Department of Veterans Affairs

Veterans Health Initiative

Cold Injury: Diagnosis and Management of Long Term Sequelae

Independent Study Course
1998, Revised May 1999
Reviewed October 2000
Revised March 2002

Sponsored by
Department of Veterans Affairs
Employee Education System

Employee Education System

This is a Veterans Health Administration System-Wide Training Program, sponsored by the Employee Education System in cooperation with the Office of Employee Education and the Office of Public Health and Environmental Hazards, Department of Veterans Affairs. It is produced by the Employee Education System.

DEPARTMENT OF VETERANS AFFAIRS
UNDER SECRETARY FOR HEALTH
WASHINGTON DC 20420

Message from the Under Secretary for Health

The independent study course, *Cold Injury: Diagnosis and Treatment of Long Term Sequelae*, was first issued in May 1998 and revised in May 1999. The issue of cold injury is an important one in military medicine. Exposure to extreme cold has presented a challenge for American service members both in combat and in training missions beginning with Valley Forge. Both World War II and the Korean Conflict saw large numbers of cold injury casualties. Veterans of the Battle of the Chosin Reservoir in Korea and the Battle of the Bulge in World War II were particularly hard hit by the cold.

It is appropriate that clinicians charged with the care of veterans be prepared to diagnose and treat cold injury. Therefore, I am pleased that the independent study course is being reissued as one of the modules of the Veterans Health Initiative, a program designed to assist clinicians in understanding the unique occupational hazards and health implications of military service. The Department of Veterans Affairs is committed to providing all veterans with health problems related to their military service high quality care for their disabilities, as well as complete and thorough examinations to help determine their appropriate disability compensation. This module includes information that will allow you to play an important role in this process.

I am pleased that you are taking this opportunity to educate yourself on cold injury. Your increased knowledge will help you better serve our nation's veterans.

Thomas L. Garthwaite, MD
Under Secretary for Health

Table of Contents Page

Independent Study Outline

Purpose

The purpose of this self-study program is to provide information to VA staff about cold injuries. In particular, this program will provide information on:

- The effects of cold injuries and the circumstances of their occurrence in the veteran population;
- The revision and expansion of evaluation criteria for cold injury in the rating schedule;
- The cold injury examination protocol; and
- Signs and symptoms associated with post cold injury medical sequelae and treatment recommendations.

Many VA physicians have had relatively little exposure to cold injury in their daily practices. However, extreme cold conditions have been a powerful enemy of American troops from Valley Forge to Korea. Cold continues to complicate training exercises and may well figure in future conflicts. The Veterans Health Administration wants to assure that veterans with cold injury receive high quality examinations and follow-up care.

Objectives

Upon completing the this self study program, participants should be able to:

- Describe conditions leading to cold injuries;
- Describe differences between freezing and non-freezing cold injuries;
- Delineate the signs and symptoms of acute cold injury;
- Delineate the signs and symptoms of long term sequelae of cold injuries;
- Identify key points to be included in the history of someone with reported cold injury;
- Understand appropriate examination and treatment protocols for patients with long term sequelae of cold injury; and
- Apply appropriate objective criteria to help in the adjudication of claims process.

Outcome

As a result of this program, clinicians should be able to apply the knowledge gained to conduct a more comprehensive examination and assessment of patients through a better understanding of the long-term sequelae of cold injuries and to the development of an appropriate management and follow-up plan.

Target Audience

The independent study is designed for VA physicians and nurses providing primary care; clinicians providing compensation and pension examinations for veterans; and for adjudication officers.

Written Materials

1. Background Material

A. One Veteran's Story, *Recollections of Hubert Edward Reeves on His experience with Frostbite in Korea* – It is suggested that you read this first because Ed Reeves story is a moving account of what combat was like under conditions of extreme cold. The Battle of Chosin occurred in –30 degree Fahrenheit temperatures in the North Korean mountains near Manchuria during November and December 1950. The allies suffered 12,000 casualties, including 3,000 killed, 6,000 wounded and many thousands of cases of frostbite. In all, over 6,000 victims of cold injury were evacuated from Korea during the winter of 1950-1951.

B. Introduction to the program by Susan H. Mather, MD, MPH – this paper covers:

1. Historical/Patient Perspectives – Interviews with Chosin Reservoir (Korean War) veterans, Donald McAllister, Ernest Pappenheimer, and Edward Beltran, and Battle of the Bulge (World War II) veterans, William Flynn and Eugene Smith, who discuss their experiences emphasizing:

   - Problems with protection from the cold
   - Nutrition
   - Access to water to prevent dehydration
   - Combination of combat injuries and the cold
   - Available medical care
   - Frequent lack of medical documentation

2. Summary of long term sequelae to cold injury

   a. Key Reference on Compensation – "Compensation Issues for Cold – Injured Veterans". Special paper prepared for this independent study, 1998 by Caroll McBrine, MD.

   b. Under Secretary for Health Information Letter, IL 10-96-030, dated December 31, 1996. *Recommendations for the Care and Examination of Veterans with Late Effects of Cold Injury:* This letter contains details of what should be included in the history and physical examination of the cold injured patient, treatment recommendations, summary of cold injury sequelae, and selected references for those wanting to expand on this basic program.

Written Materials

c. Under Secretary for Health Information Letter, IL 10-98-008, dated April 20,1998; Cold Injury Examination Protocol: This letter distributed the Cold Injury Protocol Examination and History to VA physicians in the field.

d. Compensation and Pension Examination (Cold Injury Protocol Examination, AMIE Worksheet #1730): This is taken from the Automated Medical Information Exchange (AMIE) for Compensation and Pension (C & P) examiners and provides guidance for conducting a thorough history and physical examination on individuals who have sustained cold injuries.

e. *Protocol Examination History for Cold Injuries:* This form can be used as a guideline for the clinician taking a history from a cold injury patient.

f. *Revised Cold Injury Regulations for Disabilities:* This describes the service connected cardiovascular ratings (10, 20 and 30%) allowed for specific residuals of cold injury. Amputations and complications such as scar cancers and peripheral neuropathy are evaluated under other appropriate codes.

2. *Key reference* for acute treatment of freezing cold injury

O'Malley, J., Mills, W., et al. *Frostbite: General and Specific Treatment, The Alaska Method*. Alaska Medicine Vol. 35, No. 1, Jan/Feb/March 1993.

3. *Key reference* on freezing cold injury

Mills, W. "Freezing Cold Injury". Special paper developed for this Independent Study, 1998.

4. *Key reference* on non – freezing cold injury

Oakley, E.H.N. "Non-Freezing Cold Injuries". Special paper developed for this program, 1998.

5. *Key references* on sequelae to cold injuries – These articles summarize the predominant sequelae to cold injuries. The guide contains a summary of each article, containing the most important points. Copies of the articles themselves can be obtained through your medical library.

A. Overview article:

Blair, J.R., Schatzki, R., and Orr, K.D. *Sequelae to Cold Injury in One Hundred Patients: Follow-up Study Four Years After Occurrence of Cold Injury*, JAMA April 6, 1957; 163(14): 1203-1208.

Written Materials
continued

B. Arthritis:

McKendry, R.J.R. *Frost Bite Arthritis.* CMA Journal Nov. 15, 1981; 125: 1128-1130.

C. Cancers in Frostbite sites:

Rossis, C.G., Yiacoumettis, A.M., and Elemenoglou, J. *Squamous Cell Carcinoma of The Heel Developing at Site of Previous Frostbite*. Journal Royal Society of Medicine Sept. 1982; 75: 715-718.

D. Late neurologic changes:

Altman, M.I., and Hutton, S.J. *Late Neuropathic Sequelae of Cold Injury*. Journal of Foot Surgery 1987; 26(3): 213-216.

Arvensen, J., Wilson, J., and Rosen, L. *Nerve Conduction Velocity in Human Limbs with Late Sequelae after Local Cold Injury.* European Journal of Clinical Investigation. Jun. 1996; 26(6): 443-450.

E. Conditioning to improve Raynaud's symptoms:

Jobe, J.B., Sampson, J.B., Roberts, D.E., and Beetham, W.P. *Induced Vasodilation as treatment for Raynaud's Disease*. Annals of Internal Medicine 1982; 97: 706-709.

Hamlett, Murray: *Instructions for Home Treatment of Raynaud's Disease*

Program Description

Content Materials:

- Recollections of Hubert Edward Reeves
- Introduction to Long-Term Sequelae of Cold Injury: Diagnosis and Management
- Compensation Issues for Cold Injured Veterans
- Under Secretary for Health Information Letter, IL 10-96-030
- Under Secretary for Health Information Letter, IL 10-98-008
- Compensation and Pension Examination (AMIE Worksheet #1730) (Cold Injury Protocol Examination)
- Protocol Examination History for Cold Injuries
- Revised Cold Injury Regulation for Disabilities
- Key reference for acute treatment of freezing cold injury
- Key reference on freezing cold injury
- Key reference on non-freezing cold injury
- Journal article summaries of key references on sequelae to cold injuries
- Fact-Sheet – Instructions for Home Treatment of Raynaud's Disease

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Recollections of Hubert Edward (Ed) Reeves on His Experiences with Frostbite in Korea

Introduction

There was no cold weather training for my unit going to North Korea. The 31/7th Infantry Division had winter maneuvers as occupation troops. All Army units were undermanned because of economy moves. At the start of the Korean War the 7th Infantry Division was stripped of all but a few winter trained men to flesh out units going to Pusan Perimeter fighting. Eighty percent of our unit GIs were replacements (like myself) just in from the U.S. (or volunteers–from the stockade) and there was no time for winter training.

