin marbling	Itching, discoloration, cyanosis over affected area, blanches on pressure	Subcutaneous bubble evolution causing local venous stasis	Table V recompression; Observe for serious symptoms
Lymphatic symptoms	Lymph node pain and swelling, tissue edema distal to affected nodes	Bubble accumulation within nodes, mechanical obstruction	Table V recompression; Observe for serious symptoms
Type II All other pain	Pain elsewhere than extremities	May represent referred pain from visceral sites or spinal cord involvement	Table VI recompression; Observe for other symptoms
Central nervous system	Any hard or soft neurologic symptom; paralysis, sensory deficit, seizures, extreme fatigue, change in personality, headache, visual symptoms, etc.	Bubble evolution or accumulation anywhere in brain, spinal cord, or cranial nerves	Table VI recompression
Labyrinthine	A CNS symptom also known as the "staggers"; any 8th nerve symptom, ie. unsteadiness, vertigo, tinnitus, deafness, nystagmus	Bubbles obstructing labyrinthine nutrient arteries, Endolymphatic bubbles	Table VI recompression
Pulmonary (chokes)	Increasing substernal burning pain begins within minutes of surfacing, cough and progressive dyspnea follow; often associated with respiratory failure and shock	Accumulation of bubbles within pulmonary arterial tree	Table VI
Gas Embolism	Any CNS symptom on ascent or within ten minutes after surfacing, can be rapidly progressive to circulatory collapse	Alveolar overpressurization with resultant free arterial air in cerebral and coronary circulation	Table VIA recompression; Life support measures as required

TYPE I

Type I DCS is the more common variety and it is the classic presentation of "pain-only bends." This includes pain confined to the arms or legs that may be aggravated by movement and relieved by direct pressure on the area, i.e., with a blood pressure cuff. The pain, which is usually peri-articular, involves the upper extremities three times more frequently than the lower extremities in divers and ranges from mild discomfort to severe pain.

There are many forms of skin related DCS: pruritis alone, various rashes, and skin marbling

(cutis marmorata). Gas entering the sweat and sebaceous glands during compression is thought to form pruritic bubbles as the diver surfaces. The itching of skin bends must be differentiated from the tingling of paresthesias or hypesthesia associated with spinal Type II DCS. Pruritis is not considered a true form of decompression sickness and resolves rapidly without treatment.

Marbling of the skin occurs when subcutaneous bubbles cause venous stasis. The symptoms most commonly occur over the shoulders and begin as intense itching quickly followed by discoloration of the skin. Erythema progresses to

cyanotic mottling which resolves rapidly when treated with recompression. Marbling is a true form of decompression sickness, indicating that supersaturation has occurred and may be a harbinger of systemic involvement and other more serious symptoms. Although rare, isolated lymphatic symptoms occasionally occur as a result of lymphatic obstruction by bubbles. There is usually pain and swelling of the lymph nodes with variable edema of the tissues normally drained by the affected nodes. Although recompression usually produces relief of the pain, the swelling may persist for days.

TYPE II

Type II decompression sickness includes all of the manifestations of evolved gas pathology. Pain that occurs in the head, neck, and torso region should be classified as a Type II symptom and treated accordingly. No single central nervous system symptom complex is characteristic for decompression sickness. Any neurologic symptom can be associated with the intra and extra vascular evolution of bubbles anywhere in the nervous system. Spinal cord symptoms are most common in divers while cerebral symptoms predominate in aviation personnel. Paresthesia is a very common presenting symptom of a spinal cord “hit” and may progress to ascending numbness. Cord lesions are most common in the lumbo-sacral region and can be associated with bowel and bladder disfunction and, occasionally, priapism.

Cerebral decompression sickness can present with such signs as seizures, hemiplegia, visual disturbances, blurred vision, headaches, unusual fatigue, a sense of detachment from the surrounding environment, inappropriate behavior, or any number of other variable presentations.²⁷

Although cranial nerves are not usually affected, if they are, labyrinthine or inner ear decompression sickness, also known as the “staggers,” is a common etiology in saturation divers. Immediate treatment is important because prolonged obstruction of the small nutrient arteries supplying the inner ear structures (cochlea or the vestibular apparatus) can result in permanent damage.

Pulmonary decompression sickness, “the chokes”, presents as a triad of substernal pain, cough, and dyspnea and often occurs within minutes of surfacing. The substernal pain is characteristically described as a burning sensation exacerbated by

deep inspiration. The cough initially is episodic and may progress to virulent, uncontrollable paroxysms. Respiratory failure and shock are often the terminal events in pulmonary DCS if left untreated.

Prevention and Treatment

Prevention of DCS is best accomplished by strict adherence to the U.S. Navy Dive Tables and ascending at a rate no greater than one foot per two seconds. Early recognition and treatment of decompression sickness is essential for resolution without sequelae. Patients with Type I DCS will require reassurance, oral fluids, and immediate transport to a recompression facility while on 100% O₂. Patients with Type II DCS will usually require more vigorous and therapeutic measures. Recompression is the mainstay of decompression sickness therapy. A specially trained treatment team at the recompression facility will handle the details of recompression therapy. The vast majority of treatments are performed using U.S. Navy treatment tables and dry chambers.

The three objectives in recompression therapy are to reduce the size of the bubble, promote bubble reabsorption, and prevent further bubble evolution. Reduction in bubble size is important initially and is accomplished purely by the increase in ambient pressure. This results in relief of vascular obstruction and tissue distortion enhancing reperfusion and oxygenation. Intravascular bubbles are compressed promoting distal migration and reduction in the size of the ischemic area. Bubble reabsorption is also enhanced by the increase in ambient pressure as the partial pressure of the nitrogen within the bubble exceeds the partial pressure of nitrogen in surrounding tissue. This increases the diffusion drive. Breathing 100% oxygen washes out tissue nitrogen and enhances diffusion by further widening the nitrogen partial pressure difference between the tissue and the bubble. Oxygen enhances tissue oxygenation, reduces cerebral edema, and washes nitrogen out of tissues.

Intravenous fluid therapy is a necessary adjunct in all cases of Type II decompression sickness because fluid loss, hemoconcentration, and increased blood viscosity promote vascular occlusion. A crystalloid, such as ringer’s lactate or normal saline, should be used as the initial choice. A patient’s blood pressure, hematocrit, and urinary output should be monitored and caution should be taken to avoid pulmonary edema. In case of a spinal

cord DCS injury resulting in a hemorrhagic infarct of the white matter, steroids should be administered in high dosages initially. Aspirin should be avoided due to the risk of expanding the hemorrhagic area even though its action inhibits platelet kinetics.²⁷

After Treatment

Follow advice as per the Navy Dive Manual Chapter. 21, 6.1-6.6.

INJURIES PERTAINING TO CLOSED-CIRCUIT DIVING

MK 25/DRAEGER LAR V UNDERWATER BREATHING APPARATUS (LAR V UBA)

OXYGEN AND DIVING

Air contains approximately 21% oxygen (O₂). A partial pressure of oxygen (ppO₂) between 60 to 80 mmHg combined with an oxy-hemoglobin saturation of 90% is necessary for adequate tissue oxygenation. Sixteen per cent oxygen in ambient air is the minimal percentage required to function normally.

OXYGEN TOXICITY OVERVIEW

Oxygen (O₂) toxicity is defined as the destructive effects on tissue resulting from over exposure to oxygen. The exact mechanism is unknown; however, there are several theories. One theory is that the formation of oxygen radicals (superoxides, hydrogen peroxides, hydroperoxides) is very reactive and directly cytotoxic. A second theory is that inactivation of oxidative enzymes leads to a decrease in the body's ability to form high-energy phosphate bonds. A final theory is that vasospasms lead to increased waste products.

OXYGEN TOXICITY AFFECTING THE CENTRAL NERVOUS SYSTEM

End organ damage is a direct consequence of O₂ toxicity. The pulmonary system and central nervous system (CNS) are primarily affected. O₂ toxicity usually affects the diver's CNS sooner than the lungs unless prolonged repetitive exposures to supranormal O₂ partial pressures are occurring.

Signs and Symptoms

CNS oxygen toxicity follows the Paul Bert Effect in which high doses of 100% O₂ over short-term exposure produce deleterious side effects involving the central nervous system.²⁷ The list of

symptoms manifested by the toxicity can be remembered by the acronym VENTIDC:

V - vision: Loss of peripheral vision causing tunnel vision

E - ears: tinnitus

N - nausea

T - tingling, muscle twitching

I - irritability

D - dizziness

C - convulsions

Prevention and Treatment

To prevent this condition, the diver should remain within the US Navy closed-circuit O₂ exposure limits as depicted in chapter 18 of the US Navy Dive Manual. To treat the condition, the diver should perform a controlled ascent to the surface. Once the diver reaches the surface, he should close the dive surface valve (DSV) and be removed from the air source. The diver should then be required to breathe ambient air until his symptoms abate.

PULMONARY OXYGEN TOXICITY

Pulmonary oxygen toxicity corresponds with the Lorraine Smith Effect, which states that low dose oxygen partial pressures with long-term exposure will cause adverse changes in the lung parenchyma including pulmonary edema and protein exudative formation leading to pulmonary fibrosis. This causes a restrictive type of lung disease.²⁷ Partial pressures of oxygen (0.6 ATA) can cause damage to the lung tissue. The reason pulmonary toxicity occurs at a lower partial pressure of oxygen is that the oxygen is in direct contact with pulmonary tissues.

The pathophysiology of pulmonary oxygen toxicity occurs in two phases, the exudative (early) phase and the proliferative (late) phase. The exudative phase is typified by congestion, alveolar edema, intra-alveolar hemorrhage and hyaline membrane formation and induces no permanent changes. The proliferative phase is characterized by the thickening of alveolar and inter-lobular septa and edema with alveolar hyperplasia.²⁸ These changes may be irreversible.

Signs and Symptoms

The signs and symptoms of pulmonary oxygen toxicity are mild substernal irritation exacerbated by inspiration, nonproductive cough, dyspnea, decreased lung volume (specifically vital capacity), and decreased lung compliance. The toxic effects

of oxygen and the lung parenchyma may lead to a clinical picture of adult respiratory distress syndrome (ARDS).²⁹

Prevention And Treatment

To prevent this condition, a diver should follow the US Navy Dive Manual guidelines for O₂ closed-circuit excursion limits.²⁶ To treat this condition, the diver should ascend in a controlled manner and be removed from the oxygen source. Airway management may be necessary. Ventilator support may be required due to severe respiratory distress.

HYPOXIA

Hypoxia results from lack of O₂ to the tissues. This condition arises from an improper purge procedure which leaves too much N₂ in the breathing loop, or occurs from filling the bottle with other than O₂. Hypoxia occurs within the first five minutes of a dive.

Signs and Symptoms

Signs and symptoms range from loss of consciousness, dizziness, irritability, confusion, and convulsions.

Prevention and Treatment

To prevent hypoxia, the diver should perform a proper purge procedure. To treat the condition, the diver should control his ascent. Once on the surface, the diver should close his dive surface valve. The dive medical officer or technician should provide the diver with 100% O₂ via a non-rebreathing mask.

HYPERCAPNEA (CO₂ TOXICITY)

Hypercapnea or CO₂ toxicity is caused by CO₂ absorbent failure, over-breathing the rig, and inadequate ventilation (hypoventilation and skip breathing).

Signs and Symptoms

The signs and symptoms of this condition are increased respiratory rate, labored breathing, headache, and confusion.

Prevention and Treatment

The signs and symptoms for hypoxia and hypercapnea are similar. To distinguish between the two conditions, the time of onset is the critical factor. Hypoxic symptoms will generally occur

earlier in the dive whereas hypercapnic symptoms will occur towards the end of the dive.

To prevent hypercapnea, the diver should avoid over-breathing his rig and should breathe in a controlled, relaxed manner. The diver should attempt to swim at a rate of 100 meters per every three minutes. To treat the condition, the diver should control his ascent. Once on the surface, the diver should close his dive surface valve. The dive medical officer should remove the diver from the gas source and have the diver breathe ambient air.

DRAEGER EAR (Middle Ear Absorption Syndrome)

Another complication of closed circuit diving is middle ear absorption syndrome. Oxygen is absorbed over a period of time into the middle ear cavity. Following the dive, a negative pressure develops. If the Eustachian tube does not open spontaneously, the negative pressure relative to ambient pressure remains in the middle ear cavity.

Signs and Symptoms

The signs and symptoms of this condition are minor ear pain, hearing loss, feeling of fullness, and crackling sensations due to fluid build up. These symptoms occur hours post dive or upon waking the next morning.

Treatment

The diver should perform gentle Valsalva maneuvers over the next 24 hours in order to ameliorate the feeling of fullness in the middle ear cavity aiding in equalization.

CAUSTIC COCKTAIL

The final complication of closed circuit diving is known as a caustic cocktail. The condition results when water leaks into the CO₂ absorbent canister mixing with CO₂ absorbent, creating an alkaline solution. The solution produces a chemical injury when it comes in contact with the mucosa of the oropharynx. Significant edema can result in the posterior oropharynx and laryngeal region producing stenosis of the airway leading to partial or even complete airway obstruction.

Signs and Symptoms

Signs and symptoms are choking and gagging, foul taste, and burning of the mouth and throat. Inspiratory stridor may occur if there is

marked swelling and edema.

Prevention and Treatment

To prevent this condition, the diver should keep his mouth in proper contact with his mouthpiece and check to make sure that his diver surface valve is not open when his mouthpiece is in the water. To treat this condition, the diver should rinse his mouth thoroughly with fresh water. The diver may use viscous lidocaine to temporarily anesthetize his mouth. Precautionary measures should be taken to ensure patency of the airway. If significant inspiratory stridor is detected, the dive medical officer or technician may need to secure a definitive airway. If significant laryngeal edema is noted, intravenous steroids may be used to help reduce the swelling. The dosage is Solu-medrol 60 mg IV every eight hours.

CONCLUSION

The 10th Special Forces Group has instituted this dive brief as part of the initial dive supervisor's briefing prior to putting any diver in the water. Used throughout the scuba community, the Dive Medical Brief would provide dive medical technicians with a common language and a succinct reference available to all divers in a concise format designed to address the causes of dive injuries and their prevention and medical treatment.

A CD-ROM version of the Dive Medical Brief, augmented by photographs and color, is available upon request from the 3rd Battalion 10th Special Forces Group at Fort Carson, Colorado. Contact the Battalion Medical Section at COMM 719-524-1740 or DSN 883-1740. This version can be used for presentation as a part of refresher training for a dive medical technician seminar and for a diver requalification.

Acknowledgment

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REFERENCES- see Fall Edition Vol. I Edition 3 for Part 1 references

2. Arthur DC, Margulies RA: A short course in diving medicine, *Annals Emergency Medicine*, June 1987; 16:689-701. **From Part 1.**
19. Nims LF: Environmental factors affecting decompression sickness, *Decompression Sickness*, pp 14-35. New York, WB Saunders 1951.
20. Elliott DH, Waldenbeck IM: The pathophysiology of decompression sickness, in Bennell PB, Elliott DH (eds) *The Physiology and Medicine of Diving and Compressed Air Work*, pp 76-92. Baltimore, Williams and Wilkins, 1975.
21. Hills BA: Fundamental biophysical aspects of decompression sickness, DTIC AD - A069-998, pp 112-114. Alexandria, Virginia Defense Technical Information Center, 1979.
22. Tobias CA: Studies on skin temperatures and circulation in decompression sickness, *American Journal of Physiology*, 1947; 149:626-633.
23. Gay JS: Constitutional factors affecting susceptibility to decompression sickness, *Decompression Sickness*, pp 112-115. New York, WB Saunders, 1951.
24. Straus RH: Decompression sickness, in Strauss RH (ed) *Diving Medicine*, pp 63-82. New York, Grune and Stratton 1976.
25. Hills BA: Mechanical vs ischemic mechanisms for decompression sickness, *Aviation Space Environment Medicine*, 1979; 50:363-367.
26. *US Navy Diving Manual*, Volume I, Rev. 3 NAVSEA 0994-LP-001-9010, Washington DC, Government Printing Office, February 1993.
27. Rutkowski, R: Lecture. Advanced Undersea Medicine Course. U.S. Navy, Key Largo, FL, December 2000.
28. Bennett PB, Elliott DH: *Physiology and Medicine of Diving*, pp 87-126. San Pedro, CA, Best Pub Co, 1993.
29. Bove AA, Davis JC: *Diving Medicine*, pp 150-164. Philadelphia, WB Saunders, 1990.

Centipede Envenomation

Sean P. Bush, MD; Bradley O. King, MD; Robert L. Norris, MD; and Scott A. Stockwell, PhD

Abstract

Five episodes of envenomation by centipedes in two patients are reported. These arthropods are fast-moving, frightening in appearance to some, and may display aggressive behavior. However, stings from these centipedes, like most found worldwide, caused no serious morbidity or mortality. Common effects included intense local pain, erythema, induration, and necrosis, as well as mild constitutional symptoms. All resolved without sequelae. Treatment included pain control, wound care, and tetanus immunization.

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Introduction

Centipede stings occur in the warmer climates throughout the world. The literature contains few reports concerning these envenomations, the majority describing a painful but benign syndrome. We report 5 recent occurrences of centipede stings in two patients, and update treatment recommendations based on the most recent literature.

Case 1

A 30-year-old patient presented to the emergency department with excruciating pain to his right hip. He had been putting on his jeans when he felt the sudden onset of burning pain. On examination, he saw an arthropod under the waistband, where it was still "biting" at the skin with repetitive jabbing motions of its head. On arrival to the emergency department 20 minutes later, the patient produced a captured 7-cm centipede (*Scolopendra* species), which was still thrashing vigorously. The patient's burning pain was getting more intense and was radiating throughout the buttocks and down the right leg. Other local symptoms included numbness and tingling in the right hip and weakness in the right leg. The patient also complained of dizziness, near syncope, and nausea.

Vital signs included blood pressure of 149/83 mm Hg, pulse of 76 beats per minute, breathing rate of 22 breaths per minute, and oral temperature of 37.6°C. He was anxious, writhing, and rigidly holding his right leg in an extended, contorted position. There was a 12-cm round area of swelling over the right hip. Within that area was a 3 × 2-cm wheal-like area of raised edema, where 2 tiny puncture marks, 3 mm apart, were visible. This area was extraordinarily tender, with the patient jumping at the slightest touch. Examination was otherwise normal.

The wound was cleaned, and tetanus vaccination given. The patient received 60 mg intramuscular ketorolac, 1 mg intravenous lorazepam, and 50 mg intravenous diphenhydramine. Morphine sulfate was titrated to relief of pain, with 20 mg being given intravenously over an hour. The patient was discharged with wound care instructions and a prescription for acetaminophen with hydrocodone after 6 hours of observation in the emergency department. He did not return for recommended follow-up in two days, but was contacted by telephone at one week and one month. His pain had resolved after two days; however, he developed a necrotic area approximately three cm

in diameter, which sloughed and healed spontaneously over a month's time.

Case 2

On February 5, 1998, one of the authors (S.S.), then 36 years old and in excellent health (on no medications), was stung by an approximately 13-cm specimen of *Scolopendra heros*, the giant desert centipede. The sting occurred to the ulnar aspect of the left little finger at the distal interphalangeal joint. There was immediate onset of pain, experienced as a burning discomfort at a level of "6" on a verbal pain scale of 1 to 10. Additional pain was noted sporadically in other parts of the hand within a few minutes. Within 10 minutes, there was also acute pain in the left elbow, both axillae, the clavicular area, and the neck. There was moderate soft tissue swelling of the affected finger within approximately 30 minutes, to a degree to limit range of motion by about 50%. At approximately 45 minutes following the sting, there was a red streak running from the involved digit, proximally along the dorsum of the hand to the elbow. This line was not painful. There was a general sensation of being "unwell." The victim did not take any medications for pain, but did lie down to rest. The red streak faded after another hour, and the pain and swelling rapidly began to subside. Within 3 hours the victim felt fine (almost "euphoric"). There was no necrosis and no permanent sequelae.