At full strength, a U.S. infantry platoon has 44 men. From Inchon to Chosin K/31 Infantry platoons averaged 17 U.S. and 12 to 14 R.O.K. (Republic of Korea) troops, ***but still did the combat load of full strength units.*** R.O.K.s had recently been drafted from Korean refugee camps, farms, at roadblocks, city streets, or the R.O.K. military would enter a business and "draft" the men. R.O.K.s were assigned to us at Mt. Fuji just before the Inchon invasion (The "Buddy-Buddy System"). R.O.K.s were to give us foxhole strength, but received only ***eight days*** training (mostly by copying what a GI buddy did). Then we went into combat.

In mid October 1950, K/31 Infantry was pulled from the combat area northeast of Seoul and went by truck and train to Pusan. We had been in combat wearing the same clothes since the Inchon landing in mid-September. We are quite "ragtag" and got the following "winter" clothing issue aboard a troopship heading up the northeast coast of North Korea on 29 October:

- 1 pair of **drawers, cotton**
- 1 pair **long winter underwear, wool** (These went in my pack. Too itchy to wear until freezing in mountains near Fusen Reservoir.)
- **undershirt, cotton**
- 1 pair **socks, long wool**
- 1 set **fatigues, cotton** (shirt & pants) (already had **fatigue cap with ear flaps**)
- **field pants, cotton** (Somewhat waterproof until you slide and crawl on a few mountainsides)

- **shoepacks, innersole insert** (No change of insert issued)
- **field jacket**, **cotton**
- **pile liner vest**
- **camouflage parka with hood, cotton duck cloth** reversible, white or green
- **gloves, dress leather, with wool insert** (weapon crews received trigger finger mittens)
- **sleeping bag, wool blanket material**, one for each two men. (one man is up on watch) Note: It was a blessing when an R.O.K. unit stole our blanket sleeping bags that were left behind while we attacked a mountain. New **mummy style, down-filled bags** were issued: still one bag for two men.

Battlefield Conditions:

There was growing fatigue among the troops before the Chosin fight because of frequent foot marches through rugged mountains carrying all your gear plus ammunition and boxes of C Rations. At the end of the march you had to dig a two-man foxhole, four feet deep and six feet by three feet wide. Often all you got to eat was a can of cold rations for the evening meal. You were awake all night if there was enemy activity and you spent half of each night on watch if there wasn't any.

On the days when you weren't marching, there were foot patrols for miles through mountains into enemy areas and then you still had to pull night watch.

Foxhole GIs had no way of knowing temperature; had not heard of wind chill. As we went higher into the mountains around **Fusen Reservoir** (northeast of Chosin Reservoir) it got much colder. When in position up on ridge you got more wind. A few days "we got lucky" and had old clay-thatch farm buildings in perimeter. With three men outside in positions with two automatic weapons, the rest could thaw a bit. ***Men outside were relieved every half-hour so they wouldn't freeze.***

The usual shelter was a two-man foxhole. A poncho across the top was anchored on the sides by dirt and snow. This cut the wind and let snow blow over instead of into the hole. A corner left open gave ventilation and a place to stand and watch for the enemy. Bump the poncho and frost growing inside would fall down your neck and feel like a burn.

Extreme cold made leather dress gloves so stiff fingers wouldn't bend. They were worse than no use. Also fingers kept apart would quickly freeze. It's an important fact that you can't load and shoot a rifle with gloves that won't bend. I wore spare wool socks for gloves or hands froze to the rifle metal, ammunition, or anything metal.

From mid-November on, canteens remained frozen except for brief times when we were in patched up farm houses. We melted snow for water and instant coffee *when a fire could be built.* When the company mess tent was in operation, we had to **eat and drink fast before the food froze**. You didn't put lip against metal canteen cup lip or you lost skin. On patrol, we broke through thin ice of waterfalls to get water. **Many times, we just ate snow.**

I have vivid memories of Thanksgiving dinner on 23 November. General MacArthur ordered a complete turkey dinner for every GI in Korea. I came back from patrol at dusk and ate all that rich food. Two hours later I lay on a shack floor feeling like I had triple flu. A medic checked me; no temperature. (I found later I was very allergic to the shrimp cocktail.) I lay there sick till early morning of 26 November.

The Chosin Reservoir

On 26 and 27 November, we did a two-day, non-stop foot march from the Fusen Reservoir area to the Chosin Reservoir. Early in the morning, they woke me; asked if I could carry my pack. I thought so and saddled up. We were to change into shoepacks. We didn't like them because they were clumsy, you could hardly run, and they made your feet sweat. We marched up and down mountains on twisting switchbacks. When a short break was ordered, everyone called for moving on. Standing in place stomping feet, slapping arms against your body, trying to stay warm wasted energy/time. You thought you could be moving closer to "wherever" and hoped for shelter. When waiting, it felt like sand was in your stocking and you knew ice was forming so you would stomp harder.

Extra ammunition was placed on top of already over-loaded packs. Twenty-two pound ammunition cans were passed up and down the line to be carried one in each hand. You carried them as far as you could, then passed them on. R.O.K.s were not used to carrying loads by hand and couldn't carry them very far.

At night, climbing through blowing snow, you kept intervals by reaching forward to tap the man's pack ahead of you. Men fell to the ground and lay there. Medics lifted an eyelid to shine a light in their eye to see if they were passed out or sleeping? Sleepers

were roused and sent walking. Men who had passed out were loaded in the back of a jeep until they came around. Then they walked again. In the dark (or sleep-walking), some men walked off cliffs. If the fall wasn't too far they climbed back to the road by themselves. Others crashed and bounced through rocks and underbrush on a long drop.

My knee and hip joints had growing pain. As hours passed they burned like a hot fire. I counted steps to stay alert, hoped we'd be "there" before I reached another 1,000. I kept forgetting the count and would start over. When the ammo cans were passed up or down, I'd almost go to my knees. I quit switching my rifle between shoulders because that was enough to throw me off balance and out of line (with cliffs at the roadside). At one point, I woke to find myself standing asleep on the road with the column going around me. I knew if I kept going I would walk off a cliff. I went to the uphill roadside, unrolled the sleeping bag, unfolded the poncho over it to keep snow off, and climbed in. I still had enough sense to take the shoepacks off inside the bag. The next thing I knew, it was day of 27 November. I woke at dawn. I listened, not a sound. Snow had drifted over me. Now I remembered that large vehicles often drove part way off the uphill side of the very narrow roads or when passing. None had come. I put on the shoepacks, climbed out, packed the bag, and tried to fold the Poncho. I couldn't fold the frigid material, so I left it and moved up the road at a good pace, rifle at the ready, the only thing moving. I expected CHICOMs (Chinese Communists) at each twist of the road. Fear of being a POW drove me forward. Sometime later, I caught up to the company strung along the road in knots around small fires. (Many doing the North Korean polka; turning because the side away from the fire is freezing.) They greeted me and gave me my can of C ration they'd been ready to divide. (Burnt on the bottom. middle thawed, top still frozen.) We continued climbing. Some hours later came onto a plateau. About two in the afternoon, we came to a 'Y' in the road near a village (Hagaru-ri). 5th Regiment Marine trucks waited beside the road. We were loaded in the open trucks and taken 11 miles up the east road of the reservoir. The frigid air whipped over us freezing us further. Just before dusk we climbed out of the trucks. Marines climbed in and left. We climbed a high ridge above a reservoir inlet. Defenses were a single line of foxholes hacked into the frozen gravel, 35 to 60 feet apart, going up-the ridge. We were told, "Just dig in to get out of the weather. Nothing's out there to be worried about, but keep a 50% watch. Be ready to move out for the Yalu River in the morning."

Ho, my R.O.K. buddy, and I started to thaw a bit in the foxhole's shelter, but still ached too much for sleep. Whistles, bugles, and battle yells alerted us to be ready for the mass of CHICOMs that spilled out of the night. At the same time fierce firefights erupted up and down the ridge and in the valley to our rear. Time was just a string of events. It seemed like hours, then foxholes on both flanks held CHICOMs who traded grenade

throws with us. Popping up to fire, brought automatic fire from both flanks. While I shot in one direction, squads of CHICOMs trotted past on the other side, ignoring us, headed for other objectives. With enemy on all sides, I knew it was time to join the circles of firelight I'd seen in the valley. We rolled out of the back of the hole and worked our way toward friends while mixed in with the enemy attack. We made it to M Company's very small (100 feet) headquarters perimeter and spent the night helping them beat off attacks. A 57th Field Artillery Battalion officer at Pungnyuri Inlet, where we were, recorded –36 degrees Fahrenheit in his firing log that night.

In the night, you have to fight to stay awake, but doze off even while the enemy are charging. You often have to remind your exhausted mind that a larger enemy is a closer enemy. You keep checking to see if a round is in the chamber; you can't remember. When your ammo is gone, you beg from wounded who keep dribbling in looking for help. Your hands no longer can feel your weapon. While the enemy attack out of the smoke and haze, you pull stockings from your hands and push them up opposite sleeves, stealing warmth by rubbing fingers on your forearm. Your fingers are so cold, they make your arm hurt where they touch it. Soon fingers ache and you can feel touch again. You pull hands from sleeves, pull stockings back over hands, pick up your rifle and fire. (Sometimes you had to just jerk at the trigger. You couldn't even feel the M1, but the enemy is a few feet away and there's no time for finger warming). Between attacks you pull off the sock "mittens", insert your hands into the camouflage parka hood and thaw your ears and nose. You try to remember to alternate your breathing between mouth and nose every three times to give them time to warm. You have to continually squirm and kick your feet against something to keep the blood flowing. If it doesn't hurt, you know that part is freezing. You move it, pound on it, or kick with it until it hurts again.