Case 3

While handling an approximately 20-cm *Scolopendra subspinipes* (a species native to much of Asia and the Pacific islands; Figure 1) for a television interview, the same victim as in case 2 (S.S.) was stung once on his left index finger at approximately 10 AM (October 18, 1999). The patient had



Figure 1. Photograph of *Scolopendra subspinipes* (a species native to much of Asia and the Pacific islands). Photograph by Scott Stockwell, PhD

been stung by this species once previously with minimal reported effects (little pain and no swelling). On this occasion, however, there was nearly immediate onset of burning pain that spread over the next several minutes into the back of his hand. Blood oozed from 2 puncture wounds for several minutes, and swelling and erythema began at the site. Two puncture wounds were present, approximately 1.5 cm apart. Pain engulfed the entire hand. A burning sensation was present at the leading edge of the soft tissue swelling with severe throbbing at the sting site. By 10:30 AM, the



Figure 2. Case 3. *Scolopendra subspinipes* sting to the left hand at 45 minutes. Note the puncture wounds and the mild erythema. Photograph by Scott Stockwell, PhD

swelling in the finger had become pronounced (with approximately 50% loss of flexibility) and had spread into the dorsum of the hand. Pain was now present in the left elbow and axilla.

By 11 AM, pain was present to some degree in all the digits of the hand, and pain in the wrist, elbow, and axilla was pronounced. Swelling continued over the next several hours, peaking at approximately 3:30 PM (with swelling of all 5 digits and

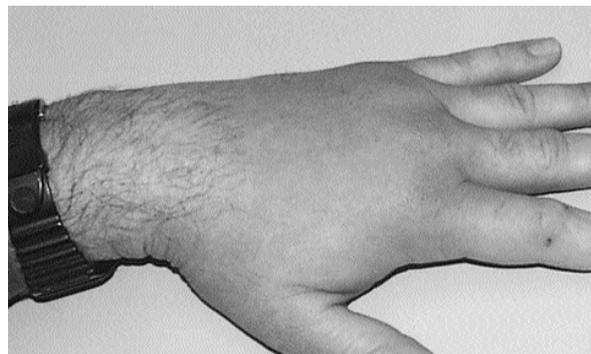


Figure 3. Case 3. Photograph taken at 7 hours and 15 minutes following envenomation. Note the progression of soft tissue swelling and erythema. Photograph by Scott Stockwell, PhD

the dorsum of the hand and proximal wrist) (Figure 3). By this point, pain was a "10" on a verbal scale of 1 to 10. There was a severe "sprained" sensation from the elbow to the hand. The patient self-medicated with acetaminophen (2000 mg per os), but noted no relief of the pain. He got some pain relief by applying local heat (by wrapping a heating pad around his hand with an elastic bandage, covering this with a large bath towel, and wedging his arm between 2 pillows to increase the insulation). (Recreation of this technique later demonstrated an attained temperature of 41°C at the skin surface.) He maintained this heat application for approximately six hours and noted his pain level was reduced to approximately a "five." The patient was unable to find a position of comfort and had a restless night secondary to the pain. On arising at 4 AM the next morning (October 19, 1999), the patient noted significant improvement in the pain, and he experienced nearly complete resolution by 7 AM. Swelling persisted, however, and as the patient used the hand throughout the day, mild ecchymosis developed over the proximal phalanges and metacarpals. The swelling began to recede by early that afternoon and was resolved by the morning of October 20, 1999. Pain did not recur and there was no necrosis.

Case 4

At approximately 4 PM on October 20, 1999, the same patient as described in cases two and three (S.S.) was stung again (when an emergency occurred in his animal facility) by the same *S subspinipes* that had stung him two days previously. On this occasion, there were two stings in the same location on the dorsum of the right hand between the first and second metacarpophalangeal joints. There were four puncture wounds. Again, there was rapid onset of pain and swelling in the hand, with radiation into the upper arm. The patient went home and began heat application (at approximately 6 PM) in similar fashion to his prior sting. He applied the heat for approximately 12 hours and again noted a definite decrease in his pain level. The patient had a light dinner at 5 PM so that he could try ibuprofen for the pain. He took 800 mg of ibuprofen at 5:45 PM. This was followed by some slight nausea, but no vomiting. Once again, a pain similar to a severe "sprain" was most bothersome. This was not significantly relieved by the ibuprofen. As with the previous sting, there was significant swelling of the digits and hand. By 8 AM the next morning (October 21, 1999), the pain was significantly improved, and it had completely

resolved by noon. The swelling took longer to dissipate, resolving by 4 PM on October 22, 1999. Again, there was no tissue loss.

On November 10, 1999, the patient noted recurrence of swelling at the sites of his envenomations on October 18 and 20. The left index finger swelled most noticeably (until flexibility was reduced by approximately 50%). Swelling in the right hand was less noticeable. The swelling was associated with intense local itching. The swelling and pruritus lasted approximately one week. There also appeared an approximately 1.5-cm hardened "lump" below the skin at the site of the stings on the right hand.

The victim visited a dermatologist in late November 1999 for the lump on his right hand. The dermatologist prescribed 0.05% clobetasol propionate ointment to be applied over the site of the lump. This treatment was used sporadically without obvious effects. The lump ultimately regressed by early February 2000.

Case 5

On February 9, 2000, at approximately 9:05 AM, S.S. was stung again while preparing for a class. The centipede was a smaller (approximately 10-cm) *S heros*, and the sting occurred to the tip of the victim's left thumb. There was an immediate, burning pain (rated "six" out of 10) at the site of the sting. Within 1 minute the victim was experiencing a very sharp, piercing pain on the ventral side of the left mandible. The thumb itself seemed quite hot and red, but there was little or no appreciable swelling. At 9:15 AM, the victim experienced another area of sharp, piercing pain near the proximal tip of the left clavicle. He generally felt unwell and slightly "flushed." By 9:20 AM, there was a vague, altered "sensation" in the tip of the left forefinger that was not truly painful. This sensation spread to all the fingertips of the involved hand by 9:25 AM. There was now a sharp pain present in the first interdigital web space. By 9:40 AM, pain had spread to his palm and wrist, and there was some discomfort in the chest and along his chin. There was also a strange, "dull" pain at the proximal phalanx of the left thumb. At 10 AM, the remote pains began to subside. Within the next hour, as the victim was teaching his class, all symptoms subsided except for a lingering dull pain at the sting site, which persisted for approximately 12 hours. At no time did the finger or hand look abnormal (no red-

ness or swelling), and there was no necrosis or any other complications.

Other than the visit to a dermatologist mentioned in case 4, the victim (S.S.) did not seek medical care for any of his stings.

Discussion

Centipedes (class Chilopoda, phylum Arthropoda) are slender, multisegmented arthropods with one pair of legs per segment and one pair of antennae.¹ Their size may range from one to 30 cm, and coloration from bright yellow to brown-black. The first pair of legs has been modified into two sharp stinging structures connected to muscular venom glands (Figure 4).² Their range is worldwide in warm temperate and tropical climates, and in the United States they are found throughout the southern states, most commonly California, Arizona, Texas, Louisiana, Alabama, Kansas, and Georgia, as well as Hawaii.^{1,3} They prefer dark damp environments, such as undersurfaces of rocks and logs, but on rainy days often retreat indoors. Centipedes are



Figure 4. Centipede venom apparatus.
Photograph by Sean P. Bush, MD

nocturnal carnivores with a wide range of prey. These fast-moving arthropods use their venom to paralyze prey prior to eating. They primarily eat insects, although they are known to prey occasionally on slugs, worms, and small snakes.² There are even reports of some of the larger species killing and eating small birds, toads, and rodents.⁴ There are four orders: Geophilomorpha (soil centipedes--small, innocuous soil dwellers), Scolopendramorpha (trop-

ical or giant centipedes--known stingers), Scutigeraomorpha (house centipedes--fast, but delicate), and Lithobiomorpha (rock or garden centipedes--resemble small scolopendramorphs in appearance with many anecdotal reports of stings). All orders are venomous. The *Scolopendra* are the largest centipedes, and therefore probably the most dangerous. On an encounter basis, lithobiomorphs and scutigeraomorphs (both are widely distributed even in temperate areas) are probably more common. *Scolopendra* range from eight to 15 cm, and *S. heros* can achieve lengths of up to 20 cm or more. *Scolopendra* usually have a yellow-brown body with orange and blue cephalic/caudal parts.

Venom from *Scolopendra* species has been analyzed by a number of authors; however, the exact compounds are still unknown. The presence of 5-hydroxytryptamine (serotonin) in the venom of *Scolopendra* has been demonstrated by many investigators.⁵⁻⁷ Gomes et al,^{6,7} used animal models to demonstrate significant cardiovascular effects of *Scolopendra* venom, mediated by histamine and a cardiodepressant factor designated as Toxin-S. They

also reported the presence of a smooth muscle contractile agent, which was recently confirmed to have muscarinic activity.⁷⁻⁹ Proteinases and lipoproteins have also been reported to be active agents in the venom, and it is thought that the venom is a lipid-toxin complex, similar to that of scorpion venom, which facilitates local cellular penetration and absorption. A cytolyisin also has been isolated from venom of the giant desert centipede, *S heros*, potentiating both its local and systemic effects.^{1,3} Most authorities recognize the cardiodepressant Toxin-S to be the lethal factor (predatory), and the histamine/serotonin components to be pain mediators (defensive).^{7,10} The lethal dose for all subjects (LD₁₀₀) in juvenile mice

was found to be 0.01 venom glands per gram body weight, which might account for the lack of human lethality (extrapolating to humans, the contents of almost 1000 venom glands would be required for a fatal sting in an average adult).²

Centipede stings often occur as the victim is putting on clothes or while in bed. They usually release from the skin immediately, although there are reports of tenacious attachments requiring removal with a noxious agent such as alcohol and even sur-

gery.¹¹ There have been a number of centipede envenomation cases reported worldwide describing a benign, albeit painful, syndrome.^{12,13} The most common scenario includes moderate to severe local symptoms associated with mild systemic symptoms. Local symptoms include pain, erythema, edema, lymphangitis/lymphadenitis, weakness, and paresthesias. Skin necrosis may occur at the site of envenomation during the weeks following the sting, but rarely becomes extensive and heals spontaneously. Systemic symptoms may include anxiety, fever, dizziness, palpitations, and nausea. Excluding local skin necrosis, findings usually resolve within two days without sequelae; however, exceptions should be mentioned. Extensive myonecrosis with subsequent compartment syndrome, rhabdomyolysis, and acute renal failure followed a sting from an Arizona *S heros*. Japanese *Scolopendra* species have been reported to cause psychological disturbances and Korsakoff syndrome.¹³ The recurrence of swelling associated with pruritus at the sting sites three weeks following multiple stings in victim S.S. (our cases three and four) may have been related to an immune complex deposition syndrome (type III hypersensitivity reaction), although the reaction was mild and required no specific therapy.

No fatality due to a centipede sting has ever been reported in the United States, although one fatality was reported following a sting by a large specimen of *S subspinipes* (the same species responsible for cases three and four in our series) to the head of a small Filipino child.¹⁴

For pain, many authors suggest infiltration with local anesthetics such as lidocaine, and there are reports of using intradermal biscochlorin alkaloid (thought to protect mitochondria), although there are no studies establishing its effectiveness.^{1,13} The apparent analgesic benefit of local heat application in two of our stings is of interest. It may be that some components of centipede venoms that are responsible for pain are heat labile. Boiling centipede venom was found to inactivate its lethality in moths.² Similar to many marine envenomation syndromes, application of local non-scalding heat (up to 45°C as tolerated) may be considered for the management of acute centipede stings. However, the lability or stability of centipede venom at this temperature has not been investigated to our knowledge. Further research is needed to explore this possibility. Otherwise, systemic analgesics are standard, and significant doses

of narcotics are often necessary to achieve relief. Antihistamines may also be used to alleviate symptoms (such as pruritus). Cleansing of the wound, tetanus prophylaxis, and routine care for any necrosis are indicated. Prophylactic antibiotics would not appear to be necessary.

Based on our experience and that described previously in the literature, the clinical course following most centipede stings, particularly of species found in the United States, appears benign, self-limited, and rarely associated with any serious sequelae. Factors such as patient age, comorbid conditions, anatomic site of envenomation, and size/species of centipede should be considered when evaluating a patient with a centipede envenomation. Despite the striking appearance of the offender and the significant pain associated with a sting, treatment for centipede envenomation is essentially pain control and routine wound care.



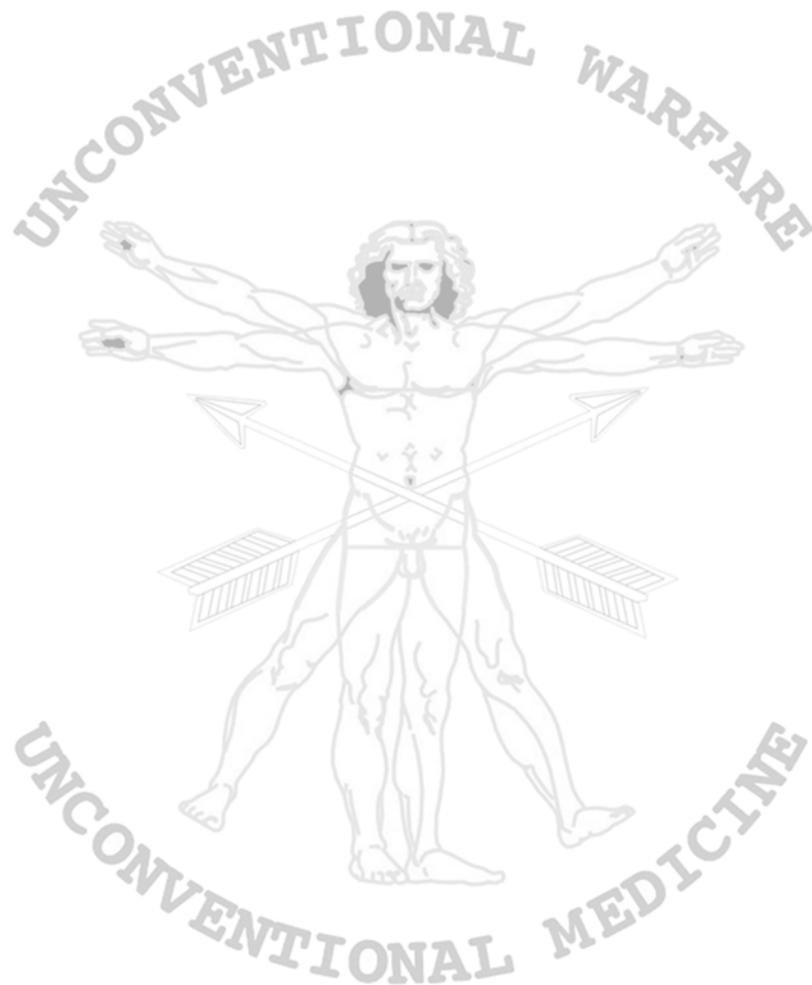
The centipede is distinguished by a segmented body, with each segment bearing a single pair of legs. The antennae and modified front pair of legs are also segmented. Centipedes are carnivorous, using the modified legs, strong mandibles, and a poison gland to capture and kill their prey.

Photo courtesy of Jerry Young

REFERENCES

1. Norris R.L. Jr., Centipede envenomations. In Adler J.N., Brenner B., Droner S., et al, eds. *eMedicine: Emergency Medicine*. Boston, MA: Boston eMedicine Corporation, 1999.
2. Menez A., Zimmerman K., Zimmerman S., Heatwole H., Venom apparatus toxicity of the centipede. *Ethostigmus rupides* (Chilopod, Scolopendridae). *Journal of Morphology*. 1990; 206:303-312.
3. Logan J.L., Ogden D.A. Rhabdomyolysis and acute renal failure following the bite of the giant desert centipede *Scolopendra heros*. *West Journal of Medicine*. 1985; 142:549-550.

4. Orange P. Incidents of predation on reptiles by invertebrates. *Herpetofauna*. 1989; 19:31-32.
5. Welsh J.H., Batty C.S. 5-Hydroxytryptamine content of some arthropod venoms and venom containing parts. *Toxicon*. 1963; 1:165.
6. Gomes A., Datta A., Sarangi B., Kar P.K., Lahiri S.C. Occurrence of histamine and histamine release by centipede venom. *Indian Journal of Medical Research*. 1982; 76:888.
7. Gomes A., Datta A., Sarangi B., Kar P.K., Lahiri S.C. Pharmacodynamics of venom of the centipede *Scolopendra subspinipes dehaani* Brandt. *Indian Journal of Experimental Biology*. 1982; 20:615-618.
8. Stankiewicz M., Hamon A., Benkhalifa R., et al. Effects of a centipede venom fraction on insect nervous system, a native *Xenopus* oocyte receptor and on an expressed *Drosophila* muscarinic receptor. *Toxicon*. 1999; 37:1431-1445.
9. Mohamed A.H., Abu-Sinna G., El-Shabaka H.A., El-Aal A.A. Effects of an extract from the centipede *Scolopendra morsitans* on intestine, uterus, and heart contractions and on blood glucose and liver and muscle glycogen levels. *Toxicon*. 1980; 18:581-589.
10. Mohamed A.H., Abu-Sinna G., El-Shabaka H.A., El-Aal A.A. Proteins, lipids, lipoproteins, and some enzyme characterizations of the venom extract from the centipede *Scolopendra morsitans*. *Toxicon*. 1983; 21:371-377.
11. Mumcuoglu K.Y., Leibovici V. Centipede (*Scolopendra*) bite: a case report. *Israel Journal of Medical Science*. 1989; 25:47-49.
12. Lin T.J., Yang C.C., Yang G.Y., Ger J., Tsai W.J., Deng J.F. Features of centipede bites in Taiwan. *Tropical Geographical Medicine*. 1995; 47:300-302.
13. Mohri S., Sugiyama A., Saito K., Nakajima H. Centipede bites in Japan. *Cutis*. 1991; 47:189-190.
14. Remington C.L. The bite and habits of a giant centipede (*Scolopendra subspinipes*) in the Philippine Islands. *American Journal of Tropical Medicine*. 1950; 30:453-455.





The Diagnosis and Treatment of Cough

Richard S. Irwin, M.D., and J. Mark Madison, MD

Abstract

Cough is one of the most common symptoms for which patients seek medical attention from primary care physicians and pulmonologists,¹ probably because cough can so profoundly and adversely affect the quality of patients' lives.² In this review, we present an approach to managing cough in adults. With a systematic approach based on the guidelines we describe, it should be possible to diagnose and treat cough successfully in the great majority of cases. The cause of chronic cough can be determined in 88 to 100 percent of cases, and determination leads to specific therapies with success rates that range from 84 to 98 percent.¹ Because of this high degree of success, there is only a limited role for nonspecific therapy for cough, which has been reviewed comprehensively elsewhere.¹

OBJECTIVES-

1. Identify the most common causes of acute, subacute, and chronic cough.
2. Identify the three most common causes of chronic cough in the normal setting, or near normal setting, chest radiograph.
3. Describe the evaluation and treatment of chronic cough due to post-nasal drip, asthma, and gastroesophageal reflux.

**Complete Test on Page 51--Answer sheet on Page 48
Completion of this article and test offers 1.0 and 1.2 CNE/CEH.**

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Disclosure:

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DURATION OF COUGH

Estimating the duration of cough is the first step in narrowing the list of possible diagnoses. There is controversy about how best to define chronic cough.¹ We propose that cough be divided into three categories: acute, defined as lasting less than three weeks; subacute, lasting three to eight weeks; and chronic, lasting more than eight weeks. Since all types of cough are acute at the outset, it is the duration of the cough at the time of presentation that determines the spectrum of likely causes.