Last Rites, Wounding and Retreat from Chosin

On the night of 27, 28 November, my vision had become a blur. If a dark shape came toward me and threw something, or there were muzzle flashes, I fired. Otherwise, I assumed it could be a GI and held my fire. Someone tapped my arm and asked, "Do you want a drink, son?" I thought, "Great, someone has found water for us." My tongue was swollen, cracked and raw. It tasted like burnt copper. I took a bottle in both stocking-mittened hands and took three big cold as ice swallows before I choked. The figure beside me retrieved the bottle before I could drop it. It was whiskey. I was so far gone, I didn't even think that you couldn't carry water in a glass bottle. I tried to get my breathing going while the figure beside me held a hand on me and talked in a foreign language. It was the chaplain giving me communion and last rites. He figured we all would soon be dead. In a few minutes, I not only had my breath going, but my vision cleared and I could see, shoot

and hit the enemy.

I was wounded the first time not long after dawn on 28 November. A CHICOM anti-tank shell hit my legs, flew high, and landed hard. I was dragged through gunfire and into a room of the farmhouse command post already full of wounded. I huddled in a corner between two shattered doors and watched the fighting outside through both. I slid into sleep often, but was not out long. (Maybe I passed out from loss of blood or from the effects of concussion or cold?) There were no medical supplies left. The wounded just had to tough out the pain. I wondered how much blood you can lose before you died, but after awhile the cold stopped the bleeding from the many wounds in my legs and feet.

Wounded men patched up other wounded by plugging holes with and bandaging with cloth torn from underclothes. Wounded who were somewhat mobile volunteered to fight and were helped out to vacant positions and given a weapon and some ammo. Some returned to have new holes plugged, then answered the next call when another attack built. Men whose wounds would have put them in hospital ICU units in the U.S. fought on because they could still move and fire a weapon. Walking wounded volunteered to fight their way to the frozen reservoir, chop a hole in the ice and bring back water for the wounded. They headed into "No Man's Land" with wooden buckets. None came back.

Eventually, we had too many wounded to get them all under shelter. When a man died, he was carried out and immediately replaced by a wounded man.

Late on the morning of 29 November, the 1st Battalion 32nd Infantry fought four miles down the eastern shore road and into the 3rd Battalion 31st Infantry perimeter. They had been under continuous attack since late on 27 November. On the trucks carrying their wounded was a bit of food. Medics came around and each wounded man got an individual C-ration sized can of lukewarm black tea dipped from a wooden bucket. I asked for more, but told that was my ration. A small box of bran flakes was laid on my chest. I ate it one flake at a time; letting it melt in my mouth. That was my last "meal and drink" till the afternoon of 5 December.

On 1 December at 1:00 p.m., **Task Force Faith Breakout** (the retreat from the Chosin Reservoir) began. It was decided that the walking wounded would continue to fight. (The number of wounds did not matter. If you could walk, carry a weapon and fire it, you fought.) More seriously wounded would ride. (Lt. Colonel Olin Beall 1MTB 1st Marine Division has stated there must have been over 600 wounded on the trucks.) They pulled my legs straight and I was placed in a mummy sleeping bag. Loaded onto the bed of an open 2 and 1/2 ton truck, we lay on parachute silks used to keep us off the steel bed.

Above us another layer of wounded was on stretchers and boards held up by the side bench seats. Other wounded sat on the benches, up in the wind, with their legs between the boards and stretcher handles. Everyone on the truck was wounded: the driver, the man riding beside him as "shotgun" with an M1, and the man with a shattered/splinted leg sitting on the tailgate with a carbine.

As soon as the attack south started, CHICOM fire grew intense. There was a steady chunk-chunk of rounds hitting the truck. When a man was re-wounded others helped plug him up. Walking wounded fought alongside the trucks, holding the enemy back. The column moved in fits and starts. Roadblocks had to be broken. Hills, rugged and high, on the flanks had to be taken. Bypasses around blown bridges were built under fire. It hurt a lot when the truck had to drive over tree stumps in the fields. Every few hundred yards, the driver would be killed. At times a walking wounded man on the road would take over. At other times the call came back, "Need a driver!" A wounded volunteer was helped forward and started driving. When the truck ran into the ditch, walking wounded reopened wounds pushing to get the truck on the road. Snow on the road turned dirty pink where enemy machine gun fire was extra heavy. All the time, wounded who were conscious on the trucks squirmed and moved to keep blood flowing, even though this caused pain. A group of walking wounded divided their ammo, three rounds apiece, and attacked CHICOMs who were on a hillside pouring fire into the moving column.

Executions and POWs

The column was stopped as it came to roadblocks entering the narrow road through Hudong-ni. The task force had run out of everything. Many vehicles were too shot up to run, were out of gas, or had no one to drive. Walking wounded were searching dead and wounded along the road for ammunition. No manpower was left to break roadblocks or take and hold flanking hills. Air cover had left at dusk. The word was passed, "Everyone who could, should leave the column, get on the ice and head for the Marines at Hagaru-ri." Some were crawling, some held onto each other, some were dragging men in sleeping bags.

With nothing to hold them back, CHICOMs overran the trucks. Poking with bayonets, they made some who hadn't left and could walk march away under guard. Before long they started burning trucks with the wounded in them. Our truck's fuel had drained out through bullet holes, so they shot us: one shot in each head. I sat in my bag watching men shot, right up the line to me. At my turn I said, "Here I come, Jesus." The muzzle blast knocked me flat. Surprised I wasn't in eternity with Jesus, I waited for the CHICOM to fire again. Instead, he climbed down from the side of the truck. They left, I slid down in the

bag and held still. I'd only received a scalp wound. Two others could be heard breathing for quite awhile.

From 2 to 4 December, I stayed with the truck and played dead when the enemy searched the dead for combat boots. They didn't want the shoe packs they felt through my sleeping bag. Many times at night I tried to leave the bag and head for the reservoir ice and Marines at Hagaru-ri. Half out of the bag I would start to faint, slide back in the bag, yank up the zipper, and pass out. Awakening I'd ask God why I was alive, when everyone else was dead. I asked Him to get me off the truck, I couldn't do it. When awake I'd keep squirming to keep blood moving, even though it hurt the wounds. I had a terrible thirst. I found a canteen in the snow beside the bag. I took it in the bag to thaw, waited hours. It didn't thaw, so I pushed the cold lump back out of the bag. In the dark, I'd sneak hands full of snow from the drifts around me, squeeze it into a ball, chew it into slush and swallow. I urinated carefully, outside bag. It was the first time I'd done that since before the march to Chosin Reservoir, there was not much, and it was very dark – blood from concussion of shell? I covered it with fresh snow so the CHICOMs wouldn't see it.

On 4 December, the CHICOMs came back searching bodies for loot, then threw the dead into piles on the road. They opened my bag. I held myself stiff like a frozen body, but they felt warmth when they opened layers of clothes to get in all the pockets. I was a live enemy, so they worked me over, then threw me on top a pile of dead in the road. When they picked up their rifles I expected a shot to the head and again said, "Here I come, Jesus." They used rifle butts on my head for this attempt. I tried to protect my head with my hands, but breaking fingers hurt more than the head. I put the hands down and thought, "Roll your head with the blows." That didn't work because these three had no rhythm. After a time of seeing flashes and hearing loud noises inside my head, a CHICOM barked an order. Another lifted my head by the hair to look at my face. I ordered myself, "Stare up the road. Don't blink. or breathe. Keep everything relaxed." He dropped the head. Through a red haze I watched them leave with their loot.

Escape

Although there were many other CHICOMs on surrounding hills, after my "executioners" had gone, I slid from the pile and tried to put my clothing together. Outer layers were open after I had been searched. Broken fingers couldn't do zippers, snaps and buttons. ***"Move out! Don't sit in the middle of their road,"*** I told myself. I crawled on elbows and knees because the hands hurt too much to use. The fields were crossed and ice reached just before dark. I crawled on the ice, counting Jody Cadence and singing childhood Sunday School songs to set a pace and stay awake. I often slipped and banged my face on

the ice. At one point, falling more than crawling, I thought I'd better rest before I fainted and froze. I rolled up in a ball with my back to the wind. Drifting snow would help warm me. I wondered how frozen the hands were and bumped fingers against my teeth. They made a hard clicking sound. I knew they were probably gone, but I would save all I could. l crossed my arms and tucked hands under my armpits inside the loose field jacket. I was drifting into "warm" sleep when a CHICOM showed up. As he came up, I figured he'd shoot me with his submachine gun. Disgusted that days of pain and effort would end this way, I threw out my hands and yelled, "Ah no!" The CHICOM spun around and ran from me. Now more alert, I continued on.

Rescue

In the morning, three and a half miles (Army estimate in the records) from where I'd started, I was at the far side of the ice, crawling slowly south near the shoreline. I met another wounded GI just before Marine fighter planes came. They made a stack right over us, dove and flew past very low to attack the east shore. The GI started writing Help in the snow. A plane saw it. They circled around us protecting us from CHICOMs curious about this activity. Marines Lt. Colonel Olin Beall and PFC Ralph Milton were out with a patrol of Marine volunteers on another of their many rescues of wounded from far into CHICOM areas. About 1:00 a.m. they picked us up in a Jeep and took us right up to a plane at the Hagaru-ri airstrip.

Medical Treatment

On the flight from Chosin to Yonpo, a flight nurse gave me my first medical treatment, a shot for pain and a drink of water. In an aid tent beside Yonpo airstrip, medics cut off my clothes while a wounded Marine in the next stretcher, George Graham, spooned hot C ration soup and strong coffee into me. I was given another pain shot, then they plugged holes and bandaged my head, hands and legs. I was put on the next flight out and was asleep as we took off. I woke on an operating table at an unknown MASH unit. They'd bathed me and were digging out shrapnel and probing wounds. My head had been shaved and a nurse scrubbed my skull with green soap while another held scalp flaps back. I went to sleep while they sewed scalp pieces together.