ACUTE COUGH

For diagnosing the cause of acute cough, we recommend a clinical approach based on trials of empirical therapies. The physician should take a history and perform a physical examination while keeping in mind the estimated frequency of conditions. Although there have been no studies of the spectrum and frequency of causes of acute cough, clinical experience suggests that the most common causes are upper respiratory tract infections such as the common cold, acute bacterial sinusitis, pertussis in

TABLE 1 GUIDELINES FOR TREATING THE MOST COMMON CAUSES OF ACUTE COUGH IN ADULTS

CAUSE	THERAPEUTIC OPTIONS	COMMENTS
Common cold	Dexbrompheniramine, 6mg, plus pseudoephedrine, 120mg, twice daily for 1 wk, or naproxen, 500mg loading dose, then 500mg 3 times daily for 5 days, or ipratropium (0.06%) nasal spray, 2 42mg sprays per nostril 3 to 4 times daily as needed for 4 days	First-generation H ₁ antagonists may be helpful, but the relatively nonsedating H ₁ antagonists will most likely be ineffective. Ipratropium should be used in patients who cannot take or tolerate the other medicines.
Allergic rhinitis	Avoidance of offending allergens Loratadine, 10mg once a day	Other oral H ₁ antagonists, nasal cromolyn, corticosteroids, and azelastine may also be helpful.
Acute bacterial sinusitis	Dexbrompheniramine, 6mg, plus pseudoephedrine, 120mg, twice daily for 2 wk Oxymetazoline, 2 sprays twice daily for 5 days Antibiotic directed against <i>Haemophilus influenzae</i> and <i>Streptococcus pneumoniae</i>	Choice of antibiotic depends on multiple factors, including cost, allergies, and local patterns of bacterial resistance. Although the appropriate duration of therapy is not well defined, we treat for 2 weeks.
Exacerbation of chronic obstructive pulmonary disease	Antibiotic directed against <i>H. influenzae</i> and <i>S. pneumoniae</i> for 10 days Systemic corticosteroids tapered over 2-wk period Continuous oxygen if PaO ₂ ≤ 55 mm Hg or SaO ₂ ≤ 88%, or if PaO ₂ ≤ 59 mm Hg and there is evidence of erythrocythemia or cor pulmonale Ipratropium, 2 18mg puffs, plus albuterol, 2 90mg puffs, 4 times daily by metered-dose inhaler with spacer Cessation of smoking	Choice of antibiotic depends on multiple factors (see above). If treatment is started in the hospital, give equivalent of methylprednisolone, 125mg every 6 hr for 72 hr, then prednisone, 60mg/day for 4 days, 40mg/day for 4 days, and 20mg/day for 4 days. Oxygen is prescribed to increase PaO ₂ to 60-80 mm Hg at rest (SaO ₂ > 90%); an additional 1 liter/min is given during exercise and sleep. Need for continued oxygen is assessed after 1 month.
<i>Bordetella pertussis</i> infection	Erythromycin, 500mg 4 times daily for 14 days, or (if allergic) trimethoprim-sulfamethoxazole, 160mg 800mg twice daily for 14 days	Given their in vitro activity, other macrolides are also likely to be effective. These drugs and doses are appropriate for treatment and prophylaxis. Systemic corticosteroids have been beneficial in severely affected children

*Specific drugs and doses are mentioned when their use is supported by double-blind, randomized, placebo-controlled studies.
PaO₂ denotes partial pressure of arterial oxygen, and SaO₂ arterial oxygen saturation.

some communities, exacerbations of chronic obstructive pulmonary disease, allergic rhinitis, and rhinitis due to environmental irritants.¹

Viral infections of the upper respiratory tract are the most common causes of acute cough. In the absence of any treatment, the prevalence of cough due to the common cold ranges from 83 percent within the first 48 hours of the cold to 26 percent on day 14.³ Cough appears to arise from the stimulation of the cough reflex in the upper respiratory tract by postnasal drip, clearing of the throat, or both.³

The common cold is diagnosed when patients present with an acute respiratory illness characterized by symptoms and signs related primarily to the nasal passages (e.g., rhinorrhea,

sneezing, nasal obstruction, and postnasal drip), with or without fever, lacrimation, and irritation of the throat, and when a chest examination is normal. In such cases, diagnostic testing is not indicated, because it has a low yield. For instance, in immunocompetent patients with these symptoms and signs, more than 97 percent of chest radiographs will be normal.⁴

For treating acute cough due to the common cold, we recommend medications that have been shown in randomized, double-blind, placebo-controlled studies (Table 1) to be efficacious in decreasing cough. These include dexbrompheniramine plus pseudoephedrine³ and naproxen.⁵ Although the effect on cough was not specifically assessed in a study that showed that intranasal ipratropium provided relief of rhinorrhea and sneezing

due to the common cold,⁶ the drug may be helpful for patients who cannot take or tolerate the older-generation antihistamines or naproxen. There is no convincing evidence that intranasal or systemic corticosteroids are beneficial^{7,8} or that zinc lozenges are consistently beneficial,⁹⁻¹¹ and the relatively non-sedating histamine H₁ antagonists (e.g., loratadine), either alone or combined with a decongestant, are likely to be ineffective.^{1,12} These H₁ antagonists have failed to alleviate cough in patients with the common cold, probably because they have little or no anticholinergic activity and the common cold is not mediated by histamine. On the other hand, when cough is due to a histamine-mediated condition such as allergic rhinitis (Table 1), it is significantly improved by the non-sedating antihistamines.¹³ We do not recommend pharmacologic therapy as a substitute for the avoidance of offending allergens.

The common cold is a viral rhinosinusitis¹⁴ that often cannot be distinguished clinically from bacterial sinusitis.¹⁵ Because viral rhinosinusitis is the more common of the two, we recommend giving antibiotics to patients with findings that are suggestive of acute sinusitis only if their symptoms fail to show progressive improvement when treated with antihistamines and decongestants and if they have at least two of the following signs and symptoms: a maxillary toothache; purulent nasal secretions; abnormal findings on transillumination of any sinus; and a history of discolored nasal discharge (Table 1). It is usually not necessary to perform imaging studies of the sinuses in order to begin antibiotic therapy.¹⁶

It is not generally recognized that the common cold, like chronic postnasal-drip syndromes arising from a variety of rhinosinus conditions, can present as a syndrome of cough and phlegm.^{17,18} Consequently, physicians tend too frequently to diagnose such a syndrome as bacterial bronchitis and to prescribe antibiotics.¹⁹ We do not diagnose bronchitis in patients with a syndrome of cough and phlegm along with acute upper respiratory tract symptoms, and with few exceptions we do not initially prescribe antibiotic therapy in these instances. We do prescribe antibiotics for patients with an exacerbation of chronic obstructive pulmonary disease (Table 1) if the acute cough is accompanied by worsening shortness of breath, wheezing, or both.²⁰ We also prescribe antibiotics for patients with acute upper respiratory tract symptoms who have had

close contact with a patient with a known case of pertussis (Table I) and for patients with coughing and vomiting suggestive of *Bordetella pertussis* infection.¹ In the absence of chronic obstructive pulmonary disease, the failure to diagnose bronchitis when it is present will probably not adversely affect the patient, because most acute respiratory infections are viral.²¹

Acute cough can be the presenting manifestation of pneumonia, left ventricular failure, asthma, or conditions that predispose patients to the aspiration of foreign matter.^{1,22} It is especially important to have a high index of suspicion for these disorders in elderly patients, because classic signs and symptoms may be nonexistent or minimal.

SUBACUTE COUGH

For diagnosing the cause of subacute cough, we recommend a clinical approach based on trials of empirical therapies and limited laboratory testing. When cough is subacute and is not associated with an obvious respiratory infection, we evaluate patients in much the same way as those with chronic cough. For a cough that began with an upper respiratory tract infection and has lasted for three to eight weeks, the most common conditions to consider are post-infectious cough, bacterial sinusitis, and asthma.

Post-infectious cough is defined as cough that begins with an acute respiratory tract infection that is not complicated by pneumonia (i.e., the chest radiograph is normal) and that ultimately resolves without treatment.¹ It may result from postnasal drip or clearing of the throat due to rhinitis, tracheobronchitis, or both, with or without transient bronchial hyper-responsiveness. If the patient reports having a postnasal drip or frequently clears his or her throat or if mucus is seen in the oropharynx, we recommend an initial course of treatment similar to that for the common cold (Table 2). If the cough has not disappeared after one week of this therapy, we perform imaging studies of the sinuses to determine whether bacterial sinusitis is present. If these studies reveal a mucosal thickening of more than 5 mm, air-fluid levels, or opacification,²³ we prescribe a nasal decongestant for five days and an antibiotic for three weeks (Table 2), and then reassess the patient's condition.

When a patient presents with wheezes, rhonchi, or crackles on physical examination, a chest

TABLE 2. GUIDELINES FOR TREATING THE MOST COMMON CAUSES OF SUBACUTE COUGH IN ADULTS.*

CAUSE	THERAPEUTIC OPTIONS	COMMENTS
Postinfection	Dexbrompheniramine plus pseudoephedrine for 1 wk, or ipratropium (0.06%) nasal spray for 1 wk Ipratropium, 4 18mg puffs 4 times daily by metered-dose inhaler with spacer for 1-3 wk Systemic corticosteroids tapered over period of 2-3 wk Central antitussives	In case of postnasal drip and throat clearing, treat similarly to common cold (see Table I). If partial or no response to therapy for the common cold. If partial or no response to above therapy. Consider an initial dose of prednisone, 30 to 40 mg/day (or equivalent) for 3 days. For protracted, troublesome cough, consider dextromethorphan and codeine. If postinfectious cough is associated with <i>Bordetella pertussis</i> infection, add antibiotic; if associated with bronchial hyperresponsiveness, treat similarly to asthma for 6-8wk.
<i>B. pertussis</i> infection	Erythromycin for 14 days, or (if allergic) trimethoprim-sulfamethoxazole	Dose same as in Table I. Always consider cough associated with <i>B. pertussis</i> to be postinfectious and consider adding the above therapy if protracted- Violent coughing can provoke gastroesophageal reflux.
Subacute bacterial sinusitis	Dexbrompheniramine plus pseudoephedrine for 3 wk Oxymetazoline for 5 days Antibiotic directed against <i>Haemophilus influenzae</i> and <i>Streptococcus pneumoniae</i>	Initial treatment is similar to that for acute bacterial sinusitis (see Table 1), but we recommend a 3-wk course of an antihistamine-decongestant and an antibiotic.
Asthma	Beclomethasone, 4 42mg puffs twice daily by metered-dose inhaler with spacer or other equivalent Albuterol, 2 90mg puffs as needed, up to 4 times daily by metered-dose inhaler with spacer or other equivalent Albuterol, 2 90 mg puffs as needed, up to 4 times daily by metered-dose inhaler with spacer	Equivalent doses of different agents should yield similar results. Try different formulations if an inhaled agent provokes coughing. If all inhaled agents fail, give oral corticosteroids.

*Specific drugs and doses are mentioned when their use is supported by double-blind, randomized, placebo-controlled studies.

radiograph should be obtained. If it is normal, we prescribe inhaled bronchodilators and corticosteroids and consider antibiotics only if we suspect a recent *B. pertussis* infection. In such cases, improvement does not mean the diagnosis is asthma, because these drugs may have alleviated the cough by increasing mucociliary clearance and decreasing the production of mucus or by decreasing transient bronchial hyper-responsiveness after a viral infection. However, cough may be the sole presenting manifestation of asthma (as in so-called cough variant asthma). This diagnosis is suggested by the presence of bronchial hyper-responsiveness (e.g., a positive result on methacholine challenge) and is confirmed only when cough resolves during asthma therapy (Table 2) and follow-up proves the chronic nature of the disease.¹

If *B. pertussis* infections have recently been reported in the community, if there is a history of contact with a patient who has a known case, or if

the patient presents with the characteristic but infrequently heard whoop or with coughing and vomiting, empirical therapy for this infection should be considered (Tables 1 and 2).¹ The later in the illness antibiotics are prescribed, the less likely it is that they will be efficacious. The laboratory diagnosis of pertussis is difficult to establish because there is usually a delay between the onset of cough and the suspicion of the disease and because there is no readily available, reliable serologic test for *B. pertussis*.^{24,25} Cultures of nasopharyngeal secretions are usually negative after two weeks, and reliable, serologic confirmation of a recent *B. pertussis* infection requires evidence of an elevated level of antibodies against one of the various virulence factors of the organism, as revealed by an enzyme-linked immunosorbent assay.

CHRONIC COUGH

Although cough that lasts longer than eight

weeks can be caused by many different diseases,²⁶ most cases are attributable to one of only a few diagnosis. Consequently, we recommend a systematic evaluation that initially assesses the likelihood of the most common causes by means of trials of empirical therapy and trials involving the avoidance of irritants and drugs, along with focused laboratory testing (e.g., chest radiography or methacholine challenge), followed by additional testing and consultation with a specialist, if necessary. The definitive diagnosis of the cause of chronic cough is then established on the basis of an observation of which specific therapy eliminates the cough. Because chronic cough can result simultaneously from more than one condition (as is the case in 18 to 93 percent of instances),^{1,2} therapy that is partially successful should not be stopped but should instead be sequentially supplemented.

Multiple studies^{1,17,18,27-32} have shown that in approximately 95 percent of cases in immunocompetent patients, chronic cough results from postnasal-drip syndrome from conditions of the nose and sinuses, asthma, gastroesophageal reflux disease, chronic bronchitis due to cigarette smoking or other irritants, bronchiectasis, eosinophilic bronchitis, or the use of an angiotensin-converting-enzyme inhibitor. In the remaining 5 percent of cases, chronic cough results from a variety of other diseases, such as bronchogenic carcinoma, carcinomatosis, sarcoidosis, left ventricular failure, and aspiration due to pharyngeal dysfunction. In our experience, psychogenic, or "habit," coughs are rare conditions best diagnosed by exclusion.^{1,26} For example, a postnasal-drip syndrome with continual clearing of the throat can be misdiagnosed as a habit cough.¹

Diagnosis and Clinical Evaluation

Physicians can narrow the list of possible diagnoses by reviewing the patient's history and physical examination and focusing on the most common causes of chronic cough (i.e., postnasal-drip syndromes, asthma, and gastroesophageal reflux disease); obtaining a chest radiograph; and determining whether the symptoms conform to the clinical profile that is usually associated with a diagnosis of postnasal-drip syndrome, asthma, gastroesophageal reflux disease, or eosinophilic bronchitis, alone or in combination. If the cough is productive of blood, the patient should be evaluated according to published guidelines for hemoptysis.³³

If the patient has a history of smoking or of exposure to other environmental irritants or is currently being treated with an angiotensin-converting-enzyme inhibitor, the first step in the evaluation of cough becomes straightforward; elimination of the irritant or discontinuation of the drug for four weeks should be encouraged because it will reveal whether the cough is partially or entirely due to chronic bronchitis or to the angiotensin-converting-enzyme inhibitor. Cough due to these factors should substantially improve or resolve within this time (Table 3).¹ A comprehensive review of cough due to angiotensin-converting-enzyme inhibitors has been published elsewhere.¹ In the absence of exposure to irritants, a diagnosis of chronic bronchitis is untenable even if the cough is productive. The character of the cough (e.g., paroxysmal, loose and self-propagating, productive, or dry), the quality of the sound (e.g., barking, honking, or brassy), and the timing of the cough (e.g., at night or with meals) have not been shown to be diagnostically useful.¹⁸

Although a history of postnasal drip or clearing of the throat and physical findings of mucus, a cobblestone appearance to the mucosa of the oropharynx, or both suggest postnasal-drip syndrome, these symptoms and signs are not specific to this diagnosis³² nor do they always appear even when this syndrome is the cause of cough. A minority of patients may have no upper respiratory symptoms or signs yet may have a favorable response to combination therapy with a first-generation H₁ antagonist and a decongestant (these patients have "silent" postnasal-drip syndrome).³¹ Although frequent heartburn and regurgitation suggest that gastroesophageal reflux disease is the cause of cough, these symptoms may be absent in up to 75 percent of cases (i.e., in patients with "silent" gastroesophageal reflux disease).³⁴

Because cough can be the sole manifestation of asthma in up to 57 percent of cases³⁵ (i.e., with cough variant asthma or "silent" asthma) and because the clinical diagnosis of asthma is unreliable even when there is a history of wheezing and a current physical finding of wheezing,³⁶ it is inadvisable to diagnose asthma on clinical grounds alone. Although the presence of other abnormal sounds such as crackles and rhonchi suggests that testing for lower respiratory tract disease is indicated, these findings, with or without confirmatory laboratory-test results (e.g., chest radiography showing

chronic interstitial pneumonia), should not be relied on exclusively in the determination of the ultimate

cause of cough. A definitive diagnosis can be made only when cough responds to specific therapy.

TABLE 3. GUIDELINES FOR TREATING THE MOST COMMON CAUSES OF CHRONIC COUGH IN ADULTS*

CAUSE	THERAPEUTIC OPTIONS	COMMENTS
Postnasal-drip syndromes Nonallergic rhinitis	Dexbrompheniramine plus pseudoephedrine for 3 wk, or ipratropium (0.06%) nasal spray for 3 wk	Doses similar to those for common cold (see Table 1). Improvement should start within 2-7 days. Initial therapy with nasal corticosteroids or second-generation H ₁ antagonists will probably yield poorer results. After cough resolves, prescribe beclomethasone (or equivalent) nasal spray, 1 or 2 84mg puffs per nostril daily for 3 months. Flares may follow subsequent colds. See comments in Table 1.
Allergic rhinitis	Avoidance of offending allergens Loratadine, 10 mg once a day	
Vasomotor rhinitis	Ipratropium (0.06%) nasal spray for 3 wk and men as needed	Doses similar to those for common cold. If necessary, add dexbrompheniramine plus pseudoephedrine.
Chronic bacterial sinusitis	Dexbrompheniramine plus pseudoephedrine for 3 wk Oxymetazoline for 5 days Antibiotic directed against <i>Haemophilus influenzae</i> , <i>Streptococcus pneumoniae</i> , and anaerobes in the mouth	Initial treatment is similar to that for acute bacterial sinusitis (see Table 1) except for a 3-week course of an antihistamine-decongestant and an antibiotic. After cough resolves, prescribe nasal corticosteroids (see above) for 3 months.
Asthma	Beclomethasone by metered-dose inhaler with spacer Albuterol by metered-dose inhaler with spacer as needed	See comments in Table 2. Cough will start to improve within 1 week and may take 6-8 week to resolve. Long-term maintenance therapy with an antiinflammatory drug may be necessary.
Gastroesophageal reflux disease	Modifications of diet and lifestyle Acid suppression Prokinetic therapy	Initial medical therapy should be intensive (dietary changes, proton-pump inhibition, and a prokinetic agent such as metoclopramide). Long-term maintenance therapy will be necessary. If there is no improvement within 3 months, do not assume that reflux disease has been ruled out. Assess the adequacy or failure of therapy by means of 24-hour monitoring of esophageal pH while patient is receiving therapy. Treat coexisting conditions (see Table 4).
Chronic bronchitis	Elimination of irritant Ipratropium, 2 18mg puffs 4 times daily by metered-dose inhaler with spacer	Cough will improve or disappear in 94-100% of patients with cessation of smoking. In those who continue to smoke, ipratropium can be helpful. If cough temporarily worsens with cessation of smoking, ipratropium and corticosteroids may be helpful.
Angiotensin-converting-enzyme inhibitors	Discontinuation of drug	The cough is not dose-related; substitution of another drug in the same class will not help. With discontinuation, cough should improve or resolve within 4 week. If these drugs must be continued, oral sulindac, indomethacin, nifedipine, and inhaled cromolyn sodium may provide relief. Therapy with angiotensin II-receptor antagonists has not been complicated by cough. Equivalent doses of other inhaled corticosteroids are also effective. Systemic corticosteroids (prednisone, 30 mg/day for 2-3 week) are sometimes required. Long-term therapy may be necessary. If associated with an environmental irritant (e.g., acrylic resin), avoidance is advised.
Eosinophilic bronchitis	Inhaled budesonide, 400 mg twice daily for 14 days	

*Specific drugs and doses are mentioned when their use is supported by double-blind, randomized, placebo-controlled studies.

The diet should be low in fat (approximately 45 g of fat per day); patients should eliminate foods and beverages that relax lower esophageal sphincter tone or are acidic (coffee, tea, soft drinks, citrus fruit, tomato, alcohol, chocolate, mint); they should eat three meals per day and no snacks; and they should have nothing to eat or drink except for taking medications for two hours before reclining. Once cough resolves, restrictions can be relaxed but not eliminated. Lifestyle changes include cessation of smoking and wearing clothes that are not constricting. The head of the bed should be elevated for the minority of patients who have reflux in the supine position. The great majority of patients who cough because of gastroesophageal reflux disease have reflux while upright, not while supine.

Chest Radiography

The chest radiograph is useful for the initial ranking of possible diagnoses and for guiding trials of empirical therapies and laboratory testing.¹ A normal radiograph in an immunocompetent patient, or a radiograph that shows no abnormality other than one consistent with an old and unrelated process, makes postnasal-drip syndrome, asthma, gastroesophageal reflux disease, chronic bronchitis, and eosinophilic bronchitis likely and bronchogenic carcinoma, sarcoidosis, tuberculosis, and bronchiectasis unlikely. If the chest radiograph is abnormal, the physician should next evaluate the possibility of the diseases suggested by the radiographic findings.

The Most Common Cause

The clinical profile associated with postnasal-drip syndrome; asthma, gastroesophageal reflux disease, eosinophilic bronchitis, or some combination of these conditions is that of a non-smoking patient with a chronic cough who is not taking an angiotensin-converting-enzyme inhibitor and has a normal or near-normal and stable chest radiograph.

Because there is no diagnostic test for postnasal-drip syndrome and because it is the most common cause of chronic cough, the patient should be evaluated for this condition first. The outcome of specific therapy will depend on the determination of the correct cause and the choice of the correct specific therapy (Table 3).¹ The differential diagnosis of postnasal-drip syndrome includes sinusitis and the following types of rhinitis, alone or in combination: non-allergic, allergic, post-infectious, vasomotor, drug-induced, and environmental-irritant-induced.¹ If the specific therapy that is chosen fails, it does not necessarily mean that there is no postnasal-drip syndrome; cough may have failed to improve because the wrong antihistamine was given.¹ The newer-generation H₁ antagonists do not appear to be effective when cough induced by postnasal drip is not mediated by histamine.¹

Because a negative result of methacholine challenge rules out asthma as a cause of chronic cough (except soon after an exposure to toluene diisocyanate),¹ we recommend that the test be routinely performed. Although its positive predictive value ranges from 60 to 88 percent,^{17,32,34,37} its

negative predictive value is 100 percent.^{17,32,34,37} Cough variant asthma should be treated the same way as asthma in general.¹ If cough does not improve with asthma treatment (Table 3), the result of methacholine challenge can be considered to have been falsely positive. On the other hand, if methacholine challenge is not performed and cough disappears after the administration of systemic corticosteroids, it should not be assumed on the basis of this empirical trial alone that the patient has asthma, because other inflammatory conditions (e.g., eosinophilic bronchitis and allergic rhinitis) also respond well to corticosteroids.¹

We do not routinely recommend diagnostic testing for the evaluation of patients for "silent" gastroesophageal reflux disease, for the following reasons: although 24-hour monitoring of esophageal pH is the single most sensitive and specific test, it has a negative predictive value of less than 100 percent and a positive predictive value as low as 89 percent^{17,32,34}; 24-hour monitoring of esophageal pH is inconvenient for patients and not widely available; and there is no consensus about the best way to interpret the results obtained through such monitoring in the diagnosis of cough due to reflux disease.^{1,33,34} Even if the attempted therapies (changes in lifestyle, acid suppression, and the addition of prokinetic drugs) do not improve cough (Table 3), it must not be assumed that gastroesophageal reflux disease has been ruled out as the cause. The therapy may not be intensive enough or may not have been sustained long enough, or the disease may not respond to even the most intensive medical therapy; in some cases, anti-reflux surgery may be successful.^{1,38} The adequacy of the regimen of medical treatment and the need for anti-reflux surgery can be assessed by means of esophageal pH monitoring while medical therapy continues.^{1,38}

Eosinophilic bronchitis is a cause of chronic cough in up to 13 percent of cases.^{30-32,35} Although an analysis of the sputum generally shows eosinophils and metachromatic cells similar to those seen in asthma, this condition is distinct from asthma because it is not associated with bronchial hyperresponsiveness.³⁹ Eosinophilic bronchitis is responsive to inhaled and especially systemic corticosteroids (Table 3).³⁰ It can be ruled out if eosinophils make up less than 3 percent of the non-squamous cells in the induced-sputum sample as determined with the use of standard methods^{30,39} or

TABLE 4. COMMON PITFALLS IN MANAGING THE MOST COMMON CAUSES OF CHRONIC COUGH.

Postnasal-drip syndrome

Failing to recognize that it can present as a syndrome of cough and phlegm.¹⁷

Assuming that all H₁ antagonists are the same.¹

Failing to consider sinusitis because it is not obvious.

Failing to consider allergic rhinitis and failing to recommend the avoidance of allergens because symptoms are perennial.

Asthma

Failing to recognize that it can present as a syndrome of cough and phlegm.¹⁷

Failing to recognize that inhaled medications may exacerbate cough.¹

Assuming that a positive result of methacholine challenge alone is diagnostic of asthma.⁴⁰

Gastroesophageal reflux disease

Failing to recognize that it can present as a syndrome of cough and phlegm.¹⁷

Failing to recognize that "silent" reflux disease can be the cause of cough and that it may take 2-3 months of intensive medical therapy before cough starts to improve and, on average, 5-6 months before cough resolves.¹

Assuming that cough cannot be due to gastroesophageal reflux disease because cough remains unchanged when gastrointestinal symptoms improve. Failing to recognize that cough may fail to improve with the most intensive medical therapy and that the adequacy of therapy and the need for surgery can be assessed by means of 24-hour monitoring of esophageal pH.¹

Failing to recognize the effects of coexisting diseases (e.g., obstructive sleep apnea or coronary artery disease) or their treatment (e.g., nitrates)^{41,42}

Failing to treat adequately coexisting causes of cough that perpetuate the cycle of cough and reflux.^{1,38}

Postnasal drip, asthma, and gastroesophageal reflux disease

Failing to consider that more than one of these conditions may be contributing simultaneously to cough.³⁸

Failing to consider these common conditions because of another "obvious" cause (e.g., chronic interstitial pneumonia).¹

if cough fails to improve with empirical corticosteroid therapy.

PERSISTENTLY TROUBLESOME CHRONIC COUGH

Because postnasal-drip syndrome, asthma, and gastroesophageal reflux disease are the most common causes of chronic cough, the first step in managing a persistently troublesome chronic cough must be to consider the most common errors in management (Table 4). In our experience,³⁵ the failure to avoid these common pitfalls is often the

reason chronic cough remains troublesome. Once potential errors in management have been addressed, additional laboratory studies¹ (e.g., studies of sputum, modified barium esophagography, 24-hour monitoring of esophageal pH, esophagoscopy, a study of gastric emptying, high-resolution computed tomography of the chest, bronchoscopy,⁴³ or noninvasive cardiac studies) and referral to a cough specialist are indicated to assess the possibilities of intrathoracic processes (e.g., bronchiectasis,⁴⁴ bronchiolitis,⁴⁴ and left ventricular failure³⁵) that were not suggested by the chest radiograph.



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REFERENCES

1. Irwin RS, Boulet L-P, Cloutier MM, et al. Managing cough as a defense mechanism and as a symptom: a consensus panel report of the American College of Chest Physicians. *Chest* 1998;114:Suppl:133S-181S.
2. French CL, Irwin RS, Curley FJ, Krikorian CJ. The impact of chronic cough on quality of life. *Archives of Internal Medicine* 1998;158:1657-61.
3. Curley FJ, Irwin RS, Pratter MR, et al. Cough and the common cold. *American Review of Respiratory Disease* 1988;138:305-11.
4. Diehr P, Wood RW, Bushyhead JB, Krueger L, Wolcott B, Tompkins RK. Prediction of pneumonia in outpatients with acute cough - a statistical approach. *Journal of Chronic Disease* 1984;37:215-25.
5. Sperber SJ, Hendley JO, Hayden FG, Riker DK, Sorrentino JV, Gwaltney JM Jr. Effects of naproxen on experimental rhinovirus colds: a randomized, double-blind, controlled trial. *Annals of Internal Medicine* 1992; 117:37-41.
6. Hayden FG, Diamond L, Wood PB, Korts DC, Wecker MT. Effectiveness and safety of intranasal ipratropium bromide in common colds: a randomized, double-blind, placebo-controlled trial. *Annals of Internal Medicine* 1996; 125: 89-97.
7. Gustafson LM, Proud D, Hendley JO, Hayden FG, Gwaltney JM Jr. Oral prednisone therapy in experimental rhinovirus infections. *Journal of Allergy and Clinical Immunology* 1996; 97 1009-14.
8. Puhakka T, Makela MJ, Malmstrom K, et al. The common cold: effects of intranasal fluticasone propionate treatment. *Journal of Allergy and Clinical Immunology* 1998;101-726-31.
9. Jackson JL, Peterson C, Lesho E. A meta-analysis of zinc salts lozenges and the common cold. *Archives of Internal Medicine* 1997;157:2373-6.
10. Macknin ML, Piedmonte M, Calendine C, Janosky J, Wald E. Zinc gluconate lozenges for treating the common cold in children: a randomized controlled trial. *Journal of the American Medical Association* 1998; 279:1962-7.
11. Prasad AS, Fitzgerald IT, Bao B, Beck FW, Chandrasekar PH. Duration of symptoms and plasma cytokine levels in patients with the common cold treated with zinc acetate. *Annals of Internal Medicine* 2000;133:245-52.
12. Irwin RS, Curley FJ, Bennett FM. Appropriate use of anti-tussives and protussives: a practical review. *Drugs* 1993; 46:80-91.
13. Ciprandi G, Buscaglia S, Catrullu A, Marchesi E, Bianchi B, Canonica GW. Loratadine in the treatment of cough associated with allergic rhinoconjunctivitis. *Annals of Allergy and Asthma Immunology* 1995; 75:115-20.
14. Gwaltney JM Jr., Phillips CD, Miller RD, Riker DK. Computed tomographic study of the common cold. *New England Journal of Medicine* 1994; 330:25-30.
15. van Buchem FL, Knotmerus JA, Schrijnemaekers VJJ, Peeters ME. Primary-care-based randomized placebo-controlled trial of antibiotic treatment in acute maxillary sinusitis. *Lancet* 1997; 349:683-7.
16. Williams JW Jr., Simel DL, Roberts L, Samsa GP. Clinical evaluation for sinusitis: making the diagnosis by history and physical examination. *Annals of Internal Medicine* 1992; 117:705-10.
17. Smyrnios NA, Irwin RS, Curley FJ. Chronic cough with a history of excessive sputum production: the spectrum and frequency of causes, key components of the diagnostic evaluation, and outcome of specific therapy. *Chest* 1995; 108:991-7.
18. Mello CJ, Irwin RS, Curley FJ. Predictive values of the character, timing, and complications of chronic cough in diagnosing its cause. *Archives of Internal Medicine* 1996; 156:997-1003.
19. Gonzales R, Steiner JF, Sande MA. Antibiotic prescribing for adults with colds, upper respiratory tract infections, and bronchitis by ambulatory care physicians. *Journal of the American Medical Association* 1997; 278:901-4.
20. Madison JM, Irwin RS. Chronic obstructive pulmonary disease. *Lancet* 1998; 352:467-73.
21. Gonzales R, Steiner JF, Lum A, Barrett PH Jr. Decreasing antibiotic use in ambulatory practice: impact of a multidimensional intervention on the treatment of uncomplicated acute bronchitis in adults. *Journal of the American Medical Association* 1999; 281:1512-9.
22. Teramoto S, Matsuse T, Ouchi Y. Clinical significance of cough as a defense mechanism or a symptom in elderly patients with aspiration and diffuse aspiration bronchiolitis. *Chest* 1999; 115:602-3.
23. Hamory BH, Sande MA, Sydnor A Jr., Seale DL, Gwaltney JM Jr. Etiology and antimicrobial therapy of acute maxillary sinusitis. *Journal of Infectious Diseases* 1979; 139:197-202.
24. Wright SW, Edwards KM, Decker MD, Zeldin MH. Pertussis infection in adults with persistent cough. *Journal of the American Medical Association* 1995; 273:1044-6.
25. Yaari E, Yafe-Zimertnan Y, Schwartz SB, et al. Clinical manifestations of *Bordetella pertussis* infection in immunized children and young adults. *Chest* 1999; 115:1254-8.
26. Irwin RS. Cough. In: Irwin RS, Curley FJ, Grossman RF, eds. Diagnosis and treatment of symptoms of the respiratory tract. Armonk, N.Y.: Futura Publishing, 1997:1-54.

27. Palombini BC, Villanova AC, Araujo E, et al. A pathogenic triad in chronic cough: asthma, postnasal drip syndrome, and gastroesophageal reflux disease. *Chest* 1999; 116:279-84.
28. Marchesani F, Cekarini L, Pela R, Sanguinetti CM. Causes of chronic persistent cough in adult patients: the results of a systematic management protocol. *Monaldi Archives of Chest Disease* 1998; 53:510-4.
29. Hoffstein V. Persistent cough in nonsmokers. *Cancer and Respiratory Journal* 1994; 1:40-7.
30. Brightling CE, Ward R, Goh KL, Wardlaw AJ, Pavord ID. Eosinophilic bronchitis is an important cause of chronic cough. *American Journal of Respiratory Critical Care Medicine* 1999; 160:406-10.
31. Pratter MR, Bartter T, Akers S, DuBois J. An algorithmic approach to chronic cough. *Annals of Internal Medicine* 1993; 119:977-83.
32. Irwin RS, Curley FJ, French CL. Chronic cough: the spectrum and frequency of causes, key components of the diagnostic evaluation, and outcome of specific therapy. *American Review of Respiratory Disease* 1990; 141:640-7.
33. Baiter MS. Hemoptysis. In: Irwin RS, Curley FJ, Grossman RF, eds. *Diagnosis and treatment of symptoms of the respiratory tract*. Armonk, N.Y.: Futura Publishing, 1997:155-97.
34. Irwin RS, French CL, Curley FJ, Zawacki JK, Bennett FM. Chronic cough due to gastroesophageal reflux: clinical, diagnostic, and pathogenetic aspects. *Chest* 1993; 104:1511-7.
35. Irwin RS, Corrao WM, Pratter MR. Chronic persistent cough in the adult: the spectrum and frequency of causes and successful outcome of specific therapy. *American Review of Respiratory Disease* 1981; 123:413-7: I.
36. Pratter MR, Hingston DM, Irwin RS. Diagnosis of bronchial asthma by clinical evaluation: an unreliable method. *Chest* 1983; 84:42-7.
37. McGarvey LPA, Heaney LG, Lawson IT, et al. Evaluation and outcome of patients with chronic non-productive cough using a comprehensive diagnostic protocol. *Thorax* 1998; 53:738-43.
38. Irwin RS, Zawacki JK. Accurately diagnosing and successfully treating chronic cough due to gastroesophageal reflux disease can be difficult. *American Journal of Gastroenterology* 1999; 94:3095-8.
39. Gibson PG, Dolovich J, Denburg J, Ramsdale EH, Hargreave FE. Chronic cough: eosinophilic bronchitis without asthma. *Lancet* 1989; 1: 1346-8.
40. Irwin RS, French CL, Smyrniotis NA, Curley FJ. Interpretation of positive results of a methacholine inhalation challenge and 1 week of inhaled bronchodilator use in diagnosing and treating cough-variant asthma. *Archives of Internal Medicine* 1997; 157:1981-7.
41. Kerr P, Shoenuit JP, Millar T, Buckle P, Kryger MH. Nasal CPAP reduces gastroesophageal reflux in obstructive sleep apnea syndrome. *Chest* 1992; 101:1539-44.
42. Shoenuit JP, Kerr P, Micflikier AB, Yarnashiro Y., Kryger MH. The effect of nasal CPAP on nocturnal reflux in patients with aperistaltic esophagus. *Chest* 1994; 106:738-41.
43. Markowitz DH, Irwin RS. Is bronchoscopy overused in the evaluation of chronic cough? Bronchoscopy is overused. *Journal of Bronchology* 1997; 4:332-6.
44. Schaefer OP, Irwin RS. Chronic cough due to clinically "silent" suppurative disease of the airways: a new clinical entry. *American Journal of Respiratory Critical Care Medicine* 1999; 159: Suppl: A830. Abstract



Synopsis on Al Udairi Range Bombing Incident Initial Medical Response

Interviewed by COL S
Prepared by Kevin Riley, MSC

Abstract

The following is a synopsis of an interview with a special forces Sergeant H that was conducted at Camp Doha on 25 Mar 01 by an Army COL. The medical response synopsis was prepared on 10 Apr 01 by then Maj Kevin Riley.

Editors Note: Due to operations security and to protect those involved and their families, specific rank, last names and units of attachment have been excluded. They will be referred to by initials only.

Sergeant H is an 18D (Special Forces Medical Sergeant). He was serving in Kuwait at the time of the incident. He has been in the Army for 6 years and was previously a 91B (conventional Combat Medic). He volunteered and entered SF training about 3 years prior and attended the 46-week 18D Special Forces Medic course at the Joint Special Operations Medical Training Center (JSOMTC). He states that he felt the JSOMTC and the medic course did a great job of training him such that he felt confident of his medical skills after completing training. He had reported to his first SF assignment within the year.

ARRIVING AT THE SITE

At the time of the incident, Sergeant H. and two other NCOs, Sergeants M and W, were entering the Al Udairi range (Kuwait) in a military vehicle. It was in the evening, and he had the driver slow down because of visibility. Responsible for navigating the vehicle, Sergeant H used GPS to keep south of OP10 (impact area). The other two NCOs in the vehicle reported that they saw a couple of flashes and felt a slight concussion wave about 5kms away from the site. When they arrived at OP9 (just south of OP10), Sergeant M (in the vehicle) looked at his watch and noted it was 1912hrs. This would be 2 minutes after the impact. When they came over the hill to OP10, Sergeant H described the scene to look as if someone had started a bon-

fire. They had heard nothing on their radio and, given the limited visibility, there was no indication that anything looked wrong at the site.

At approximately 500 meters away from the source of the fire, Sergeant H's feelings changed and he ordered the vehicle to stop. He checked the GPS to make sure they were still out of the impact area. They proceeded further, again stopping. Sergeant H states "Something is not feeling right here." About 50 meters from the site, they realized it was a Suburban vehicle that was burning. Seeing shapes moving toward them, they proceeded to enter the site. Once there, two sergeants, Sergeant A and Sergeant S ran up to Sergeant H's vehicle and reported they had just been bombed. Everyone in Sergeant H's vehicle got out and proceeded to assess the situation.

IMMEDIATE FIRST AID AND TRIAGE

Sergeant H followed Sergeant S who took him to the first casualty.

Sergeant WG; a 29 year-old EOD specialist, was deceased. On initial assessment, Sergeant H noted the back half of his head was missing; he appeared charred and there were multiple shrapnel wounds in his chest. Sergeant H reported that he determined that his injuries were fatal and proceeded to the next person.

Special Forces Sergeant MS actually called out to Sergeant H by name (they served in the same company) and said his eye was "messed up". Sergeant H noticed that he had some damage to his eyelids and a shrapnel injury to his cheek. Sergeant H took off his own brown tee shirt and had Sergeant MS hold it against his own cheek as a pressure dressing.

Special Forces Sergeant SN was lying down and had an almost complete amputation of his left leg just below the knee. A tourniquet had already been applied prior to Sergeant H's arrival. While it appeared to Sergeant H that he may have lost much blood, Sergeant SN was conscious and quite calm.

Special Forces Sergeant WG was deceased. A fellow 18D and classmate of Sergeant H, Sergeant WG had severe multiple injuries to his abdomen and legs including an apparent fatal wound to his head.

The next scene that he came across were the two tactical air controllers.

Sergeant F, an AF tactical air controller, was deceased. When Sergeant H arrived, he noticed that the other AF tactical air controller was attempting to give Sergeant F first aid, while manning the radio. Sergeant H noted that Sergeant F had extensive injuries to his arms and that the back half of his chest was blown out. Sergeant H allowed the sergeant to continue to assist Sergeant F although it appeared that there were fatal injuries. Sergeant F died at the scene.

Sergeant C When Sergeant H arrived, he not only saw that Sergeant C was giving first aid, but was also handling all the radio traffic. Sergeant C had managed to call in a MEDEVAC, and despite his own injuries, remained focused on his task and the situation around him. Although he had some shrapnel wounds and a open fracture of both bones in his left lower leg, he refused treatment and asked Sergeant H to treat the others first. Sergeant H left one of the least injured NCOs with Sergeant C and asked him to make sure that he was not bleeding from any other injuries. When Sergeant F died, Sergeant C asked Sergeant H if he should start rescue breathing. Sergeant C remained on the radio until he was evacuated.

Maj MT, a New Zealand Army officer, was deceased. Sergeant H felt he had suffered internal

blast injuries and possible cervical spine injuries. Maj MT was spitting frothy blood from his mouth when he breathed. Sergeant H cleared his airway and put him in a better position to help with his breathing. Despite all the efforts to keep Maj MT alive, his injuries were too extensive and he soon expired

Sergeant B, an EOD specialist, was found by Sergeant H lying in a pool of tissue matter and vomit, his leg was severely mangled, and had deep labored respirations but no apparent bleeding. Sergeant H had Sergeant W stay with Sergeant B to clear his airway and monitor his status.

Sergeant FH, EOD specialist, was deceased. Sergeant H noticed that part of his chest cavity had been ripped out and assessed his injuries as fatal.

After his initial triage, Sergeant H went back and forth among the wounded to check on breathing and bleeding.

Special Forces Sergeants S and A, although in the same impact area and standing next to individuals who were killed, they both suffered very minor injuries. Sergeant A, a weapons NCO, completed only part of the 18D program, and was able to use the training he did receive to provide some of the initial care and life saving treatment moments after the impact. He used a radio antenna as a tourniquet device to put on Sergeant SN's leg.