The next time I awoke, I was on a flight to Tokyo. There, without checking the medical records sticking out from under my pillow, a doctor flipped the blanket back and asked if I'd been wounded. I thought red and white bandages should have clued him and that he was not very apt. I told him, "No Sir, I tried to commit suicide and botched it up." I refused to be put in an examining room where men were coming out with a blanket over

their head. I insisted on going upstairs for treatment. Upstairs, was a very small 'private room.' A large syringe of morphine was kept on the bedside table. Whenever I woke and moaned from pain, a Japanese nurse rushed in and gave me a measured dose.

Full of morphine, I refused food and just wanted painless rest in the nice white, warm bed. A doctor in the hallway told other doctors not to waste time checking on me, I was dying. Angry, I rebelled the only way I could; ate three trays of food. Now doctors came and looked under the bandages and did medical thumping/listening. Feet, fingers, knees and ears were turning various dark colors. The same doctor who'd said I was dying now wanted to amputate above both knees and both wrists "to get most of the gangrene." Not trusting his views of my survivability, I declined (forcefully). Everything was re-bandaged. The ranking doctor asked why there were no head X-rays for missiles inside. The doctor who wanted to amputate said he thought it a waste of time in this case. The ranking doctor said, "X-ray. Now."

With no missiles found inside my head; I was moved to a room with two other "maybe they'll make it" patients. On 12 December, I got in the pipeline to a U.S. hospital for treatment. On all flights, I was kept fairly well sedated and slept a lot. I was flown from Tokyo to Wake Island and then to Tripler Hospital, Hawaii, (checkup and a few hours rest). From there, I went to California, where I was transferred to a plane to Texas. I was in the hospital overnight where the head nurse almost killed me making sure I had a bowel movement.

Then I was sent on to the Percy Jones Army Hospital in Battle Creek, Michigan, where I found myself in another very small private room. This time morphine or codeine max dose was given every four hours. When neither drug worked, they give me alcohol/saline intravenously for a few days to clear my system, then it was back on pain drugs. The surgery chief told my mother by phone they must amputate my feet and fingers, or gangrene would kill me. He said the operations probably would too, but there was no choice. Said, even if I did live my life was over. I would be bedbound for life. I was probably already addicted; if not, would be on strong drugs the rest of my life to stop pain. "And, he'll probably stay drunk to forget Korea. Stay home and remember your son the way he was, his life is over." My parents didn't listen to the colonel but came to the hospital.

In January and February 1951, both feet and all my fingers were amputated and then work began at clearing infected wounds. I was finally moved to a ward with other men. In March 1951, I was flown to Valley Forge Hospital for hand and knee skin grafts and surgery. In November 1951, I was retired by the Army and transferred to Hines VA Hospital. Frostbitten infected bone was removed from my left knee and the left hand palm

bones were shortened because they were sticking through skin grafts. My left leg stump was revised. After all wounds healed, I got prosthetic feet and took walking classes.

In the summer of 1953, I left Hines VA Hospital.

Continuing Effects

The following are the continuing effects from my cold injuries.

1. Sensitivity to cold.
    a. Hands, condyles at the wrists, below the knee, feet and fingers (although feet and fingers are gone, damaged nerves above the amputations respond), knees, ears, scalp wounds (blood froze on scalp) – all these ache when temperatures drop into the 40's. The colder it is, the greater the ache. (This is the main reason for the move from Illinois to Arizona.) *Even cold water from a faucet makes hands ache.*
    b. It takes a long time to warm up frostbitten parts and stop the ache after they are cold. The longer in the cold and the colder it is, the longer (sometimes hours) it takes to warm up.
    c. The nerves in frostbitten parts are very sensitive to barometric pressure changes. Sometimes, I feel like I'm stepping on nails that pierce through the foot, or that toes or shin bones are breaking and fingers are being bent backwards. I'm very thankful this is not all the time!
    d. All these sensitivities have become stronger over the years.
    e. Slightly cold ears felt like they have been burned. External left ear (most frozen of two) often aches if I sleep on that side.
2. Ghost feelings in affected parts
    a. The waking up "tingle" sensation of past years has become much stronger. It often feels like toes are standing on an electric wire. (Sometimes the feeling is stronger than at other times.) Aspirin helps. I eat a lot of aspirin. My wife says, "You're dancing with your feet again, do you need aspirin?" I feel like I am standing in cold water up to my knees.
    b. Hands itch often. Rubbing brings temporary relief.
3. Skin infection and cracking – Skin and scars of frostbitten areas are dry and crack if

not kept well lubricated. These areas are also prone to rashes and infections, and take extra care time to heal.

4. Allergic reactions – Working with computer paper or any acidic substance causes frostbitten skin to break out in a rash and crack open.

5. Frostbitten knees – Prosthesis must be made to avoid pressure or rubbing at this area. Scars and damaged skin break down very easily. Strong knee hinges, reinforce damaged joints. (Today, most BK legs have no lacer with hinges, but use a knee strap or roll up sleeve.)

Summary

Everything considered, this "*height challenged*" (How's that for political correctness?) Army-retired Private has had a blessed, exciting, fulfilling life to this point, and expects it to continue.

Ed Reeves enlisted in the Infantry in 1949, at age 17. He had completed eight years of schooling at the time, but eventually qualified for OCS. Before he was old enough to attend, he volunteered to go to Korea and at age 19 was severely wounded at the Chosin Reservoir in December 1950. He married Beverly Jean Hall in September 1951 while on medical leave from an Army Hospital. (They have eight children and fifteen grandchildren) Following his discharge from Hines VA Hospital, he earned his B.S. degree from University of Illinois at Urbana (1958) and was employed by the DoD as a systems analyst until 1971, when he resigned to devote himself to full-time ministry. He has served as a missionary in North, Central and South America as well as Russia.

Introduction to "Long-Term Sequelae of Cold Injury: Diagnosis & Management"

Susan H. Mather, MD, MPH
Chief Public Health and Environmental Hazards Officer, Veterans Health Administration

"It was cold, cold, cold – below zero much of the time," recalls Eugene R. Smith, reminiscing about the Battle of the Bulge. He told us that snow was over his knees and the ground was too frozen to dig a foxhole. He was an Army squad leader who saw his 14 men turn over twice from cold injury. He thinks the cold did more damage at the Bulge than gunshots did. William J. Flynn, another veteran of the Battle of the Bulge, recalls that he fought with no combat boots, only ankle type shoes, summer socks, OD pants and shirts, winter underwear and a type of pea coat. While it is possible to provide the kind of clothing and gear, which protect against extreme cold, this often proves difficult in combat situations. Ernie Pappenheimer, a Marine veteran of the Battle of the Chosin Reservoir in Korea, recalls that they were provided with cold weather sleeping bags, but the zipper in the inner lining often froze so that they couldn't get out of them. Some of the men were even caught in the cocoon bags by the enemy, bayonetted, and killed. Edward R. Beltram, another Marine at Chosin, abandoned his sleeping bag for blankets, which didn't offer as much protection from the cold, but could be flipped off quickly.

In addition to physical protection, adequate nutrition and hydration are important in protecting against cold injury. However, these basics are not easy to provide when fighting under frigid conditions. William Flynn says that during the Battle of the Bulge, he and his men existed on cheese and chocolate bars; a diet, which was constipating to some while others complained of dysentary. Ed Beltram recalls that all the food at the Chosin Reservoir was frozen so they ate sweets, particularly Tootsie Rolls, which led to their nick-name, the "Tootsie Roll Marines." He said they ate crackers with snow and sugar on them, instant cocoa, candy, cakes and sugar packs, adding that out in the flanks and on the point, it was difficult to warm anything up. Donald F. McAllister, an Army veteran of the same campaign, remembers chipping frozen food out of the cans with his bayonnet. Water froze in the canteens. Ernie Pappenheimer says that water intake was limited to melting snow in your mouth. They also took condensed milk, coffee or cocoa and made a slush.

However, Don McAllister believes the extreme cold had at least one positive effect for him. He was wounded, and feels that he may have bled to death without medical attention had the flesh around the wounds not frozen. Ernie Pappenheimer recalls that his unit was grateful to have a "big mouthed corpsman," who kept the syninges of morphine from freezing by putting them in his mouth. However, when Ernie was wounded, he was evacuated by an observation helicopter with his feet sticking out in the cold air, thus aggravating his

cold injuries. Not everyone got much immediate medical attention. Ed Beltram was evacuated from Korea on a hospital ship, but received no medical attention until he passed out in the chow line, and they discovered he had a head wound as well as a wounded foot. He ended up in a hospital in Japan.

In the history of warfare, cold has always been a fearsome enemy. Washington fought cold, hunger, and disease in Valley Forge during the winter of 1777 and 1778, and of the 11,000 men there, over 3,000 died without firing a shot.

In Europe, Napoleon's ultimate defeat was sealed during the long retreat from Moscow in 1812, when his solders battled snowstorms and terrible cold. Of the 600,000 men in his forces, 500,000 were lost.

More recently, during World War II, the Battle of the Bulge was fought under conditions of extreme cold in December 1944, when Hitler's forces staged a last stand onslaught in the Ardennes Forest. By January 1945, when the Allies had recovered ground lost earlier in the battle, both sides had experienced severe casualties.

Five years later, during the Korean War, Thanksgiving 1950 found 20,000 American Marines and Infantry men at the Chosin Reservoir greatly outnumbered by Chinese who had poured across the border into North Korea. During the retreat from Chosin, roughly a mile high, the savage cold temperatures, by modern chill factors, at times reached -100° Fahrenheit and possibly lower.