EVACUATION

After his complete scene assessment, Sergeant H prioritized the first 4 or 5 guys for evacuation.

Evacuation helicopters arrived about 20-25 minutes after the incident. The fire and lights at the site had washed out pilots' night vision, forcing them to land about 500 meters away from the site.

Sergeant H requested an airway kit from the flight medic on the aircraft; since the medic did not have an airway or intubation kit, he inserted a J tube into Sergeant B's oropharynx. He also directed Sergeant A to complete the amputation on Sergeant S's leg.

The priority for evacuation was Sergeant SN first, Sergeant B second, and if room was available Sergeant MS and Sergeant C should be evacuated. After confirming that the remaining casualties had indeed died, Sergeant H accompanied his casualties on the helicopter.

IN THE HELICOPTER

Sergeant H and the flight medic started an IV on Sergeant SN. While enroute, Sergeant B stopped breathing. Although they attempted to start an IV on Sergeant B, he had no pulse and his veins did not engorge for a successful cannulation. Sergeant B's pupils were also noted to be fixed and dilated. The flight medic continued to assist his breathing with an Ambu bag, but Sergeant B was pronounced dead on arrival.

AT THE KUWAITI HOSPITAL

In the Emergency Room, the Kuwaitis assigned a doctor to each of the wounded. While in the ER, Sergeant H noticed that Sergeant C was still armed, prompting him to clear Sergeant C's weapon and check everyone else for weapons. Sergeant H remained with Sergeant SN, Sergeant MS and Sergeant C through their treatment in the Emergency Room.

Editors Note: Some points which should be extracted from this story would be the following:

1. ALL SOF personnel should receive self-aid/buddy aid training. As noted in the narrative, even though Sergeant H arrived within 5 minutes of the bombing, ALL the wounded had already received initial treatment.
2. Sergeant H attests that the rigorous SOF medic training and EMT-Paramedic training was absolutely on-target. This validates the present JSOMTC curriculum. The reason this is important is that the SOF Medic training continually comes under fire from numerous quarters. For example: Senior (older) medics criticize the new SOF medic training for being TOO trauma intense; line commanders criticize the training pipeline for being too long; unit commanders criticize the extent of sustainment medical training (which goes along with being a Paramedic) because it pulls the medics from other unit duties. I don't think anyone on the ground during the Kuwait incident would have wanted any shortcuts in medical training.
3. Even Sergeant A, who had failed out of the 18D program, claimed that his medical training was critical to his ability to help with the injuries.
4. The ingenuity and field expediency demonstrated by the SOF guys, and by Sergeant H, was incredible. A testament to their ability to think, solve problems, and react. This is a credit to the selection process for SOF, as well as their training and maturity beyond their age.
5. ALL the SOF personnel (including Sergeant C, even tho' not technically SOF) acted extremely composed in the face of unimaginable violence and chaos. This can only be a testament to their selection for maturity and strength of character, their devotion to duty, and their overall military training, regardless of MOS/AFSC. It all added up to confidence when the world was going to hell.

Steven J. Yevich, COL, MC, (Ret)
USSOCOM Command Surgeon (1998 -2001)

UNCONVENTIONAL WARFARE



UNCONVENTIONAL MEDICINE

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POST-TEST – Answer Sheet

Article 1 Dive Medical Brief -Part 2 Page 18

Please circle the letter that corresponds to the correct answer:

1. a b c d

5. a b c d

9. a b c d e

2. a b c d

6. a b c d e

10. T F

3. a b c d

7. a b c d e

4. a b c d e

8. T F

Article 2 Treatment and Diagnosis of Cough Page 31

Please circle the letter that corresponds to the correct answer:

- | | |
|--------------|--------------|
| 1. T F | 6. a b c |
| 2. a b c d e | 7. T F |
| 3. T F | 8. T F |
| 4. a b c | 9. a b c d e |
| 5. T F | 10. T F |

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CONTINUING MEDICAL EDUCATION TEST

NO.1

Dive Medical Brief: Part 2

A Comprehensive Review for The Special Forces Dive Medical Technician

JSOM



1. What is the difference between Type I and Type II Decompression Sickness (DCS), how they are likely to present, and the implications for treatment.
 - a. Type I bubbles form in the extremities; Type II bubbles form in the central nervous system, lungs, and inner ear.
 - b. Type I may manifest as skin itching, shoulder pain, marbling of the skin; Type II may manifest as bladder paralysis with urinary retention, blurred vision, substernal pain, cough, vertigo
 - c. Implications for treatment: Type I: not all must go to a decompression chamber, only those with pain in a joint; Type II: all must go to a decompression chamber.
 - d. All of the above
2. Which of the following correctly explains the pathophysiology of pulmonary oxygen toxicity?
 - a. The proliferative phase of pulmonary oxygen toxicity is characterized by alveolar edema, hemorrhage, and hyaline membrane formations.
 - b. Pulmonary oxygen toxicity causes an obstructive type of lung disease leading to pulmonary fibrosis.
 - c. Pulmonary oxygen toxicity corresponds with the Paul Bert Effect, which states that oxygen partial pressures with long-term exposure will cause lung damage.
 - d. Pulmonary oxygen toxicity occurs in two phases: the exudative phase and proliferative phase and may lead to a clinical picture of ARDS.
3. When a diver experiences pain in his ear while diving, he is close to the pressure differential at which he will be unable to clear. What is the most appropriate step for the diver to take at this time?
 - a. Continue his descent slowly
 - b. Perform a forceful Valsalva in an attempt to clear his ears
 - c. Use in-water decongestants like pseudophed
 - d. Stop the descent and ascend slowly to a depth of relief
4. Which of the following gas laws explains the pathophysiology of decompression sickness?
 - a. Gay-Lussac's Law
 - b. Dalton's Law
 - c. Charles' Law
 - d. Henry's Law
 - e. General Gas Law
5. What does the acronym VENTIDC stand for and with what diving injury is it associated?
 - a. Acronym: V - vision, E - ears tinnitus, N - nausea, T - tingling and muscle twitching, I - irritability, D - dizziness, C - convulsions
 - b. Side effects are associated with central nervous system (CNS) toxicity.
 - c. a & b
 - d. None of the above
6. Which of the following statements are true?
 - 1) Tissue metabolism removes nitrogen, but oxygen and carbon dioxide remain.
 - 2) Exercise decreases gas uptake and elimination by increasing the respiratory rate, cardiac output, and tissue perfusion.
 - 3) Gasses become rapidly dissolved in fat but are dissolved very slowly in muscle.
 - 4) Fat can take on vast amounts of nitrogen because nitrogen is five times more soluble in water than it is in fat.

- a. 1) and 2)
- b. 2) and 4)
- c. 1), 2) and 3)
- d. All of the above
- e. None of the above

7. The lungs are very good filters of bubbles; however, they may be overwhelmed by massive amounts of bubbles leading to pulmonary DCS (chokes). To what medical clinical picture is the similar?

- a. Pneumonia
- b. Bronchiectasis
- c. Pulmonary edema
- d. Pulmonary embolism
- e. Emphysema

8. The symptoms of Type II DCS involving the central nervous system and the symptoms related to an arterial gas embolism can be differentiated between based on the onset of symptoms after the dive. AGE symptoms manifest within 10 minutes after surfacing whereas symptoms of decompression sickness usually take one to four hours to develop.

T or F

9. Of all the diving injuries which one is considered the most potentially fatal?

- a. Pulmonary oxygen toxicity
- b. Type II DCS
- c. Caustic cocktail
- d. TM rupture
- e. AGE

10. A complication of closed circuit diving is known as a caustic cocktail: A condition that produces a chemical injury when it comes in contact with the mucosa of the oropharynx resulting in significant edema in the posterior oropharynx and laryngeal region producing stenosis of the airway leading to partial or even complete airway obstruction

T or F

CONTINUING MEDICAL EDUCATION TEST

NO.2

The Diagnosis and Treatment of Cough

JSOM



1. The most common cause of a chronic cough is postnasal drip. T or F
2. The following should be considered in evaluating a chronic cough:
 - a. Asthma
 - b. GERD
 - c. Medication
 - d. a & b only
 - e. a, b, c
3. Estimating the cough duration to help guide the diagnosis is an important first step. This paper divides a cough into Acute (< 3 wks), Subacute (3-8 wks), Chronic (> 8 wks). T or F
4. Upper respiratory infections such as the common cold is one of the etiologies of an acute cough. The recommended guideline for treating this cause of an acute cough includes:
 - a. Using a antihistamine/decongestant combination drug, NSAID or a anticholinergic nasal inhaler.
 - b. Using a antihistamine/decongestant combination drug, sympathomimetic nasal inhaler and antibiotic.
 - c. Azithromycin (Z-pack)
5. In evaluating a subacute cough that began with an upper respiratory tract infection, the most common conditions to consider are post infectious cough, bacterial sinusitis and asthma. T or F
6. If radiological studies of a patient's sinuses reveal a mucosal thickening of more than 5 mm air-fluid levels or opacification the recommendation for treatment includes:
 - a. Azithromycin (Z-Pack) and decongestant
 - b. The appropriate antibiotic x 3 wks, decongestant/antihistamine combination drug and nasal sympathomimetic.
 - c. Azithromycin (Z-pack) then repeat in 3 wks.
7. Previous studies have shown etiologies for chronic cough such as bronchogenic carcinoma, carcinomatosis, sarcoidosis, left ventricular failure and aspiration due to pharyngeal dysfunction are 7% of the overall total. T or F
8. Noting the character of cough, the quality of sound and/or the pattern of a cough are useful diagnostic tools. T or F
9. Common pitfalls in the management of a chronic cough include:
 - a. Failing to consider sinusitis because it's not obvious.
 - b. Failing to recognize that inhaled medications may exacerbate cough.
 - c. Failing to consider that more than one condition may contribute simultaneously to cough.
 - d. a & c
 - e. a, b & c
10. Treating a chronic cough with attempted therapies such as changes in life style, acid suppression, and the addition of prokinetic drugs which does not improve the cough, you can rule out GERD as a cause. T or F

CASE STUDY

GET TIGHT OR GET TWEAKED: A Case Report of a Both Bone Forearm Fracture Occurring during Parachute Opening.

Timothy S. Talbot, MD; Creighton C. Tubb,
MD; Mark T. Raymor, 18D

INTRODUCTION

Medical history is replete with reports of fractures associated with military static line parachuting. Jump related injuries occur in 8.1 per 1000 aircraft exits¹ with injuries at altitude occurring with an incidence of only 0.46 per 1000 aircraft exits.² Epidemiological reports have shown the majority of the injuries occur to the lower extremity, followed by closed head injury, and back injury.¹ The majority of these injuries occur during the landing phase with the performance of a parachute-landing fall (PLF). Injuries to the upper extremity are rare in comparison and typically occur from static line entanglement immediately before exit or result from improper PLF with the upper extremity striking the ground resulting in humeral or shoulder injuries. Injuries to the forearm are even more rare; those few reported have occurred during the PLF phase of the jump, and none have ever been reported to occur during the deceleration phase of the parachute opening.^{2,3,4,5,6,7} We present a unique case of a mid-shaft comminuted radial-ulnar fracture occurring during the parachute-opening phase of a tailgate static line jump.

CASE REPORT

The parachutist is a 29 year old active duty right hand dominant male assigned to United States Army Special Operations Command. The jumper was an experienced basic parachutist with >30 jumps. The soldier was assigned as the 5th jumper in the second stick of a routine quarterly airborne operation. The jump was a daylight, non-combat equipment; airborne proficiency operation utilizing a C-130 aircraft configured for ramp exit at 1250 feet AGL (above ground level) and 130 kts (knots). The parachutist was rigged with the MC1-1C para-

chute and T-10 reserve parachute. The soldier's static line was attached to the starboard cable and was being controlled with the left hand using a reverse bite. The jumper proceeded to the tailgate in the normal fashion allowing the excess static line to fall from his grip naturally while maintaining his right hand and arm over the edge of his reserve parachute. After dropping his static line, he attempted to bring his left arm up to cover the left side of his reserve and assume a tight body position upon exiting the tail of the aircraft. Instead of attaining a grip on his reserve, his left hand slipped behind his reserve chute and posterior to his main lift web adjacent to the D ring trapping his forearm. During the parachute deployment and opening shock, the jumper did not note any pain or audible snap. The jumper noted his injury while attempting to complete his second point of performance; he was unable to lift his left arm up to the left toggle. The jumper continued with his actions in the air despite the pain and executed a right-sided PLF. He immediately called for aid and was noted to have a gross





deformity of the mid left forearm with intact sensation, pulse and limited motor strength. He was placed in a splint and sling, and immediately transported to the local medical center for evaluation and treatment. Radiological evaluation was notable for a mid shaft comminuted both bone forearm fracture with apex radial angulation and volar displacement of the distal fragments.

No other injuries were noted during the comprehensive evaluation. The jumper's fractures were reduced, splinted, and he was taken to the OR for open reduction and internal fixation. The magni-



tude of the force transmitted to the forearm at the time of injury was evidenced by the severe degree of comminution found intra-operatively. After acceptable alignment was obtained through a two-incision technique, the radius and ulna were stabilized with stainless steel plates bridging the fractures and secured with screws.

The jumper sustained a good outcome to date and is continuing physical and occupational therapy. He is expected to return to full duty without restrictions.

DISCUSSION

Military static line parachuting places unique and dynamic forces on the body of the jumper, subjecting them to injuries not commonly seen in the normal soldier. This led many of the early medical pioneers of the airborne community to call for stricter physical induction standards, psychological assessment, and modification of training regimens to produce the finest airborne soldier. Neel provided the first published epidemiological description of the occupational unique injuries to the airborne soldier.³ Modification of induction standards, training methods, and equipment has occurred throughout the years in an attempt to lower the injury rates occurring during performance of an airborne operation.

Injuries during airborne operations can best be grouped according to those that occur during exit, descent, and landing. The majority of injuries occur during the landing phase of the parachute operation. Early analysis of injury patterns led to the conversion from the forward landing roll (open stance conducting a forward roll over outstretched arms) in the 1940s to the modern day PLF⁷. The addition of a mesh canopy skirt and modification of the canopy design in the 1950s were made to decrease oscillation.³ The addition of the helmet shock pad in the 1980s, and the ankle brace in the 1990s⁸ substantially decreased head injury and lower extremity injury rates.

The second largest group of injuries occurs during the exit phase. This includes injuries to the upper extremity from static line entanglement, injuries to the lower extremity from suspension line entanglement, and the rare injury from striking the body of the aircraft. Injury rates in this group have declined with the adoption in 1994 of a new exit body position, significantly reducing static line entanglement injuries.⁷ Emphasis on a tight body

position and the requirement for pre-jump training to reinforce the five points of performance has significantly reduced the number of suspension line entanglements as well.

Injuries occurring during the descent phase are rare with only cursory mention in epidemiological surveys. These predominantly occur due to collision between jumpers resulting in injury upon landing. Altitude injury is classified as an injury occurring from the moment of exit until impact with the ground. Recent review by Craig and Lee show that historically this has not been a significant contributor to parachute injury rates with an incidence of 0.46 injuries for every 1000 aircraft exits. Fifty-four percent of the injuries occurred during exiting before complete parachute deployment and 46% occurred during the opening shock of the parachute. Injuries occurring during the opening shock of the parachute were induced by riser/suspension line entanglement (46%), strikes from unsecured equipment (1%), or opening shock deceleration (1%) resulting in an abrasion/laceration.²

The parachute harness in use by the U. S. military is designed to distribute the opening shock of the parachute deployment throughout the torso. The opening shock of parachute deployment has been reported to be as high as 4 Gs (four times the parachutist's weight on earth).³ Every jumper is required to undergo a Jumpmaster Personnel Inspection (JMPI) prior to each airborne operation in which the integrity and wear of the equipment is checked. Any deficiencies are corrected at the time of the inspection prior to boarding the aircraft for the airborne operation. Loosening of the web harness may occur in the transition from the personnel staging area to the jump platform and prior to exiting the aircraft. It is incumbent on the parachutist to report any deficiencies to the primary jumpmaster or safety personnel prior to exiting the aircraft.

Any deficiency in the wear of the parachute harness will result in unequal distribution of forces in the harness. As well, it may allow for vertical translation of the harness with gaps between the body and main lift web resulting in injury to the parachutist. This is the case with the parachutist mentioned above. Due to a gap between the main lift web and his body he inadvertently placed his left arm into this space. He

was unable to remove it in time to avoid the opening shock and vertical translation of the harness. This resulted in the high-energy fracture pattern of the radius and ulna noted in the x-ray with sparing of the hand, wrist and elbow. The harness should be worn tight to the body to alleviate any potential space as well as distribute the significant force of the opening shock. Correct wear of the equipment will allow the harness to fulfill its designed role in distributing these dynamic forces and protect the parachutist from injuring during the altitude phase of an airborne operation.

REFERENCES

1. Craig SC, Morgan J: Parachuting injury surveillance, Fort Bragg, North Carolina, May 1993 to December 1994. *Military Medicine* 1997; 162: 162-4.
2. Craig SC, Lee T: Attention to Detail: Injuries at Altitude among U.S. Army Military Static Line Parachutists. *Military Medicine* 2000; 165: 268-71.
3. Neel SH: Medical Aspects of Military Parachuting. *Military Surgery* 1951; 108: 91-105.
4. Kragh JF Jr, Amarosa PJ, Jones BH, Heekin RD: Parachuting Injuries among Army Rangers: A Prospective Survey of an Elite Airborne Battalion. *Military Medicine* 1996; 161: 416-9.
5. Bar-Dayan Y, Bar-Dayan Y, Shemer J: Parachuting injuries: a retrospective study of 43,542 military jumps. *Military Medicine* 1998; 163: 1-2.
6. Glorioso JE Jr, Batts KB, Ward WS: Military Free Fall Training Injuries. *Military Medicine* 1999; 164: 526-30.
7. Bricknell MCM, Craig SC: Military parachuting injuries: a literature review. *Occupational Medicine* (Lond) 1999; 49: 17-26.
8. Schumacher JT Jr, Creedon JF, Pope RW: The Effectiveness of the Parachutist Ankle Brace in Reducing Ankle Injuries in an Airborne Ranger Battalion. *Military Medicine* 2000; 165: 944- 48.

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THANKS FOR MY EVERYTHING

James L. (Jim) DeVoss, 1/Lt, USAF
(Ret.)



“Party flight suits” were worn to squadron mates’ end of tour celebrations. I didn’t make mine, but I’m told my mates were able to hold one in my honor—thanks to those who risked their lives and got me out!

For over 32 years I have been telling groups of one or more a story at every opportunity. On December 10, 2001, I had my most recent and greatest opportunity to tell the story. Appropriately, it was modified from the "civilian" version in which I focus more on training before, rather than on my life and accomplishments after, being shot down during the Vietnam War. The audience on that date was the most special group of people I have ever had the privilege to address. The group was comprised of individuals just like you--SOF medical professionals, those who serve that others may live. Like June 16, 1969, I will remember December 10, 2001, for the rest of my life.

THE SHOOTDOWN

From the first time I heard and saw an airplane soar overhead, I had the desire to fly. This desire grew as I grew up, while I attended middle school, high school, and, ultimately, college at the University of Michigan. My bachelor's degree was one of the requirements for entering the Air Force's Officer Training School (OTS). Following OTS, I entered the Undergraduate Pilot Training (UPT) Program.

The training I had was standard to UPT. In extreme summary, we were taught that whenever we got into a cockpit and situated ourselves--setting all the fittings, harness, lapbelt, oxygen connection, anti-G suit connection, etc.--we were never, ever "strapping in" to the aircraft. Rather, we were strapping the aircraft on. When an aircraft is strapped on,

it is part of you, always doing what you want it to (provided, of course, its systems allow). If you are ever strapped into an aircraft, there is always the likelihood that, one day, one time, it could do what it wanted--with you merely as a passenger.

We always realized an aircraft and any of its systems could break--at any time. Thus, a critical and extensive part of our training was recognizing emergencies and, even more importantly, recovering from them. Recovery ranged from (1) fixing the challenge and continuing the mission to (2) continuing the mission, if possible, with something broken to (3) terminating the mission and landing as soon as practical if the challenge precluded continuing a safe, mission-fulfilling flight. Ultimately, "recovery" could entail having to leave the aircraft as soon as we recognized the challenge was so severe as to preclude getting the aircraft on the ground, safely or otherwise. Recovery, in that instance, was utilizing the ejection seat to maximize the probability of escaping with minimal or no injury. After ejection training, we were trained to get on the ground safely and survive no matter where we went down, even if in enemy territory.

Near the end of my UPT program, we were given the opportunity to array the selection of aircraft for which pilots were needed. Naturally, we ordered our preferences from the aircraft we most wanted to fly to the aircraft we least wanted to fly. The F-105 Thunderchief, affectionately known as the "Thud"--and one of only two single-seat fighters available at the time--had always been my goal. It led my list. Fortunately, pilots were needed to fly it.

More fortunately, I had done well enough throughout UPT to get to fly it. That led to 120 more flight hours preparing for combat. Suffice it to say that throughout that training, too, recognizing and recovering from emergency situations was a continual emphasis, especially given our next assignment would be flying combat missions with an operational squadron.

Every second of all that recognition and recovery training was put into action on June 16,



F-105 Thunderchief- The Thud. "The biggest, badest, fastest fighter that's ever flown." (Jim's personal opinion)

1969. I was on my 71st mission, flying as Kingfish 1, leader of a flight of four Thuds. We were fragged to work with a Forward Air Controller (FAC) who would locate and have us put our ordnance onto suspected "truck parks" along the Ho Chi Minh Trail in Northern Laos. I had made only one pass on such a park when enemy guns came up. All of the rest of our ordnance was expended on the 37 and 57mm gun sites that had begun shooting at us.

While pulling off my last pass and calling for my wingmen to join on my aircraft to head for the post-strike tanker, things began to happen in the cockpit. Namely, my emergency panel lit up like a Christmas tree. I had taken sufficient hits to, among other things, knock out my aircraft's hydraulic systems--all three of them. Instead of the 3,000 PSI hydraulic pressure needed to move the Thud's flight control systems, my aircraft had zero. There were a number of other things going wrong, too, but the loss of hydraulic pressure was the most critical.

As the pressure went to zero, I initiated the slab-lock procedure by flipping a switch that caused a big vice grips-type device to grab and hold fixed the bar connecting the two sides of the elevator (the horizontal sections of the tail). Supposedly, this procedure would allow the aircraft to maintain straight and level flight--allowing me to egress the target/enemy territory--at an "equilibrium" airspeed of approximately 400 knots. Because the aircraft was going faster than 400 when I locked in the slab, it initially started climbing--trading airspeed for altitude while attempting to get to 400. When it reached 400, my Thud did start to level off. Naturally, it didn't get

level at exactly 400, a bit under actually, so it should have started a slight descent to again trade altitude for increasing airspeed.

What happened, however, was the aircraft attempted to maintain the altitude at which it had leveled off. Almost immediately I observed that I lacked the power to maintain altitude and airspeed. Given that my Thud was maintaining altitude, speed immediately bled off. Within seconds I had to be most concerned about the aircraft going

below stall speed and into a spin. To preclude this for as long as possible, I blew off all the external stores to lighten the load and reduce drag. When that happened, instead of slowing down less rapidly, the nose of the aircraft merely fell, and she started towards the ground, rapidly accelerating--with me strapped in.

Initially, my only thought was back to what we had been told in training: "If your butt is more than one-half inch out of the seat when you initiate the ejection system, the force of its explosion will drive the seat into your butt with sufficient force to compress your spine, causing at least paralysis and most probably death!" When the aircraft nosed over, a force of 3 negative Gs hit me. Compare that, if you will, to the first downward motion of a roller coaster. In that situation, riders experience about one-half of a positive G. At 3 negative Gs, I found myself pushed out of the seat, and I knew there were inches between my butt and it. And the aircraft was accelerating like a rocket towards the ground. Bottom line, by the time (1) I had mentally gone over all procedures confirming I had done them all and (2) the aircraft had stabilized into its dive (so that negative G forces were as low as they were going to get and I was as firmly in the seat as I was going to get), the aircraft had accelerated to at least 600 knots. The thoughts in my mind by then were two: (1) ejection at speeds in excess of 500 knots are always lethal and (2) if I don't get out of this alive, my wife is going to kill me!

A standard ejection from a Thud goes as follows: You grab one or both of the ejection handles located to the outside of each knee. Raising them causes the triggers directly under the handles

to spring down. Squeezing one or the other or both of the triggers first causes the canopy to unlock and be blown from the aircraft by the slipstream. Two-tenths of a second later, an explosive charge equivalent to five pounds of TNT goes off, starting the seat up the rails on which it is positioned to give it an upward trajectory. Once the seat has moved 8 inches, a lanyard is pulled that fires a rocket on the back of the seat. That rocket is capable of pushing 400 pounds (the pilot and the seat) 250 feet into the air. That allows clearance over the tail plus allows for a zero airspeed/zero altitude ground ejection should that ever be required. One second after the rocket fires, three things happen simultaneously. First, the lapbelt and shoulder harness are explosively blown apart. Second, take-up reels at the front of the seat and the top of the back of the seat spin in opposite directions causing a strap connecting the two to become taut, thus pitching the pilot out of the seat. Third, a small drogue parachute is blown out of the back of the seat. That drogue chute slows the seat, completing man/seat separation and preventing the seat from tumbling into the pilot. One second after those three things happen, another small drogue chute is blown from the pilot's parachute pack. This catches in the wind and immediately deploys the main parachute. From rocket fire to parachute deployment, the pilot is fully suspended in a canopy within 3.2 seconds.

The ejection system in my crippled Thud worked perfectly; less than one second after I pulled the triggers, the charge under the seat went off. Although I was in the seat as tight as I could be, my spine was still compressed, much like a diver's is if he attempts a dive into too shallow a body of water and his head hits the ground. Not too long ago, 32 years after my butt and the seat had their troublesome experience, I had x-rays done before a medical exam. Before I entered the exam room, the doctor greeted my wife, and his first question was, "How long has it been since your husband has been able to walk? When was his diving accident?" What they told us in class was true!

The real challenge, however, beyond the tenuous connection between the seat and I, was the estimated 600 knots of airspeed to which my Thud had accelerated during the time necessary for me to be able to get down onto the seat as best I could. As the seat and I started out of the cock-

pit, the 600-knot windblast initially hit my head and shattered my visor, blowing pieces of it back into my face, causing a number of cuts. The windblast then hit my oxygen mask. At speeds over 500 knots the deadly concern is the mask being blown off and the windblast entering the nose so rapidly it over-expands the lungs, causing the lungs to rupture and the pilot to bleed to death. In my case, however, my face must have been positioned directly into the wind because the mask was driven straight back. It was driven back hard enough, however, that even the soft, pliable rubber at the top of the mask blackened both of my eyes and, at the bottom, cut into my neck.

As the seat and I continued up, the windblast next hit my shoulders and drove them both backwards, causing them to separate. Then the wind caught my arms. In the Thud's seat, there were small devices designed to catch the pilot's elbows so that the forearms were tucked into the stomach area. My right arm did tuck, but the left was pushed outward and then flailed behind the seat. That flailing against the back of the seat shattered the upper arm bone, shattered the elbow, broke both bones of the forearm, and also several bones in the hand. When the windblast hit my abdominal area, it pushed my torso backwards. With my thighs on the seat, both of my hips were separated. As the seat and I fully exited the cockpit, the windblast finally hit the tops of my boots. The 14 G ejection force caused them both to point straight down and the windblast caused them to tuck under the seat. This caused both of my knees to separate. Surgeons later told me that the right knee was separated to one-eighth of an inch to being "chicken leg torn from chicken thigh"-everything in the knee was completely torn apart with only one nerve, one blood vessel, and the skin around it holding the lower leg on. I am sure this was an exaggeration, but obviously there was considerable damage. As for the left knee, it was in similar shape, but while everything else was ripped apart, the patellar tendon was only torn three-quarters through. Unfortunately, though, I must have done a really lousy PLF (parachute landing fall), as the bones in my lower leg were broken when I hit the ground. Finally, I landed in a patch of bamboo, impaling myself on some of the younger shoots.

So when I hit the ground, my shoulders, hips, and knees were separated; many bones in my body were shattered and broken; and my face and

neck were bleeding. I landed over 130 miles behind enemy lines, well north in Laos--south of the "fishes' mouth" and East of the Plaine des Jars. I was actually closer to Hanoi than to friendly territory. We had been told during the intelligence briefing before the flight that we were "doing such a good job of halting the flow of military supplies down the Ho Chi Minh Trail that, if we went down and they couldn't walk us to a POW camp, they would stick a knife in us rather than waste a bullet." In the briefing, that sounded good. On the ground and in my condition, it didn't.

Despite my broken limbs and bloody body, I felt relatively calm (adrenaline is AMAZING!). My three wingmen were overhead and I thought I was doing a pretty good job of keeping conscious. Most importantly, from participating in ResCaps myself, I knew that everything literally came to a halt when a pilot was down, with whatever it took ready, willing, and able to do the necessary to "make the save."

And make the save they did. My three wingmen stayed overhead and other flights of two and four stayed on standby in case they were needed. Indeed, things did come "to a halt" until I had been successfully rescued. Most importantly, an Air Rescue and Recovery Service task force of two Jolly Green Giant helicopters and four A1E Skyraider Sandies came to get me. Upon entering the area, the Sandies first dragged the area, flying low and slow, doing all they could to entice any enemy in the area to shoot so they could wipe them out before the slower, and even more vulnerable, Jollies came in. When the area was deemed reasonably clear, a Jolly came in, hovering while lowering one PJ. While he stayed on the ground determining a second PJ was necessary to pick me off the bamboo and put me in a litter, the Jolly stayed close by, in harm's way, awaiting his evaluation. Once the evaluation was relayed, the crew again put themselves directly over me while the litter and the second PJ were lowered. The two PJs picked me straight up off the bamboo and gently placed me in the litter. The Jolly then returned and pulled us all out of enemy territory. Never once, in 70 previous missions, did I spend so much time directly over enemy territory! Yet, they did it freely, as part of their job, to get me out. They risked everything for me.

I am convinced it was the initial, skillful stabilizing of my limbs accomplished by the PJs

during the 90 minutes it took to get back to Udorn that allowed me to recover so successfully. Once at Udorn, I was immediately put into an ambulance to head for the hospital. This despite my loudly verbalized desires to stay for the end-of-tour celebration for the PJ--T/Sgt. Lorenzo (don't call me Lorenzo, my name is "Tony") Willis--who had been the first at my side deep within enemy territory.

THE RECOVERY

When I hit the Udorn "hospital," I was checked over and declared to be in mild acute distress and in severe need of orthopedic surgery. I was later told that when the base commander learned of this--and the fact that there was no orthopedic surgeon in Thailand at the time--he fraged in the soonest available aircraft capable of transporting me to the USAF Medical Center at Clark AFB in the Philippines. That aircraft was a C-141! I clearly remember being in a litter at the base of the flight deck bulkhead and being able to look towards the tail of the aircraft and see nothing but the nurse who attended me on the flight from Udorn to Clark. That huge airplane, just for me.

On June 17, the day after the shootdown, a medical team operated on me to repair my left upper arm and right knee. Twice during that surgery, while my brain was anesthetized, my body quit on them. But they didn't quit on me--they brought me back both times. They also decided that to continue to try and repair the left knee was inappropriate, and waited a week to perform the second surgery. Once again, during that repair, my body quit on them. For the next 2 weeks, it was touch-and-go; most of the medical staff was convinced I wasn't going to make it. Over the following 8 weeks, the doctors eventually agreed I would make it, but, because of the extent of my injuries, I would never walk again. During that 10-week period, I was airlifted from Clark through Japan to Scott AFB and then to the AF Medical Center at Wright-Patterson AFB. There they replaced the original casts put on after surgery and sent me home for a month of R&R. Throughout, my care was constant and professional.

At Wright-Patterson the first week of September, Major LeGalle, the head nurse of the Orthopedic ward, came into my room. The Major was one very unique lady. To this day I feel her twin sister must have been Nurse Kratchet in One

Flew Over the Cuckoo's Nest. She ruled the Orthopedic Ward with an iron fist, but, believe me, it was necessary to keep us broken but not sick guys from terrorizing her staff at every opportunity. Deep down, I knew she was devoted, professional, and extremely caring.

Upon entering my room for the first time, she announced, "Lieutenant, you stink!" "Ma'am, when you spend 10 weeks half plastered, well, half in plaster, that happens!" I replied. She gave directions to have my leg casts removed so I could be given my first real bath in 10 weeks! Unfortunately, only bivalving of the casts occurred that day. More unfortunately, when my legs were picked up out of the bottom halves of the casts, I could see what had happened to them while they were in the casts.

While convalescing at home as well as in the hospital, I had been doing hundreds of leg lifts a day. I thought that when the casts came off, I would hit the ground running, back to join my squadron and complete my tour. When I saw my atrophied legs, I began to wonder if what was being said about my never being able to walk again was true. About 3 am the next morning, during a sleepless night, my concern overcame common sense. The cast pieces had merely been taped together because, again, there had not been time for a bath the previous evening. I undid the tape, removed the cast tops, and swung my legs over the side of the bed. There was zero range of motion in both, and the right was bent backward 5 degrees at the knee. Nonetheless, I grabbed the back of the bedside chair with my good right hand and stood up. At that moment, I had to know if I could stand on my own two feet. When I found I could, I pushed the chair ahead of me and shuffled across the room and back. It was only later that I realized I had done the most stupid thing I could have done--what would have happened had my legs given out, either knee had twisted, or I had fallen over onto my left arm?

But none of that happened. The next morning I did the second most stupid thing: I told my wife what I had done. She immediately told Major LeGalle. Within minutes the major was in my room with a gurney. She picked me up (my weight was 175 when I was shot down; now--well, when next I stood on a scale--I was at 123), put me on it, and then pushed me into a small treatment room. There she gave me a 5-minute lecture on stupidity

and finished with the ultimate threat to a fighter pilot: "Lieutenant, anyone who does something that dumb belongs in Pediatrics!" She left and my wife had to retrieve me from the treatment room.

I finally had a bath and then later that day a corpsman came into the room asking, "Are you Lt Dumbdumb?" He must have been talking with Major LeGalle. He put me in a wheelchair, gave my wife directions, and told her to pack me up. Admittedly, I wondered if we were going to the Pediatric ward. The directions, however, led us off base and I wondered if Major LeGalle had somehow gotten me discharged. Thankfully, the directions then led us back on base and to a Quonset hut. Inside we were told to wait in a curtained area. Soon a gentleman in a white coat came in, and he, also, inquired if I was Lt Dumbdumb. I replied, "I guess so, who are you?" His response was, "I am the brace man. Major LeGalle said that if you were going to do it, we are going to make sure you do it right!"

I was measured for leg braces then and there. The very next morning they were delivered to the hospital and fitted to my atrophied legs. That afternoon I was sent to Physical Therapy. There I was introduced to two corpsmen therapists who said they were going to bend me, and I should tell them when it hurt. I asked if they would know if they were about "to hurt it." When they responded in the affirmative, I kindly asked that they "stop just short of hurting it, and let's not worry about hurting me." My wife used to say I would return from the twice-daily sessions with my whole body looking that pale yellowish-green color you see just before a bruise completely heals. My wife was a saint throughout my recovery; she was by my side in the hospital whenever she could be throughout each and every day. At night she stayed in base housing, alone. I am convinced the support and encouragement she gave me was every bit as important as the professional care bestowed upon me by the medical professionals.

Although the initial physical therapy was most uncomfortable, things did get better. Once I regained some flexibility, another therapist--whom I called the "nerve lady"--was assigned to me. In addition to being a therapist, she was also Miss Wright-Patterson 1969. She was about five feet four inches tall with long blonde hair and deep blue eyes. She was beautiful. And did she ever stimulate me! Admittedly, however, to this day I wish that

just once she had not used electricity to do it!

The results of the continuous medical, therapeutic, and personal attention resulted in my being placed on Temporary Duty Retirement List (TDRL) status, admittedly under a 100% disability rating, just over 6 months after being shot down. Although I had leg braces on both legs and the fingers of my left hand were suspended in a contraption we called a monkey swing, I left the hospital able to get on with the rest of my life.

The care didn't stop, though. Using benefits under the GI Bill, I earned an MBA. Twelve months after first being put on TDRL, I went back to Wright-Patterson for re-evaluation. Physicians, nurses, and specialists continued to look after me to assure there was

no deterioration in my condition. Instead of deterioration, the evaluation confirmed nerve regeneration would allow me to get rid of the short brace on my left leg. Then, at the 24-month evaluation, a physician found that one of the tendons in my right knee had somehow reat-

tached along the interior side. He felt that if it were again severed and relocated to the lower front of the knee, I could possibly get rid of the right full-length brace. He went on to say that chances were one in a million and even if it did work, I would have to exercise and maintain condition for the rest of my life--but, but, but. It was obvious he wanted to try the surgery, but it was just as obvious he had extreme doubts about whether the tendon could stay attached to what basically amounted to scar tissue.

Regardless, he was telling me about the possibility of not having to strap in to what I had come to call my "leg iron" just to walk around every day. Therefore, I asked him to stop talking about all the "buts" and to please schedule the surgery. It was done, and I have never again worn a leg iron. Indeed, 6 weeks after recovering from this



"All the parts were still there; they just needed--and received--the healing touch!"

last surgery, I was permanently retired with only a 60% disability.

THE EPILOGUE

Obviously, I was given not just life, but a capability for a wonderful quality of life. That quality has included so many additional blessings. My wife and I have thoroughly enjoyed over 32 more years of marriage. We have been truly blessed by two incredibly special children. Our daughter, Dànielle Nicole, was recently awarded a Ph.D. at Michigan Technological University and is currently a tenure-track Assistant Professor at Michigan State University. Our son, Jason James, having graduated Magna cum Laude (and a Phi Beta Kappa) with a BS in Cellular Molecular

Biology from the University of Michigan, is now pursuing a Ph.D. in Immunology at the Stanford University School of Medicine. My wife and I both wonder and dream about the differences they are each making and are going to make in the lives of thousands of people.

As for me, while working towards my MBA, I also had the opportunity to collaborate on the script for and be featured in the Air Force film *Faces of Rescue*. I have been told this award-winning film was not only the documentary of concerted air rescue and recovery efforts in Southeast Asia, it was also used in PJ training for some 20 years. Personally, I still use it when giving my "civilian" talk and loan it to anyone who shows even the slightest interest in viewing it.

Following the awarding of my MBA, I took a position with Amway Corporation and served there for over 28 years. I also used Veterans' Benefits to obtain both Flight Instructor Airplane and Flight Instructor Instrument pilot ratings. I was given back the capability to teach people to do what I loved so very much. And, in

my "spare" time before job, family, and other obligations precluded being able to take the time, I did just that. Job responsibilities did increase rather quickly as I moved through taking on different roles and performing different tasks with Amway. The culmination of all this work came throughout the 1990s when I worked in an area called New Market Development. While I worked in this exciting new department, I had the huge good fortune to be able to personally start seven new affiliate companies in Hungary, Poland, the Czech Republic, Slovakia, Slovenia, Greece, and Romania. I lived in most of these countries for over one year each and had the privilege of getting to know their people, their cultures, their customs, and their lands.

Since the early development, the companies started have generated well over one billion dollars in sales. Over six hundred million of those dollars have gone right back into the hands of individuals who, mostly, had not dared even dream of starting their own businesses and earning that kind of money just years before, while they lived behind the Iron Curtain. My years with New Market Development also provided the opportunity for me to return to Southeast Asia. Ironically, I stepped off an airplane in Hanoi to start researching company-starting possibilities in Vietnam shortly after celebrating my 50th birthday. Unbelievably, when I checked a cal-

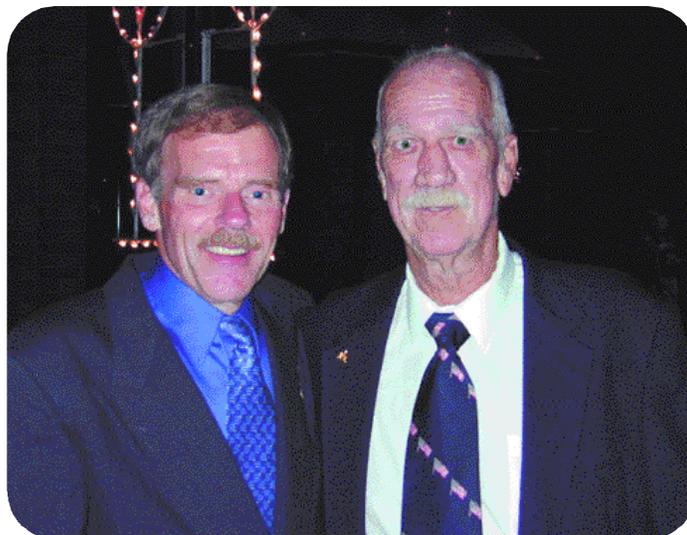
endar while reflecting on being 25 when I was shot down, I confirmed the day I stepped off that plane was the day on which I had lived exactly as long after my shoot-down as before.