In preparing for this program on cold injury we talked to Ed Reeves and asked him to contribute his very compelling story to this project. You can find his story on page 1 of the booklet.

Other veterans also shared their stories with us, stories of extreme hardship in battle and long struggles with the residuals of cold injury long after the battles. Some tried to ignore their cold injuries, or were forced to ignore them in the beginning as more seriously wounded were cared for. In this extreme and chaotic condition at Chosin Reservoir, many soldiers never sought care, or if they did, their medical records were lost so no documentation exists of cold injury suffered at that time. Over time, they adapted to their injuries by moving south, abandoning outside activities in the winter, or wearing double socks. The lack of documentation of their injuries made it difficult to get compensation from VA.

Research has shown that some of the effects from extensive cold injuries, even those believed healed after exposure with asymptomatic period, may be irreversible lifetime wounds that progressively worsen with age. Cold injury may result in tissue damage, loss of function, neurocirculatory loss, amputation, and even death. Ill-equipped U.S. service personnel in both Korea and World War II suffered varying types and degrees of both non-freezing and freezing cold injuries.

Both non-freezing and freezing cold injuries may result in neurologic and vascular injuries, as well as changes in muscle, skin, nails, ligaments, and bones. Neurologic injury is manifested by bouts of pain in the extremities, hot or cold tingling paraesthesia, conduction velocity decrements and numbness, that may remain throughout one's life. Vascular injury shows up as Raynaud's like phenomenon, white or cyanotic when cold, failure to dilate after mild, alcohol-spray cooling, and slowed rewarming once a periphery has become cold.

This continuing education program on cold injury is based on a teleconference sponsored by the Department of Veterans Affairs June 12, 1997. A videotape of that conference and selected print materials were published as an independent study program in May 1998. Subsequently the independent study was revised so that the material on the videotape could be incorporated in print format, which will stand alone.

Compensation Issues for Cold Injured Veterans

Caroll McBrine, MD
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Introduction

Korean War as well as World War II veterans and veterans who experienced cold injury as a result of accidental exposure seek assistance for two reasons. The first is for treatment of their clinical problems. The second is for assistance in pursuing a claim for the compensation of resulting disabilities. The Veterans Benefits Administration has taken a number of steps to assist veterans in the claims process and to enhance the adjudication of claims for residuals of cold injury. They include:

- the revision and expansion of the evaluation criteria for residuals of cold injury in the rating schedule (Part 4 of Title 38 of the Code of Federal Regulations) (see page 41),
- the addition of a section on cold injury in the adjudication procedures manual, M21-1, part 6, and
- the development of a cold injury examination protocol (see page 26).

Examination Protocol

The detailed history and comprehensive physical examination that constitute the protocol examination will ensure that the medical problems of each individual who seeks compensation for residuals of cold injury are documented so that they can be addressed both for clinical treatment purposes and for the determination of disability compensation. It will also provide a database that can be used by clinical researchers and by historians.

Revision of the Rating Schedule

The rating schedule contains a list of disabilities with evaluation criteria to guide their evaluation for purposes of disability compensation. It has included a diagnostic code and evaluation criteria for frozen feet since 1933. The original criteria were revised in 1945 and remained unchanged until the current criteria were introduced. Under the largely subjective 1945 criteria, a 10% evaluation was assigned for mild symptoms or chilblains,

whether present in one foot or in both feet. A 20% (or 30% for both feet) evaluation was assigned for persistent moderate swelling, tenderness, redness, etc. A 30% (or 50% for both feet) evaluation was assigned for loss of toes or parts, and persistent severe symptoms.

First, the criteria have been updated in several ways. Diagnostic code 7122 in the rating schedule was formerly titled "frozen feet, residuals of." The scope of the evaluation under diagnostic code 7122 has been broadened to include cold injury of any part of the body. Next, instead of a single 10% evaluation being assigned whether there are mild symptoms of either one foot or both feet, each foot, as well as any other affected part of the body must now be separately evaluated, and those evaluations combined. The bilateral factor will be applied, if appropriate, when paired extremities are affected. Therefore, some evaluations may be higher under the new criteria than under the cold criteria, for the same disability. In addition, these revisions allow for the individual assessment of each foot when the feet are not affected equally, a determination which was difficult under the old criteria, because the feet were evaluated as a pair, at each level of evaluation.

In order to assure consistent evaluations, subjective terms in the evaluation criteria, such as "mild" and "severe," have been eliminated and replaced, to the extent possible, with more objective criteria. A 10% evaluation now calls for pain, numbness, cold sensitivity, or arthralgia; a 20% evaluation calls for any of those symptoms plus tissue loss, nail abnormalities, color changes, locally impaired sensation, hyperhidrosis, or X-ray abnormalities; and a 30% evaluation calls for criteria identical to those for a 20% evaluation except that 2, rather than 1, of the objective findings of tissue loss, nail abnormalities, etc., are required. A note in the schedule provides an expanded list of some of the complications that may warrant additional separate evaluations, such as amputations, squamous cell carcinoma at the site of a cold injury scar, and peripheral neuropathy.

The adjudication manual (M21-1)

The manual provides guidance on adjudication procedures. It contains three paragraphs on residuals of cold injury during the Korean War, one on possible residuals and complications of cold exposure, one on a description of acute cold injury, and one on the Chosin Reservoir campaign. This material is largely based on three training letters issued by the Veterans Benefits Administration between 1993 and 1995. The manual emphasizes that the circumstances of service must be considered when a veteran has a disability found to be due to cold injury. For example, when the fact of participation in the Chosin Reservoir campaign is established, exposure to extreme cold can be conceded, despite the lack of any specific record of a cold injury. While the circumstances of service are always, by statute, for consideration, they are especially important when a determination regarding

service connection for the residuals of cold injury must be made, because it is common for the service medical records to contain no information regarding a cold injury. And discharge examinations commonly show no residuals of cold injury – because the acute injury has healed, and late residuals have not yet appeared. Despite the lack of evidence of cold injury in the service medical records or on the discharge physical examination, knowledge of the circumstances in which the veteran served may allow VA to establish service connection for residuals of cold injury that are diagnosed long after service.

Summary

VA wants to assure that the claims of veterans for residuals of cold injury are adjudicated as fairly and accurately as possible. Providing more information to VA personnel about the effects of cold injuries and the circumstances of their occurrence is an important first step. Furnishing better documentation of the residuals of cold injury and establishing expanded, updated, and more objective evaluation criteria will also help. And, with a better understanding of the symptoms associated with post-cold injury sequelae, veterans can and will be treated with high-quality examinations, follow-up, and compassionate care.

Under Secretary for Health Information Letter – IL 10-96-030

Recommendations for the Care and Examination of Veterans with Late Effects of Cold Injuries

In Reply Refer To: 13
*December 31, 1996

1. During World War II and the Korean War, thousands of United States service personnel suffered cold injuries including frostbite; i.e., Freezing Cold Injury (FCI) and immersion foot; i.e., Non-freezing Cold Injury (NFCI). Veterans of the Battle of the Chosin Reservoir in Korea are recognized as having suffered especially high rates of severe cold injuries (i.e., FCI). In many instances service members did not seek or were unable to obtain medical care after cold injuries because of battlefield conditions, and documentation may never have been made in their medical records, or may no longer be available.

3. It is important for Department of Veterans Affairs (VA) staff examining and caring for veterans who have experienced cold injuries to be familiar with the recognized long-term and delayed sequelae, including peripheral neuropathy, skin cancer in frostbite scars (which may occur in such locations as the heels and earlobes), arthritis in involved areas, chronic tinea pedis, fallen arches and stiff toes, nocturnal pain, and cold sensitization. These veterans' cold-related problems may worsen as they grow older and develop complicating conditions such as diabetes and peripheral vascular disease which place them at higher risk for late amputations. It needs to be acknowledged that there has only been limited research into delayed effects following cold injuries and many unanswered questions exist (such as whether there are long term or delayed effects on internal organs of individuals surviving acute cold injury, and the diagnosis of the subtle sequelae of NFCI which can also be very debilitating).

4. Veterans suffering from long-term and delayed effects of cold injuries frequently require continuing medical care as well as specialty consultations and periodic reevaluations. Enrollment of these veterans in VA Primary Care programs therefore is encouraged. These veterans should also be encouraged to see a Veterans Benefits Counselor to discuss submission of a benefits claim, if appropriate.

5. Recommendations for inclusion into the medical history of cold-injured veterans are:

    a. A description of the acute cold injury; e.g., circumstances (ambient temperature, length of exposure, wet and/or dry conditions), signs, symptoms, diagnosis made, treatment provided. Individuals at higher risk for cold injuries include riflemen of lower ranks, African-Americans, those who were poorly nourished, and those

having concomitant injuries (see Barber, "Cold Injuries in the Military," Medical Bulletin of the U.S Army, Europe, 1980, pp. 22-27).

b. A description of signs and symptoms in the interval following the acute cold injury; e.g., amputations or other tissue loss, cold sensitization, Raynaud's phenomenon, hyperhidrosis and/or excessive sweating, sensory neuropathy and/or disturbances of sensation; chronic pain resembling causalgia and/or reflex sympathetic dystrophy, weakness and/or reduced strength, sleep disturbances, recurrent fungal infections, breakdown and/or ulcerations of scars, disturbances of nail growth, skin cancer in chronic ulcers or scars, arthritis and/or joint stiffness and/or loss of range of motion in the affected limbs, reduction in mobility, swelling, pain and/or paresthesia and/or numbness, changes in skin color, skin thickness, intermittent blisters and scaling of skin to the knee.

c. Medical treatments and other measures; e.g., having to use multiple pairs of socks, sleeping with socks on, moving to a warmer climate, etc.

d. Current symptoms and other subjective data. Ask about pain (including location, intensity, nocturnal pain, sleep disturbances), cold feelings (including differences between winter and summer), numbness, excessive sweating, tingling, and burning sensations.

e. Information about smoking; current medications; and other diseases such as diabetes, hypertension, etc.