To note just one more phenomenal life experience, I noted earlier that I missed the end-of-tour celebration for the PJ who initially dropped from the Jolly to assess my condition on the June day in 1969 when I was shot down. On December 10, 2001, after I told my story before an audience for perhaps the 200th time, Tony Willis came on stage. I had the chance to meet and again thank this phenomenal individual who plucked my butt out of the jungle. In the midst of the hugging that ensued, Tony said to me, "Man, I'm a PJ, I can't cry!" I told him "Tony, I'm a steely eyed fighter pilot; I'll cry for both of us!" Tony later told me he had personally rescued ten downed pilots, and I was the first he had ever met again. What an incredible thrill - and honor - for me.

Truly, it has been an incredible life. I have looked forward to every sunrise and enjoyed every sunset. And, I look forward to many, many more. America, in general, and all of you reading this in particular, thank YOU for my everything!

ACKNOWLEDGMENT

I would like to thank my daughter, Dànielle, for assisting with the editing of this piece and helping to prepare it for submission.



"Isn't it wonderful that I can grow old thanks to the gentleman next to me!

And to so many others!!"

Jim DeVoss (left) with Lorenzo Willis (right)

Photo courtesy of MSG Samuel R. Rodriguez

Psycho-endocrine Research in Vietnam

Peter G. Bourne, M.A., M.D.

Drafted into the Army as a physician in 1964, I happily negotiated a deal to be assigned to Walter Reed Army Institute of Research (WRAIR) in Washington for three years instead of the two I was originally supposed to spend in, what I assumed would be, the very boring de-militarized zone in Korea. At that time there was considerable concern about the high percentage of recruits who developed serious viral upper respiratory infections (URI) in basic training resulting in some fatalities. Given the very large volume of draftees coming in as a result of the Vietnam war this posed a serious problem for the higher command. Although it was in the early days of our understanding of the mind/body relationship, my superiors at WRAIR believed there was a correlation between an inability to adapt to the psychological (and physical) stresses of basic training and the development of viral infection. I was sent to Fort Dix, New Jersey to establish a research protocol which sought to correlate psychological adaptation, corticosteroid levels, and the onset of URIs. This was unfortunately before the era when the response of the immune system could be directly monitored. However, suffice it to say that a strong correlation did emerge. But it was frequently those who adapted least well to civilian society who tolerated the stresses of basic training the best. Those who had come from tough impoverished families, had been in juvenile detention centers or even prison not only adapted easily to military life and stayed physically healthy but some even regarded basic training as a degree less stressful than their previous existence. On the other hand, recruits from highly sheltered backgrounds, many of whom had never slept a night away from their parents or even eaten a full meal outside their home were the least able to cope, had the highest corticosteroid levels, and were the most likely to develop severe viral infection. I recall a brilliant, very well adjusted and physically capable librarian from Boston for whom the culture shock of the military was overwhelming and who came as near to dying as any of the men I studied.

By mid-1965, the war in Vietnam had escalated and WRAIR was increasingly focusing its

attention on combat related medical issues. It established a center in Saigon to study a full range of medical issues affecting the army in the war, especially malaria, dengue, and gastro-intestinal diseases. By then, increasingly viewed as the in-house expert on the psychological and physiological aspects of stress I was selected to go to Vietnam for a year, as part of the WRAIR medical team, to look at stress in combat as opposed to basic training. There had been a long, proud, tradition of clinical research on "combat fatigue" in the US military dating back to World War I. Although studies had always been done on those who became casualties, no one had tried to look at those who adapted successfully to the stress of combat. No one either had looked at the physiological changes that occurred in a war situation. Corticosteroid levels had been looked at in the parents of children dying from leukemia, in athletes in highly competitive events, in race car drivers and in USAF bomber crews flying peace time missions. No one, however, had looked at corticosteroid changes in people actually in combat and tried to correlate it with the life threatening experience they were exposed to. I set out to do this.

I need to find groups in high stress situations and decided to study helicopter ambulance crews who were subjected to short stretches of highly life-threatening stress interspersed with long periods of boredom as they waited, sometimes for days, at their base to be called into a firefight. And secondly, I chose Special Forces "A" Teams who operated under permanent threat with periodic combat episodes and the ever present possibility that their bases would be over-run. Separately during my period in Vietnam, I also did a comparative study of psychiatric casualties in US and Vietnamese troops and of Vietnamese civilians hospitalized with psychiatric problems.

Shortly after my arrival in country I went to the Medevac helicopter base at Saigon's Ton San Nhut airbase to talk with the commanding officer about doing the study on his men. He was fully supportive and enthusiastic. However, while I was sitting drinking coffee with him in his office, a call came in that there was a major firefight going on in

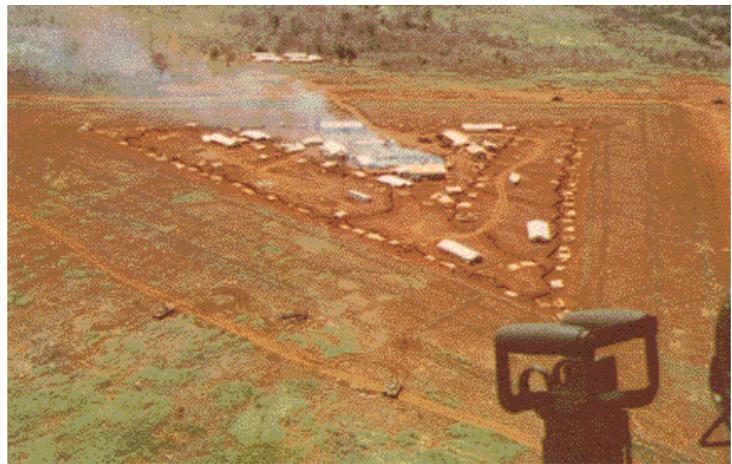
the Iron Triangle with significant casualties. Jumping up and throwing me a flak jacket he said, "Why don't you come with us and see what it is all about?" I confess that it was the only time in a year in Vietnam that I consciously felt fear. I was totally unprepared psychologically for what I had planned as a routine office meeting to suddenly become a life-endangering combat experience. In a major skirmish involving the 173rd Airborne there were already 44 US fatalities with a substantial number of wounded. We spent the rest of the day and much of the next evacuating the dead and wounded under heavy fire. I was able to put my medical training to good use and gained a rapid induction into combat as well as a good deal of insight into what my prospective subjects were enduring on a daily basis. I would have extensive combat experience over the next twelve months, but, perhaps because it was my first exposure, the memories of the terrible suffering I saw that day are some of the most enduring of my life. I particularly remember a blond-haired 18-year-old, shot through the spine, who throughout the flight kept asking "My legs will be okay won't they?" It was reflective of the strange nature of the Vietnam conflict that when I returned to the WRAIR administrative offices in the center of Saigon at the end of the afternoon, prior to heading to a cocktail party for the visiting Surgeon General of the Army, my commanding officer, who had warned me against taking unnecessary risks, said "What did you do today?" I replied "Oh, I just had some meetings at Ton San Nhut."

The implementation of the study of the dozen Medevac crew members proved fairly straightforward. They lived together in a compound near the airport making it relatively simple to supervise the collection of twenty-four hour urine samples and blood for the corticosteroid studies. The proximity to the WRAIR headquarters and labs in Saigon made it possible to freeze these samples and ship them back to Washington for analysis. It also made it easy for me and my assistant to interview the subjects, including having them take various psychological tests, and to spend considerable informal time with the group getting to know each of them intimately.

The plan to work with Special Forces posed a far greater problem. Fortunately I had, in basic

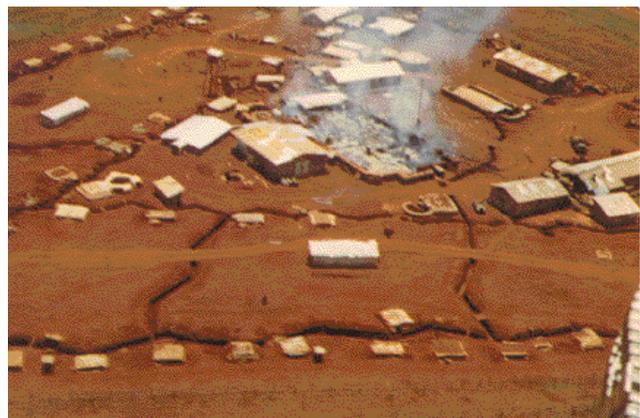
training at Fort Sam Houston, become friendly with a Yale medical school graduate, Craig Llewelyn, who was beginning what would prove a very distinguished career in military medicine. He had preceded me to Vietnam where he was the chief medical officer for 5th Special Forces. He arranged for me to visit a series of isolated 'A' Teams along the Cambodian and Laotian border to try to sell them, with his backing, on collaborating with my planned study. Travel to isolated camps was always difficult and unpredictable so to some extent the camps I visited were determined by where I could hitch a helicopter ride to.

The first camp I flew into was Plei Me. It had just survived an all out Viet Cong attack and much of it was still in smoking ruins. The attack led



Flying into the Special Forces camp at Plei Me as the Viet Cong siege was being lifted. The siege was the prelude to the battle of the Ia Drang valley, the first major battle between conventional units in the Vietnam war.

to the battle of the Ia Drang valley, the first major engagement between US regular forces and the North Vietnamese Army (NVA).



Coming in to land at Plei Me. The team house, set on fire during the enemy attack, is still burning.

It quickly became apparent that the survivors were in no mood to collaborate with a still rather green medical corps captain, especially committing themselves to providing twenty-four hour urine samples and sitting still for hours of incongruous interviews. In addition the camp was, for some reason, over-run with rats and mice, that kept me awake by walking on me at night and I did not fancy weeks of insomnia. At a number of other camps either there was overt hostility from the team commanders who felt who they did not want to be bothered with something that seemed irrelevant to their mission. In others I considered the chemistry was just not right in terms of getting the full collaboration of the team. This was usually because of serious pre-existing conflicts between the team members or with their Vietnamese counterparts. Finally at Duc Co I found a receptive team leader, Captain Conway. He was already on his third Vietnam tour and described to me how he and four other Alabamians had come together to Vietnam with 5th Special Forces. Over time, the others had all been killed. He was, so far, the last survivor he told me with a wistful sense of fatalism. He was a sophisticated mature individual, very committed to the military, and very willing to buy the notion that my strange requests, inconvenient though they might seem for his men at the moment, could have important long-range benefits for combat effectiveness. He called the team together and, explaining the importance of the study, told them he wanted me to have their full cooperation. It was a major breakthrough and I was highly elated as I returned to Saigon to prepare for an extended stay in the camp. The biggest obstacle I faced was that the twenty-four urine specimens had to be frozen right after collection and kept frozen all the back to Washington, D.C. Initially it sounded like an insurmountable problem. However, a technician in Saigon told me that he could modify a field refrigerator, run off a generator and used to cool blood and plasma for transfusion, so that it would go down well below freezing. With this and a variety of other equipment I prepared to return to Duc Co for a three month stay with my assistant, William Coli. As we were preparing to leave I received a cable from a Captain Wells Cunningham to say that Captain Conway had been killed in action and that he was his replacement. Because of the commitment made to me by Captain Conway I was still welcome to

proceed with the study. It was, however, with some trepidation that I returned to Duc Co, begging with the usual relentless delays and frustrations, the C-130 and helicopter transportation for us and our equipment each step of the way.

I need not have worried. Captain Wells Cunningham and I hit it off immediately and we ended up as close personal friends sharing a bunker to sleep in together. With his support I was able to



The author with Captain Wells Cunningham, commander of the "A" Team at Duc Co.

get the 24 hour urines I needed and with the camp much like a little island it was relatively easy to pin people down for interviews and other data gathering I needed. In addition to the biochemical studies I was particularly interested in group dynamics as they played out among the twelve members of the Team in a confined environment subjected to constant stressful demands. Most were strong unique personalities who were more inclined to be loners than team players. I was also fortunately able to supplement what I found in this one camp by periodically visiting others along the Vietnamese border, convincing helicopter pilots who had flown in for other reasons to take me where I wanted to go.

Periodically mortared by the enemy and subjected to occasional probing attacks through the camp's perimeter, every member of the "A" Team had a specific job that crucially contributed to the group's security, while the role of my assistant and myself was somewhat nebulous. At the same time, sustaining our acceptance was essential to the success of the studies. Not only were we not contributing in a combat sense we were relying on others to risk their lives to protect us. Our research work took only a small part of our time so I decided it was essential, to justify our presence there in the eyes of



The author during the battle for Plei Beng

our subjects, that we create a clear role for ourselves consistent with the overall mission of the 'A' Team. Initially this took the form of actively upgrading the medical civic action program that the Team's medics had initiated with the local Montagnard population.

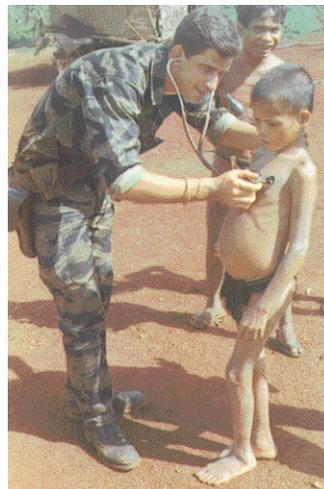
We launched extensive vaccination and sick-call programs in the villages accessible from the camp and cleaned up a water source that had been the cause of several cases of typhoid. I also



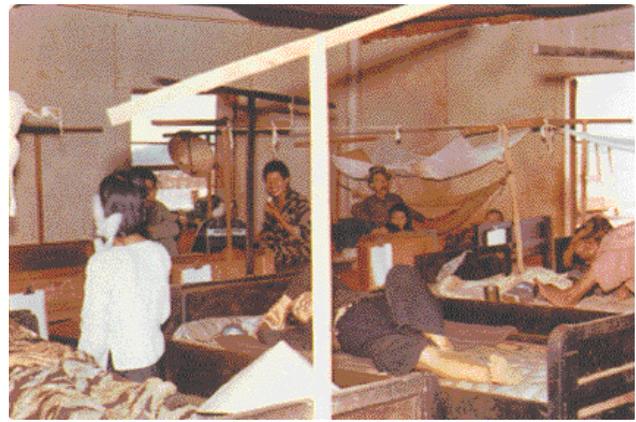
The author immunizing children in a Montagnard village.

spent much of each day working in the small hospital that had been set up inside the camp's perimeter to treat the families of the hundred or so Montagnard soldiers fighting with the Special Forces team.

Yet I still felt vulnerable to the criticism that I did not really know what it was like out on patrol in the jungle, especially the long-range patrols into Cambodia, what the ever present fear of ambush or treading on a mine was like, or what a serious fire-fight involved when you were heavily out-numbered. In



Holding sick call.

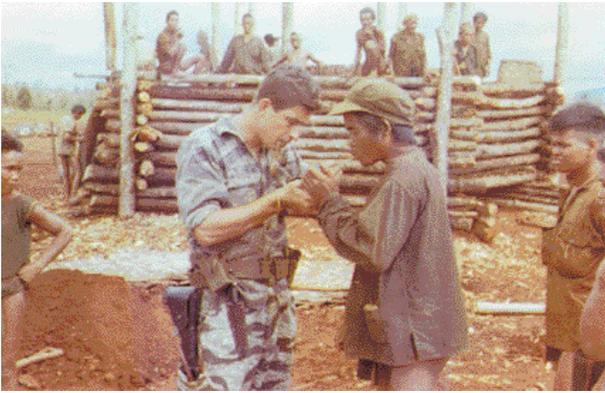


Inside the small make-shift hospital at the camp.

retrospect, I, perhaps foolishly, succumbed to these pressures and began going out on patrol as a way of trying to establish credibility and acceptance with my subjects. These experiences reached a peak with a night and day march through heavy jungle terrain in order to attack and capture a Viet Cong village called Plei Beng. Perhaps never fully accepted, this putting aside of my medical role, which my superiors could never understand, did go a long way in helping me to be accepted as just another member of the group. In retrospect it was essential to getting my primary job accomplished.

At the end of the three months we had collected all of the data we needed and had a substantial quantity of frozen urine. The question was how to get it from the highlands of Vietnam to WRAIR in Washington without it thawing out. I needed a supply of dry ice but no medical facility in Vietnam had any. Eventually, in Saigon I found the French beer company "Beer La Rue" produced it and I obtained a substantial quantity which I packed into several styro-foam boxes. Then, with sympathetic pilots I had come to know over the preceding months, I lined up a dash back to Duc Co to collect the urine samples, rushing back to Saigon before the dry ice and urine melted. Repacked with fresh dry ice, I put the samples on a plane at the Saigon airport and arranged for a colleague from WRAIR to meet the flight when it arrived in Washington.

A few months later Captain Wells Cunningham, who had been so instrumental to my success, was killed in combat. At that time the life expectancy of a Special



Sharing a smoke with Montagnard friends.

Forces “A” Team leader was three months; the worst mortality rate for any American unit in any war. I would finish my tour in Vietnam and return for a year to WRAIR in Washington. There the data I had collected became the basis for several scientific papers and two books. Over the years, our knowledge and understanding of stress in both a military and civilian setting has advanced substantially. At the time, however, these studies significantly enhanced our knowledge of the psychological and physiological aspects of stress, and as far as I know, they have never been duplicated in any similar combat setting.

REFERENCES

- Bourne, P., (Ed.) *Psychology and Physiology of Stress*, Academic Press, New York, 1969
Bourne, P., *Men, Stress, and Vietnam*, Little Brown, Boston, 1970



Peter G. Bourne, M.A., M.D. former Captain, US Army Medical Corps, is now a Visiting Scholar at Green College, Oxford and Vice Chancellor Emeritus, St. George's University, Grenada, West Indies.



Guerrilla Hospital Quality of Care--Part 2

Warner Anderson, MD

In the last edition I wrote about the recent recommendations by several professional societies and government agencies regarding antibiotic use. Universally, they recommend less, rather than more, antibiotic use. Specifically, most cases of otitis media, sinusitis, sore throat, and bronchitis in otherwise-healthy adults will get better without antibiotics, although supportive therapy with analgesics and sometimes decongestants is recommended.

Some other common outpatient problems are traditionally treated with antibiotics in the United States, in spite of strong evidence that antibiotic use does not help, and indeed may harm, the patient. In addition, many bacterial infections which are known to respond to antibiotics are treated for much longer (more unneeded doses; more side effects) than required. For example, an infection which may usually require three days of antibiotics may be treated for ten or even fourteen days. In an austere environment, the foolishness of this thoughtless superstition is most evident.

Skin infections represent a big antibiotic requirement in an austere environment, or any other operational environment. Ranging from impetigo to abscesses to cellulitis, they can interfere with operational readiness and, improperly treated, even lead to death. An environment with poor sanitation, where it is difficult to wash the infected area or one's hands, sets the soldier up for such problems and makes them even harder to treat and contain.

A localized area of impetigo is adequately treated with regular applications of bacitracin, twice or three times a day, after a good scrub. To avoid spread of the infection, it is sometimes best for the medic to provide 2x2 or 4x4 gauze sponges which can be discarded after each scrub. Any soap works about as well as povidone iodine (Betadine®), so there is no real reason to dig into the medic's stocks of that. The lesion should be covered with a Band-Aid® or similar simple dressing when possible; this prevents lesion-to-nose colonization. Similarly, if

the patient has any insect bites, poison ivy or other sites that are prone to scratching, covering them with a minimal dressing can prevent infection spread.

More widespread impetigo, three or more lesions, calls for the big guns. Although the chain of assumption has traditionally been "impetigo = Group A Streptococcus pyogenes, therefore ten days' treatment," in fact it's more often staphylococcus. In any event, treating with a good anti-strep, anti-staph antibiotic such as cephalexin for three to five days is usually enough to clean up the lesions for natural defenses to complete the job with half the total doses. And no antibiotic is going to adequately substitute for poor hygiene.

An abscess is a bit of a peculiar lesion, in that many practitioners load the patient with antibiotics for days prior to draining it, in the superstitious hope that it will resolve without the application of stainless steel. In fact, a well-localized abscess can be drained without antibiotics prior or after drainage. I would give a patient antibiotics orally - again cephalexin is a good choice - if the abscess were extensive and septated, requiring a lot of exploration and adhesions to be disrupted. But if you cut it with a scalpel blade and the pus runs out without a lot of poking and prodding, the packing will prove curative and the antibiotic a waste of resources.

Cellulitis is a pretty solid indication for antibiotics. Basically, if the patient could fight the infection without antibiotics, he wouldn't have the cellulitis to begin with. The question is really a matter of timing. If the patient is febrile (>100 degrees) then the antibiotics should be given until the patient has been afebrile for 24 - 48 hours. An already-afebrile patient probably has a less serious infection, still requiring antibiotics, but the duration can be abbreviated. Draw a line around the advancing edge of the cellulitis; when the redness begins to retreat, continue treatment for another day or two. In any case, do not forget warm compresses to increase circulation to the affected part - but do not let the skin macerate from too much moisture.

Diarrhea may rarely require antibiotic treatment. Fever and purulent stool are usually thought

to be signs of invasive disease, and most would treat this type of diarrhea empirically with antibiotics. Salmonella, however, may actually be made worse with antibiotic therapy. Importantly, these symptoms in a patient recently treated with antibiotics should prompt consideration of pseudomembranous colitis - a condition that is often fatal and can be hard to treat.

Turista, which occurs in travelers to the U.S. almost as often as in Americans traveling abroad, usually responds to loperamide (Imodium®) and bismuth subsalicylate (Pepto-Bismol®). However, if the diarrhea is disabling, three days of ciprofloxacin 500 mg bid is a reasonable intervention. A longer course for turista is probably a waste.

Urinary tract infection in women can be thought of as either pyelonephritis or cystitis, although there are a few other less-common conditions, such as hemorrhagic cystitis or urethritis to keep in mind. However, a woman with a fever and constitutional symptoms such as vomiting, along with a positive urinalysis, usually can be assumed to have pyelonephritis. If vomiting is a problem then intravenous or intramuscular antibiotics are needed. However, if the patient can keep down oral medication, ciprofloxacin 500 mg bid alone or with cephalexin will almost always be adequate, without the need for IV fluid.

A woman with dysuria and no fever or vomiting and catheterized dirty urine can be considered to have cystitis. Numerous studies have shown that these patients will resolve with either a single dose of medication, such as gentamicin 80 mg IM, or a large dose of amoxicillin, such as three grams orally at one dose. Slightly better results are obtained from three days of cephalexin 500 mg qid for three days,

or TMP-SMX DS bid for three days. If symptoms do not improve within the three days, then extension to seven or ten days is warranted, or the antibiotic should be changed.

In trichomonas vaginitis, the medic can choose from two standard therapies. He can give metronidazole 500 mg bid for seven days, totaling fourteen tablets, or he can give a single dose of two grams po stat, a total of four tablets. However, the incidence of vomiting is high in the single dose therapy, meaning that the savings may be illusory. If twenty percent of women vomit, then for every twenty tablets given, four will be wasted, and the one who vomits will need the bid for seven days regimen. However, treating those same five women with fourteen doses of metronidazole will cost a total of ninety-eight tablets. So the total metronidazole used in treating five women with single-dose therapy turns out to be about thirty-four tablets versus the longer regimen's cost of ninety-eight tablets - about a third of the cost in resources.

Again, saving a bottle of tablets may seem insignificant to the practitioner sitting behind the desk in a comfortable TMC or sick bay, but when everything has to be air dropped into the AO in the middle of the night and rucked to the G hospital, seemingly minor savings can become a big deal.

The SOF team medic usually enjoys one advantage that other health care personnel do not have - they are in contact with their team members on a daily basis. Thus, instead of giving antibiotics for some mindless and irrational interval, he can provide them a dose or two at a time and watch for the desired result. When the patient's infection is resolving, then there is often little to be gained by pouring more antibiotics down the black hole.



Jungle Survival

Steve Anderson, PA-C

This was my first trip to a Central American country (case of beer). We were taking part in some jungle training the unit had planned that was approximately 2 to 2^{1/2} weeks long. As a SF medic of about 7 years at the time, I had really grown up in 10th Special Forces Group, which concentrates its training in the European areas of the world. I had just recently moved to this unit, which operated in a variety of locations and environments. I anticipated the countryside and climate to be quite hilly, hot and humid and wasn't disappointed. As we moved into our base camp, we commenced classes/lectures and field craft review that would be used in the upcoming days. That first few days of preparation also allowed for some acclimatization (pretty sure none occurred but...). After several days we loaded up and moved out to remote training areas where we were dropped off solo at sporadic intervals (500-1000 meters I'm guessing) with standard LBE, weapon and knife. After some scouting I found what I thought was a suitable hooch site. Meticulous planning and solid construction resulted in a lean-to that would easily rival any dilapidated cardboard box shanty I've seen in other parts of the world. OK, the hooch needed some work. It got dark so I settled in for the night. I whiled away the time trying to avoid that tree root I somehow missed in my thorough survey of my ground floor. I noted just how dark it was and rolled outside my hooch think'n maybe it's just the finely woven thatch roof that was blocking any light from coming through. Nope... that wasn't it. Jeez, I couldn't even see my hand 2 inches from my face! I rolled back under my hooch, d'oh...frick'n root.

More time passed then the inevitable light pitter-patter of rain hitting foliage began. The crescendo was quick and the noise roaring. Just a quick flick of the backlight button on the 'ol Casio to note the onset of the rain...holy #hit! Just about blinded myself with that sudden flash... man these things are bright. Then, the sound and sensation of rain finding its way through the impenetrable roof

of my condo hitting my legs, chest and forehead...#hit. OK...the hooch needed some work. The rain subsided about as quickly as it started. Things were now still and quiet, save for the occasional drip still slapping me in the face. Off to my right in what seemed to be hundreds of meters away, some kind screech was heard. Hmm...wonder what that was...no matter, pretty far off. Time was flying now at a snail's pace. Another screech. Closer, still off to my right. Hmm...wonder what that was...no matter...hmmm, moving this way. My fingers were now lightly tapping my CAR 15 as thoughts of the recently released movie "Predator" floated through my mind.

Another screech...#hit! That one was definitely closer and seemed to be coming up from the trees, still to my right and slightly front. This thing must be pretty good sized I was thinking. My fingers were now tapping faster on the CAR, thoughts of Predator now more frequent in the 'ol brain housing group. Another screech, this one just about overhead and so loud I jumped. This frick'n thing must be huge I thought! OK...no way am I die'n lying here on my back. I rolled from under my hooch and stood, weapon at port arms, head swiveling back and forth, scanning. There I was...a romp'n stomp'n barrel chested freedom fighter...ready. Can't see #hit. Another screech. This one more to my left, still loud, seemed to be 20-30 meters off now. Hmm...was it moving on or just circling? More thoughts of Predator. My focus shifted for a second as I noted my knees were shaking. Huh...hadn't realized it cooled off so much to make me shiver. Now thinking...you're not cold, dumb #hit, you're scared. Another screech. This one way off to my left, not nearly as loud. Man, this thing moves fast...no noise in movement...#hit!

OK, I was thinking this thing was moving off towards my buddy Brown. Good luck buddy! Hope that thing misses you too. I relaxed and dropped back down to the jungle floor and rolled under my hooch. D'oh...frick'n root...#hit.

Two days later, I made a link-up as planned with my buds. We swapped lies...Did ya catch anything, find anything to eat? Hey, did you guys hear

that screaming out of the trees a couple nights ago? Yeah, was the reply with an awful nonchalant tone. What was that thing? Oh that was some howler monkeys, one of 'em said. Howler monkeys...suckers must be huge?

Chuckles and snickers erupted. Oh ...yeah...they said, they're gigantic. By now my

razor-like mind is sensing some sarcasm. Turned out as it was explained to me, these man-eaters stand about 2 foot high, make a horrendous howl, and can literally move through treetops at a run. Well, feeling a little foolish I figured I could chalk it up to experience. Hell...could have been some kind of Predator thing...#hit!



Vs.

A team of commandos, on a mission in a Central American jungle, find themselves hunted by an extra-terrestrial warrior... NOT





I am a former Ranger medic and currently a medical student interested in subscribing to the Journal of Special Operations Medicine. I am very excited that such a journal exists. I am on a military (Navy) scholarship and hope to serve as a spec ops doctor once I am done with school. Thank you for your assistance in putting me on the distribution list. After looking for several years to find a way to stay current on spec op medicine topics I was finally referred to JSOM by a friend.

Sincerely,
Brian Miller

I really enjoy reading the SOF Medical Journal, and through it I have met a few people (on line) that have the same views as I do and are able to help me direct my questions to somebody who knows. Again, being a Marine and unfortunately semi-out of the loop, I have been able to address issues to individuals that were acknowledged to the point, I feel, that the word is starting to get out about training issues. I would like to see the Marines and the Reconnaissance Units get more on track and crosstrain with the medics of their sister services and gain and disseminate the information they learn to their community. I am also passing on to my fellow Corpsman in the Marine Infantry Units areas to find such information. They are few and far between and have limited knowledge; of course they are not put into the scenarios like SOF medics, but the Marine Infantry is sent to all walks of life and has the same injuries and/or medical problems as anybody, only on a larger scale, i.e. an A team compared to a 40-60 man Rifle platoon with one Corpsman.

Thanks again for including me in the loop.
Semper Fi
GySgt Brian T. Foy

I have received the copy of Special Ops Medicine and am very favorably impressed with its content.

Thanks,
Michael Fuenfer, COL, MC, USAR

I am attending the Joint Special Operations Medical Officer Orientation Course where I received a copy of the journal, which i really enjoyed reading and is pertinent to my duties as an Environmental Science Officer.

1LT(P) Curtis White

Thanks, Looks great; good spectrum of articles across different interests.

Keep it up!
Steve Yevich COL (Ret)
(Former USSOCOM SG)

Announcements

The Pararescue Reunion is set for Sept 12-15, 2002, in Albuquerque, New Mexico, at the Radisson Hotel and Convention Center.

Hypothermia in the Austere Environment

Warner Anderson, MD

The last edition of the Journal of Special Operations Medicine, Winter, contained an article on hypothermia. The author, Gordon Giesbrecht, PhD, provided a good overview of the current state of the art in this troublesome problem. However, the problems of field treatment by SOF medics remain.

Recent combat experience in Afghanistan, as reported in Army Times and elsewhere, has involved unexpected exposure to the cold at high altitude. A helicopter insertion expected to last only a few hours stretched into a night-long ordeal when the LZ came under intense, determined enemy fire. For troops going into combat, every pound of "snivel gear" they carry is a pound of ammo they must leave behind.

Hypothermia is arbitrarily divided into mild, moderate, and severe. This division is based upon core body temperature and serves a specific purpose - guiding therapy in the emergency department. However, in the field and under the care of the SOF medic, another way to classify hypothermia is shivering hypothermia and non-shivering hypothermia.

In shivering hypothermia, the patient needs insulation, fluids and fuel. The patient will almost always be able to swallow fluids, and so hot fluids, where tactically sound, make good sense. However, heating the fluid is definitely a secondary consideration.

Why?

For ease of calculation, your patient is a 100-kilogram soldier, or 220 pounds. His body temperature, if you had a thermometer, would measure 94 degrees. You heat a liter of fluid to 114 degrees, 20 degrees above his temperature. When he drinks the fluid, assuming it remains at 114 degrees the entire time he drinks it, he will in effect be making 1% of his body weight 20 degrees higher - and making his whole body weight 0.2 degrees

higher. If you want to raise his core temperature to 98 degrees, then you are only 5% of the way there. Of course, that's better than cooling him further with ambient (cold) fluids, but the point is that health care personnel often overestimate the efficacy of their interventions.

Another recommended intervention, heated intravenous fluids, is frequently used in the emergency department. Here, the vein will not tolerate a 114-degree fluid, so this intervention seems almost silly. Indeed, in the best-case scenario, the nurse places the liter of fluid in the microwave for a couple minutes prior to giving the fluid. Then, almost invariably, no one wraps the IV tubing on its course to the IV site. The fluid, regardless of how hot it is leaving the bag, will enter the vein at ambient temperature, effectively cooling the patient. If the tubing is wrapped with fluffy gauze and the tubing routed along the arm, then the best one can hope for is that the fluid will enter the vein at the patient's (hypothermic) temperature.

In the field, I have successfully heated IV fluid by dropping a loop of the tubing into a canteen cup of hot water just before it enters the vein. Others have successfully used MRE chemical heaters for the same purpose, but there is always danger of thermal burns and explosion of the heated air, depending upon the method used.

Another frequently recommended technique of re-warming, inhalation of heated mist, requires a fairly elaborate setup and probably is hard to do in an austere environment, aside from putting another canteen cup of very hot water under a facial tent fashioned from a Mylar® casualty blanket.

In the emergency department, it is common for the respiratory therapist to place the heater at the portion of the inhalation circuit distal to the patient, and unless a veritable blanket of gauze is wrapped in the tubing, the temperature will again be ambient.

A study of chilled SEALs, conducted at the US Navy Experimental Diving Unit in Panama City, Florida showed that warmed inhalation prevents heat loss through expired air, but does almost nothing to actually re-warm the patient.

In the end, the shivering patient needs

insulation, fluid, and fuel so he can make his own heat. Providing a euthermic person a sleeping bag will be helpful, and placing warm canteens on the patient's axillae, inguinal regions, and neck will help speed the process. Also, a warm canteen on the scalp will provide a great deal of comfort and enhance the re-warming. A surgical mask, oxygen mask or cravat ("bandit") mask will help prevent inhalation of cold ambient air.

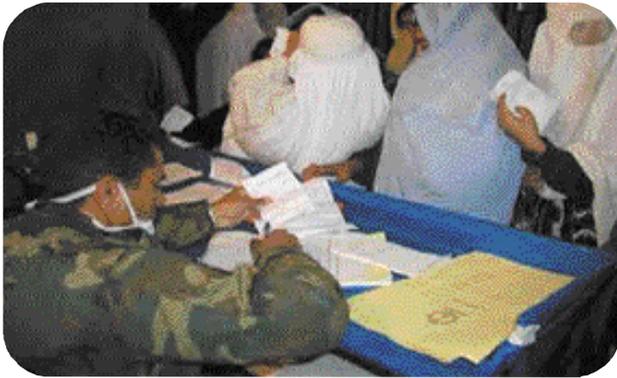
A non-shivering patient in an austere environment will not make his own heat, and will continue to lose heat unless actively re-warmed. Gentle handling and getting a couple other soldiers under the insulation with the patient will help as much as

anything. Again, warm canteens in the appropriate places and a mask will help. Rescue breathing, given enough people, can continue almost indefinitely in the absence of spontaneous respirations, and will even supply warmed air to the patient. In the absence of a pulse, in an austere environment, there is little utility in chest compressions and the medic should take some comfort from the realization that compressions may convert a very weak bradycardia into a ventricular arrhythmia, and thus could do more damage than help. Under the best hospital conditions, re-warming a moderately-severe hypothermic patient takes several hours. The prognosis is guarded, at best.

Warner Anderson MD



Photo Gallery



Afghanistan women dressed in the traditional Burka, registrar at a Jordanian hospital in Mazar E Sharif, Afghanistan

A local Afghan child in Mazar-e Sharif, Afghanistan, holds up a leaflet warning them not to pick up unexploded ordnances in their area, Dec. 17, 2001. The U.S. soldiers in Mazar-e Sharif have handed out over 7000 leaflets such as this one to help protect and warn the local populace of theses dangers in support of Operation Enduring Freedom.

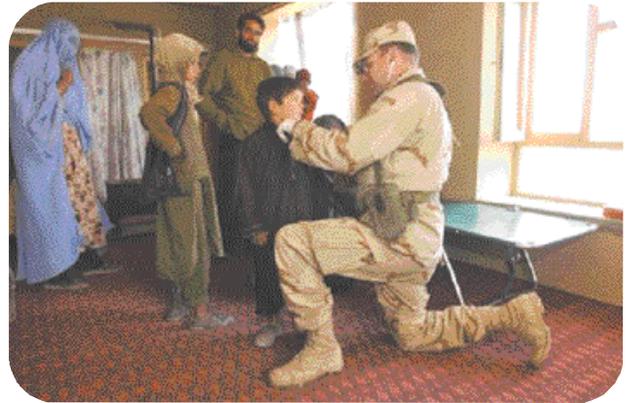
U.S. Air Force Photo courtesy of Staff Sgt. Cecilio Ricardo



Sgt. 1st Class Bob, Civil Affairs, U.S. Army visits an Internally Displaced Persons camp to discern the security concerns Humanitarian Aid agencies have on the camp, Dec. 23, 2001. Afghanistan locals in the IDP camp, Sakhi camp, in Mazar-e Sharif, gather to collect their portion of humanitarian aid being passed out by Doctors without Borders agency.

U.S. Air Force Photos courtesy of Staff Sgt. Cecilio Ricardo

Operation Enduring Freedom- A US medical person gives care to an Afgan boy.



Operation Enduring Freedom- A US medical person gives care to an Afgan infant.

A really cool picture!. A view of a flight deck while hanging by a rope from a helicopter.
Photo courtesy of COL Warner Anderson



Dedication

Tech. Sgt. Ronald O'Steen, a fellow pararescueman at Kadena's 320th Special Tactics Squadron, 353rd Special Operations Group, sketched this portrait of Staff Sgt. Juan Ridout (left) and Master Sgt. William McDaniel for the families of the two fallen comrades.



These two pararescueman and eight soldiers from Echo Company, 160th Special Operations Regiment in Taegu, South Korea, died Feb 22 in a helicopter crash in the Philippines. This mission was in direct support of Operation Enduring Freedom, assisting the people of the Philippines in their efforts to rid their country of the same terrorist network that attacked our country on September 11.

SSGT JUAN M. RIDOUT

25 June 1972 - 22 February 2002

SSgt Ridout was assigned to the 320th Special Tactics Squadron, Kadena AB Japan as a Special Tactics Pararescue Journeyman. SSgt Ridout's previous assignment was the 22nd Special Tactics Squadron where he participated in Operation Allied Force. During this operation, SSgt Ridout was involved on the Hammer 34 mission which ended in successful recovery of a downed Air Force pilot. He was named the 320th's Pararescueman of the Year for 1999. His awards and decorations include the Air Medal, Air Force Commendation Medal, Combat Readiness Medal, Air Force Good Conduct Medal, Armed Forces Expeditionary Medal, Armed Forces Service Medal, Small Arms Expert Marksmanship Ribbon, PME Graduate Ribbon, and the NATO Medal.

MSGT WILLIAM L. MCDANIEL II

12 June 1965- 22 February 2002

MSgt McDaniel was assigned to the 320th Special Tactics Squadron, Kadena AB Japan as the Special Tactics Pararescue Flight Superintendent. He was a veteran of Desert Shield and Desert Storm as an F-117 Crew Chief. MSgt McDaniel cross trained as a PJ in 1997. His first assignment was at the 66th Rescue Squadron, Nellis AFB, NV. While at Nellis, he participated in Operations Northern and Southern Watch. He was named the 320th's Pararescueman of the Year for 2001. His awards and decorations consist of the Air Force Commendation Medal (4 devices), Air Force Outstanding Unit Award, AF Good Conduct Medal, National Defense Service Medal, Armed Service Expeditionary Medal, South West Asia Service Medal with Bronze Star, Armed Forces Service Medal, Air Force Overseas Long Tour, PME Graduate Ribbon, Small Arms Expert Marksmanship Ribbon with Bronze Star, NATO Medal.

Ghosts

*Watching on a hollowed day
Wearing Class A's and a Green Beret
Anticipation on his face
Fearing ghosts from another place
Remembering those who gave their all
Their names engraved in a granite wall
Hands caressing warm black rock
Tells of all that we have lost
Warmth exudes, their presence felt
He traced their names in hopes to help
erase those ghosts from years gone past
To us a name, but not to him
A lonesome memory for a long lost friend*

SFC Stephen L. Young



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1. Use the active voice when possible.
2. Secure permission before including names of personnel mentioned in your piece. Do not violate copy right laws. If the work has been published before, include that information with your submission.
3. Articles should be double-spaced, twelve point font, aligned on the left and justified on the right.
4. Important: Include an abstract, biography, and photo of yourself as part of the article.
5. Use of acronyms should be held to a minimum and when used they must be spelled out the first time.
6. Remember that your audience is inter-service, civilian, and international.
7. Every article has a point to make, which is traditionally stated in the introductory paragraph and restated in the closing or summary. Subtlety is not usually a virtue in a medical publication.
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