6. Recommendations for objective data to be obtained as part of the physical examination of cold-injured veterans include:

a. Blood pressure. In addition to arm blood pressure, blood pressure in the leg and the ratio of ankle blood pressure and/or brachial blood pressure may be useful in evaluating for occlusive arterial disease of the lower extremity (see Isselbacher et al., Harrison's Principles of Internal Medicine,. 13th edition, 1994, p. 1136).

b. Examination for loss of body parts and/or tissue and amputations.

c. Examination of the extremities for edema, color changes, and peripheral pulses.

d. Examination of the skin for temperature, color, hair, sweating during examination, scars, ulcerations, fungal infections, skin cancer, and nail changes including onychomycosis.

e. Neurological examination for decreased strength, reflexes, pin-prick, temperature sensation discrimination, and light touch.

f. Joint examination for signs of arthritis and loss of range of motion.

7. Objective data from other types of testing include:

a. Additional testing if clinically indicated such as blood sugar, X-rays of joints, nerve conduction velocities, and studies of peripheral circulation, e.g., ultrasound Doppler.

b. Specialized testing using Infra-Red Thermography, if available – Temperature gradients from ankle to toes and re-warming rate and pattern after alcohol-spray cooling are significantly modified. NOTE: Laser Doppler testing for measuring blood flow through small vessels appears to be primarily a research technique at the present time.

c. Specialty consultations if clinically indicated including Podiatry, Neurology, Dermatology, and Rheumatology;

8. Treatment recommendations include-

a. Enrollment in primary care programs.

b. Foot care including involvement of podiatry for patients felt to be at high risk for foot complications.

c. Smoking cessation.

d. Control of lipid disorders.

e. Weight control.

f. Control of hypertension.

g. Control of pain including effective nocturnal analgesics; NOTE: Consider use of Capsaicin (Zostrix) topical analgesic cream, topical 50 percent dimethyl sulfoxide (DMSO), tricyclic antidepressants and low-dose anticonvulsants:

h. General preventive medicine measures recommended for the patient’s age group; e.g., immunizations against influenza and pneumococcus.

9. The following individuals with special interest and expertise in cold injuries may be contacted for additional information and assistance:

a. Dr. Cameron C. Bangs
Bangs Professional Building
728 Motalla Ave.
Oregon City, OR 97045
503-655-5177; FAX 503-655-9397

b. Dr. John A. Boswick, Jr.
2005 Franklin St., Suite 355
Denver, CO 80205
303-839-1694; FAX 303-839-1695

c. Dr. Ben Eisman
Vascular Surgery and/or Dept of Surgery
University of Colorado
Campus Box C312, Rm 5521
4900 E. 9th Street
Denver, CO 80262
303-399-8020 ext 2982

d. Maj. W. Bryan Gamble
Plastic Surgeon
U.S. Army Aeromedical Center
Building 301 Andrew Street
Ft. Rucker, AL 6362-5333
343-255-7363

e. Dr. Murray Hamlet
U.S. Army Research Institute of
Environmental Medicine
Natick, MA 01760-5007
508-233-4865; FAX 508-233-5298

f. Dr. Frank Hubbel
RFD #1, Box 163
Conway, NH 03818-3150
603-447-6710; FAX 603-447-2310

g. Cdr. Percival D. McCormack
Thermal Stress Division Code 053
Naval Medical Research Institute
Bethesda, MD 20889-5607
301-295-0777; FAX 301-295-2720

h. Dr. William Mills
742 K Street
Anchorage, AK 99501
907-272-0194

i. Dr. Bruce C. Paton
5380 East Mansfield Ave.
Denver, CO 80237
303-692-8225

10. Administrative questions may be addressed to the Office of Public Health and Environmental Hazards (13), VA Central Office, telephone 202-233-8575, FAX 202-273-9080. VA Central Office is planning to issue additional guidance relating to cold injuries including revisions to the Veterans Benefits Administration (VBA) procedure manual and rating schedule, Physician's Guide for Disability Evaluation Examinations section and Automated Medical Information Exchange (AMIE) worksheet and a new Compensation and Pension (C&P) examination protocol.

11. See Attachment A for additional information on NFCls and FCls provided by Dr. Murray Hamlet, U.S. Army Research Institute of Environmental Medicine.

12. Selected references are listed as follows:

a. Altman, M.L., and Hutton, S.J., "Late Neuropathic Sequelae of Cold Injury," Journal of Foot Surgery, 1987; 26:213-216.

b. Blair, J.R., Schatzki, R., and Orr, K.D., "Sequelae to Cold Injuries in One Hundred Patients," Journal of the American Medical Association, 1957; 163:1203-08.

c. Home, M.J. Ellen, "Post-Frostbite Sequelae, Clinical and Experimental Study," Report No. 130, Army Medical Research Laboratory, Ft. Knox, KY, December 1953, pages 1-31.d. Kaplan, R.P., "Cancer Complicating Chronic Ulcerative and Scarifying Mucocutaneous Disorders," Advances in Dermatology, 1987; 2:19-46.

e. McKendry, R.J.R., "Frostbite Arthritis," Canadian Medical Association Journal, 198 1; 125:11281130.

f. Purdue, Gary and Hunt, John, "Cold Injury: A Collective Review," Journal of Burn Care and Rehabilitation, 1986; 7:331-342.

Attachment A – Summary of Cold Injury Sequelae – IL10-96-030

1. Non-Freezing Cold Injuries, Immersion Injuries, Trench Foot, Pernio. Produces severe postinjury sequelae, depending on severity of the original injury. Multiple chilblain may produce some cold sensitivity, tingling and paraesthesia.

    a. Pernio (Superficial necrotic plaques on the dorsum of the hands or feet). Produces residual pain, Raynaud's Syndrome, cold or hot feet and hands. Often, healed skin appears normal or blotchy red on cooling.

    b. Trench Foot

        1. Necrosis of Tissue. May lose toes or fingers. Residual pain, hot or cold extremities, Raynaud's, and cold sensitivity, bone and joint pathology (arthritis), muscle wasting and chronic weakness.

        2. Nerve Damage. There may be numbness or pain, stabbing and/or shootings in the feet, ankles and legs, conduction velocity decrements which extend proximal to the lesions, sensitivity to cold with warm skin.

        3. Vesicle Blisters. Multiple and recurring formations that lead to scaling and last for a life-time. Blisters may lead to ulcerations; slow to heal. Ichthyosis-like scaling extending up the calf, usually seasonal in the winter.

        4. Fungal Infections (Lifetime Injury). Thickening of nails and nail beds; irregular shape, hard, curled-edge nails become tube-like; extensive undermining by the fungus. Ingrown great toe is almost universal.

        5. Hyperhydrosis. Enhances the fungal infections; often a history of rotted shoes and socks.

        6. Flexion Contractures. Usually early, but can occur late in the injury.

        7. Ulcerations. Tips of toes, pressure points on foot. Very painful and slow to heal; may recur.

        8. Thick, Fissured Calluses. Heels, medial and lateral side of the ball of the foot; not from ill-fitting shoes.

        9. Gait Modifications. Widened stance, shuffling gait, short steps. Loss of proprioception leads to tendency to walk on heels, not balls of feet. Standing produces great leg and foot cramps.

2. Freezing Cold Injuries

    a. Mild. Cold blanched extremities. Painful re-warming. No blister formation. Red and tender on rewarming. No residual injury. May be sensitive to the cold.

    b. Moderate. Blisters on recovery. Clear, fluid-filled blisters extend to the base of the nails. Bluegray color.

    c. Severe (Frostbite). Hemorrhagic blisters. Ulcerations. Mummification and acute amputation of shell-like portions of the digits or whole digits. Long hospitalization. Painful on standing. Some proximal muscle loss.

    d. Skin

        1. Scaling. Recurring blisters followed by scaling and ulceration. Pain. Constant aching or stabbing, sharp pains in the hands, feet and calves.

        2. Hot and cold sensitivity and numbness.

        3. Blotchy red or brown. Can extend up the legs with age.

        4. Hyperhydrosis. Leads to chronic, long-term paronychial fungal infections.

        5. Thick, hard-curled nails, heavily undermined with fungus.

    e. Muscle. Loss of mass, weakness, cramps after use.

3. Found in Both Non-Freezing and Freezing Cold Injuries

    a. Neurologic Injury. Pain in extremities. Hot or cold tingling paraesthesia. Conduction velocity decrements and numbness. May have numbness throughout their life, interspersed with bouts of pain,

    b. Vascular Injury. Raynaud's. White or cyanotic when cold. Failure to dilate after mild, alcohol spray cooling. Constant cold feet and hands.

S/ by Mike Hughes for
Kenneth W. Kizer, M.D., M.P.H.
Under Secretary for Health

Attachment

DISTRIBUTION: CO: E-mailed 12/31/96
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Under Secretary for Health Information Letter – IL 10-98-008
Cold Injury Examination Protocol

In Reply Refer To: 13
April 20, 1998

1. This Information Letter distributes the Department of Veterans Affairs (VA) Cold Injury Protocol Examination and History.

2. Background

   a. Thousands of United States (U.S.) service personnel have suffered from frostbite and trench foot including but not limited to veterans of the Battle of the Bulge in World War II and the Battle of the Chosin Reservoir in Korea. VA is committed to providing thorough examinations and high quality medical care to veterans with long-term and late sequelae of cold injuries. Recently, new VA regulations for rating residuals of cold injuries were published in the Federal Register which may generate additional questions and concerns from veterans (see paragraph 7).

   b. Under Secretary for Health's Information Letter IL 10-96-030 entitled "Recommendations for the Care and Examination of Veterans with Late Effects of Cold Injury," was published on December 31, 1996, and provided clinical guidelines and information for caring for these veterans.

   c. The following additional points should be emphasized especially when performing examinations for compensation claims:

      1. There may be no symptoms, once the acute cold injury has resolved, until many years later; and

      2. The fact that a veteran has another disease, such as hypertension or diabetes, that can result in findings similar to cold injury residuals, should not preclude the examiner from identifying such findings as residuals of cold injury, if it is at least as likely as not that they are.

   d. It is necessary to correct an error which has appeared in a number of news publications and has confused some veterans. The VA Chief Public Health and Environmental Hazards Officer was misquoted as implying that cold injuries can cause diabetes. It is important to clarify to veterans that subsequent development of diabetes can complicate medical problems due to cold injuries, but diabetes is not the result of the cold injury.

3. Educational activities. On June 12, 1997, a satellite videoconference on cold injuries was broadcast to VA medical centers. Further efforts to provide staff education are encouraged so VA staff can better address veterans' cold injury questions. Copies of the videotape from this broadcast can be requested by each facility's Library Service for viewing by staff who were unable to participate in the live program (see paragraph 7). A Cold Injury Self Study Program is being developed which will include the opportunity for physicians to earn continuing medical education credit.

4. Examination Protocol. A Cold Injury Protocol Examination and a Protocol Examination History have now been developed (Atts. A and B). Veterans Health Administration (VHA) encourages use of these protocols especially when examining veterans for compensation purposes to assure that a detailed history is obtained from the veteran and a comprehensive evaluation is performed and documented. The examination protocol (not the history protocol) can be accessed electronically through VCA's Veterans Health Information Systems and Technology Architecture (VISTA) computer system (formerly the Decentralized Hospital Computer System (DHCP)) as Automated Medical Information Exchange (AMIE) worksheet #1730. Physicians may receive assistance in accessing this protocol from Compensation and Pension (C&P) clerks, Information Resources Management (IRM) staff, Chiefs of Health Administration Services (HAS), or other staff members depending on the facility's local organization.

5. Recommended Distribution. Copies of this Information Letter need to be provided to the C&P Coordinator and Associate Chief of Staff for Ambulatory Care.

6. Further Information. Questions regarding cold injuries may be addressed as follows:

   a. General administrative or clinical questions. Contact the Office of Public Health and Environmental Hazards (13), VA Central Office, telephone 202-273-8575, fax 202-273-9080.

   b. Compensation questions. If a veteran wishes to file a claim or has questions about the status of a claim, the veteran needs to call 1-800-827-1000.

7. References

   a. Federal Register, December 11, 1997 – pages 65207, 65218, 65223 (62 FR 65207).

   b. Videotape "Long-Term Sequelae of Cold Injury: Diagnosis and Management."

S/Robyn Nishimi, Ph.D. for
Kenneth W. Kizer, M.D., M.P.H.
Under Secretary for Health

Attachments

Attachment A – Compensation and Pension Examination (AMIE Worksheet #1730) (see page 29 in this book)

Attachment B – Protocol Examination History for Cold Injuries (see page 34, in this book)

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Compensation and Pension Examination (AMIE Worksheet #1730)
Cold Injury Protocol Examination

Name: _____ SSN: _____

Date of Exam: _____ C-number: _____

Place of Exam: _____

Narration: Veterans during World War II, the Korean War, and in smaller numbers during other campaigns, have suffered cold injuries, including frostbite (freezing cold injury or FCI) and immersion foot (nonfreezing cold injury or NCI). Documentation of such injuries may be lacking because of battlefield conditions. A number of long-term and delayed sequelae to cold injuries are recognized, including peripheral neuropathy, skin cancer in frostbite scars, and arthritis in involved limbs.

Review Examination: Any veteran examined for residuals of cold injury should undergo a cold injury protocol examination if it has not already been carried out. If the veteran has already had a cold injury protocol examination, only an interval history is required, and the extent of the examination, laboratory tests performed, etc., will be determined by the examiner based on the history, and as requested.

A. **Review of Medical Records:**

B. **Medical History (Subjective Complaints):**

History of Cold Injury: If the cold injury protocol form has been filled out by the veteran, most details about the circumstances of the acute cold injury and its subsequent course will be recorded. Review for any needed expansion or clarification by the veteran. If the protocol history form has not been completed, obtain the following history and comment on each:

1. Description of the circumstances of the cold injury.
2. Parts of the body affected.
3. Signs and symptoms – at time of acute injury.
4. The type of treatment and where it was administered.
5. Any treatment since service – where and what type.

6. Current symptoms – specifically inquire about:
    a. Amputations or other tissue loss.
    b. Cold sensitization.
    c. Raynaud's phenomenon.
    d. Hyperhidrosis.
    e. Paresthesias, numbness.
    f. Chronic pain resembling causalgia or reflex sympathetic dystrophy.
    g. Recurrent fungal infections.
    h. Breakdown or ulceration of frostbite scars.
    I. Disturbances of nail growth.
    j. Skin cancer in chronic ulcers or scars.
    k. Arthritis or joint stiffness, including limitation of motion of affected areas.
    l. Edema.
    m. Changes in skin color.
    n. Skin thickening or thinning.
    o. Any sleep disturbance due to associated symptoms.
    p. Cold feeling (relationship to season or not).
    q. Numbness, tingling, burning.
    r. Excess sweating.
    s. Pain – location, intensity, constancy, precipitating factors (cold, walking, standing, night pain); type (sharp, burning, etc.).

7. Current treatment, including nonmedical measures taken – moving to warmer climate, wearing multiple pairs of socks, etc.

Other Medical History:

1. Major illnesses, surgery, current medical conditions and their treatment, including diabetes mellitus or hypertension.

2. Smoking history, other risk factors for vascular disease, history of skin cancer.

C. **Physical Examination (Objective Findings):**

Address each of the following and fully describe current findings:

1. **General:** Carriage, gait, posture.
2. **Skin:**
    a. Color.
    b. Edema.
    c. Temperature.
    d. Atrophy.
    e. Dry or moist.
    f. Texture.
    g. Ulceration.
    h. Hair growth.
    i. Evidence of fungus or other infection.
3. **Scars:**
    a. Location.
    b. Length.
    c. Width.
    d. Color.
    e. Tenderness.
    f. Raised or depressed.
    g. If of head or neck, any disfigurement.

4. **Nails:**
    a. All or part missing
    b. Evidence of fungus infection
    c. Deformed or atrophic
5. **Neurological:**
    a. Reflexes.
    b. Sensory – subjective complaints of pain, numbness, etc., Objective sensory changes – pinprick, touch.
    c. Motor – weakness, atrophy.
6. **Orthopedic:**
    a. Pain or stiffness of any joints affected by cold injury.
    b. Deformity or swelling of any joints.
    c. Measure range of motion of all affected joints.
    d. Strength of ligaments in affected areas.
    e. Pes planus.
    f. Callus.
    g. Pain on manipulation of joints.
    h. Loss of tissue of digits or other affected parts.
7. **Vascular:**
    a. Status of peripheral pulses.
    b. Doppler study to confirm vascular compromise, if indicated.
    c. Evidence of vascular insufficiency – edema, hair loss, shiny atrophic skin, etc.
    d. Blood pressure in arms and legs (is ratio normal?).
    e. Evidence of Raynaud's phenomenon.

D. **Diagnostic and Clinical Tests:**

Provide:

1. X-rays of affected areas of extremities if never done or if not done in past five years.
2. Doppler study of blood vessels, if indicated.
3. Nerve conduction studies, if indicated.
4. Biopsy of any area suspicious for malignancy.
5. Scrapings to confirm fungus infection.
6. Include results of all diagnostic and clinical tests conducted in the examination report.

E. **Diagnosis:**

1. List each diagnosis and state whether related to cold injury (if that can be determined).
2. Specialty exams that might be needed:
    a. Neurology.
    b. Podiatry.
    c. Dermatology.
    d. Rheumatology.
    e. Others as needed.

Signature _____ Date _____

Protocol Examination History for Cold Injuries 1 of 7

Name–Last _____ First _____ Middle _____

Social Security Number _____ VA claim number: _____

Age now _____

Age at time of cold injury _____

Circumstances of Injury

1. Where were you when you suffered a cold injury? _____

   _____

2. Type of cold injury (if you know.)

   ❑ Frostbite ❑ Frostnip ❑ Frozen Feet

   ❑ Trenchfoot ❑ Immersion Foot ❑ Other – specify _____

   _____

3. Parts of body affected by cold injury (please mark all that apply)

   Hands . . . . . ❑ Left ❑ Right

   Feet . . . . . ❑ Left ❑ Right

   Ears . . . . . ❑ Left ❑ Right

   Cheeks . . . . . ❑ Left ❑ Right

   Temples . . . . . ❑ Left ❑ Right

   ❑ Nose

   ❑ Other – specify _____

4. What was the approximate date of your cold injury? _____

5. What were the conditions at the time of the injury?

   Weather _____ Temperature _____

   Length of your exposure ❑ Minutes ❑ Hours ❑ Days ❑ Weeks

   ❑ Wet or ❑ Dry

   Activity of unit at time of exposure _____

   _____

Protocol Examination History for Cold Injuries 2 of 7

6. How did you become aware that you had suffered a cold injury? _____

_____

7. What were your symptoms at the time of the injury?

❑ Pain ❑ Swelling ❑ Discoloration – white, red, blue, black

❑ Blisters ❑ Tissue loss, loss of toes or fingers or parts of them

❑ Numbness, tingling ❑ Stiffness ❑ Weakness

❑ Other – specify _____

Treatment

8. Did you seek treatment at the time of the injury? . . . . . ❑ Yes ❑ No

If yes, where were you treated?

❑ Hospital ❑ Aid station ❑ In the field

❑ Other – specify _____

If hospitalized, where and for how long?

Where? _____ How long? _____

Where? _____ How long? _____

Where? _____ How long? _____

Where? _____ How long? _____

Where? _____ How long? _____

Where? _____ How long? _____

Who treated you?

❑ Doctor ❑ Nurse ❑ Medic ❑ Comrades ❑ Self

❑ Other – specify _____

How were you treated?

❑ Bedrest ❑ Surgery ❑ Medicines ❑ No treatment

❑ Other – specify _____

_____

Protocol Examination History for Cold Injuries — 3 of 7

9. After your cold injury, were you relieved from duty? . . . . . ❑ Yes ❑ No

   If yes, did you return to duty? . . . . . ❑ Yes ❑ No

   If yes, how long after the injury did you return to duty?

   ❑ Hours ❑ Days ❑ Weeks ❑ Months

   ❑ Other – specify _____

10. The acute injury

    How long did the cold injury symptoms last?

    ❑ Hours ❑ Days ❑ Weeks ❑ Months

    ❑ Other – specify _____

    Did the appearance of the injured parts return to normal? ❑ Yes ❑ No

    If yes, how long did that take?

    ❑ Hours ❑ Days ❑ Weeks ❑ Months

    ❑ Other – specify _____

    Were you left with any scars? . . . . . ❑ Yes ❑ No

    If yes, where? Please describe _____

    _____

    _____

11. Did others in your unit also have cold injuries? . . . . . ❑ Yes ❑ No

    If yes, how many? _____

    What types of problems did they have? _____

    _____

    _____

12. Have you had any other cold injuries? . . . . . ❑ Yes ❑ No

    If yes, did they occur when you were

    ❑ In military service ❑ Before military service ❑ After military service

    Please describe? _____

    _____

    _____

Protocol Examination History for Cold Injuries — 4 of 7

13. Did any symptoms remain after the injured part was healed? ❑ Yes ❑ No

If yes, please complete the following (please mark all responses that apply to you.)

a. Pain – **When**

❑ All the time ❑ Worse in cold weather ❑ Worse at night

❑ Other – specify _____

Pain – **Where**

❑ Tips of fingers or toes ❑ In joints of fingers or toes ❑ In arches of feet

❑ In legs ❑ all over affected parts

❑ Other – specify _____

Type of pain

❑ Sharp ❑ Dull ❑ Burning ❑ Heaviness

❑ Other – specify _____

b. Numbness . . . . . ❑

c. Tingling or pins and needles feeling . . . . . ❑

d. Weakness of hands, feet, legs . . . . . ❑

e. Swelling . . . . . ❑

f. Changes in color of affected parts . . . . . ❑

g. Sensitive to cold . . . . . ❑

h. Excessive sweating of feet or other affected parts . . . . . ❑

i. Fungus infection (athlete's foot, for example) . . . . . ❑

j. Ulcers of cold injured parts . . . . . ❑

k. Misshapen nails . . . . . ❑

l. Breakdown of skin of cold injured parts . . . . . ❑

m. Decrease or loss of sensation . . . . . ❑

n. Change in thickness of skin of affected parts (thicker or thinner) . . . . . ❑

o. Skin cancer diagnosed in affected area . . . . . ❑

p. Arthritis diagnosed in affected area . . . . . ❑

q. Other – specify _____

Protocol Examination History for Cold Injuries 5 of 7

After Service

14. Did you take any special precautions or make changes in your life or lifestyle after service specifically because of the cold injury? Please explain. _____

_____

_____

_____

_____

15. Occupational effects

What was your occupation prior to service? _____

How long did you have that job? _____

What was your occupation after service? _____

How long did you have that job? _____

Did the cold injury have any effect on your work? . . . . . ❑ Yes ❑ No

If yes, what was the effect? _____

_____

_____

16. Treatment after service

Did you receive any treatment after service for problems that you felt were related to the cold injury? . . . . . ❑ Yes ❑ No

If yes, what were you treated for? _____

_____

_____

_____

Where were you treated? _____

_____

_____

_____

When? _____

Protocol Examination History for Cold Injuries 6 of 7

Current Situation and Treatment

17. Have you discussed your cold injury with your current doctors? . . . . . ❑ Yes ❑ No

    If yes, have they recommended or prescribed any treatment, special foot care, etc.? . . . . . ❑ Yes ❑ No

18. Are you receiving any treatment now for problems you believe are related to the cold injury? . . . . . ❑ Yes ❑ No

    If yes, what treatment are you receiving? _____

    _____

    _____

    _____

    Where are you being treated? _____

19. What do you believe is the major problem your cold injury is causing you now? _____

    _____

    _____

    _____

20. Is there anything else you would like us to know about your cold injury or its aftereffects? . . . . . ❑ Yes ❑ No

    If yes, please explain._____

    _____

    _____

    _____

21. Are there any questions you have for your doctor about the effects of your cold injury? . . . . . ❑ Yes ❑ No

    If yes, please explain._____

    _____

    _____

    _____

    _____

Protocol Examination History for Cold Injuries

22. How would you describe your overall state of health at present? _____

_____

_____

_____

_____

Briefly describe any problems you have other than the effects of cold injury. _____

_____

_____

_____

_____

_____

_____

_____

Signature _____ Date _____

Revised Cold Injury Regulation for Disabilities (part of 38 CFR 4.104)

The final revision of the cold injury regulations was published in the *Federal Register* on July 14, 1998 (63 FR 37778) and went into effect on **August 13, 1998.**

(The numbers on the right represent percentage of disability evaluation.)

7122 Cold injury residuals:

With the following in affected parts:

Arthralgia or other pain, numbness, or cold sensitivity plus two or more of the following: tissue loss, nail abnormalities, color changes, locally impaired sensation, hyperhidrosis, X-ray abnormalities (osteoporosis, subarticular punched out lesions, or osteoarthritis) 30

Arthralgia or other pain, numbness, or cold sensitivity plus tissue loss, nail abnormalities, color changes, locally impaired sensation, hyperhidrosis, or X-ray abnormalities (osteoporosis, subarticular punched out lesions, or osteoarthritis) 20

Arthralgia or other pain, numbness, or cold sensitivity 10

Note 1: Separately evaluate amputations of fingers or toes, and complications such as squamous cell carcinoma at the site of a cold injury scar or peripheral neuropathy under other diagnostic codes. Separately evaluate other disabilities that have been diagnosed as the residual effects of cold injury, such as Raynaud's phenomenon, muscle atrophy, etc., unless they are used to support an evaluation under diagnostic code 7122.

Note 2: Evaluate each affected part (e.g., hand, foot, ear, nose) separately and combine the ratings in accordance with §§ 4.25 and 4.26.

Frostbite: General and Specific Treatment
The Alaskan Method

James O'Malley, M.D.; William Mills, M.D.; Bruno Kappes, Ph.D.; Scott Sullivan, R.N.

This section is a clinically oriented synopsis of the Alaskan frostbite treatment methods…

The basis of treatment for severe freezing injury is to keep the patient quiet, well nourished, well hydrated and relatively pain free. Excellent and meticulous wound care with avoidance of any sort of trauma is essential. In treatment, the injured extremities are elevated above the level of the heart to decrease edema, and the patient's mobility is limited only by the necessity to prevent any trauma to his injured extremities.

With the patient hospitalized, the affected extremities are treated with clean, dry dressings and twice-daily whirlpool baths using surgical scrub soap such as Hibiclens or Phisohex. Loose stockinette material is used to keep the extremity covered. Bed cradles are used over the feet, and two-finger gloves can be fashioned from stockinette material so the patient may feed himself. It is our opinion that superficial blebs should remain intact for several reasons: the intact bleb makes an excellent biological dressing; sometimes the wound is less painful if the bleb is kept intact. At a point several days later when the vesicular fluid resorbs, the bleb may burst or be traumatically lacerated, but usually by this time, the underlying injured epithelium healed. As time passes dead skin will shrink onto the extremity and may cause a tourniquet-like effect on the healed skin beneath it necessitating debridement.

All patients are given five grains of aspirin per day for its antiplatelet effect. Aspirin also tends to block the arachidonic acid cascade and prevent additional clotting in the micro-circulation. In addition, they are given phenoxybenzamine chloride in doses of 10 mg twice daily for five days with a tapering dose after that. Phenoxybenzamine is an alpha adrenergic blocking agent, which reduces total peripheral resistance, venous tone and blood pressure.

More recently, we have reactivated the use of low molecular weight dextran (Dextran 40) at a rate of 25 cc an hour using an IV infusion pump. Dextran had been shown to be quite useful in experimental freezing injuries, and more recently we have seen a salutary effect on the clinical outcome of freezing injuries to the extremities. It is thought that the usefulness of Dextran is secondary to its antiplatelet effect at the level of the microcirculation.

If the patient has recently transferred from extremes in altitude or cold, we assume dehydration, and we correct this with vigorous IV fluid replacement. A nutritional assessment is done, and ordinarily a high-protein, high-fiber diet is given.

A technetium 99^{m} scan is done of the affected extremity shortly after the patient arrives. This study can be repeated in two to three days. Our experience with technetium is that it gives a good picture of the initial circulation of the affected extremity and when repeated shows progression. Technetium scans are most helpful in predicting the extent of injury and to aid in establishing a prognosis as well as documenting the success of the treatment.

The usefulness of sympathetic block (another often used modality of the past) in treating freezing injuries is in the process of further clinical evaluations. This study has involved selected cases over the last three years, and certain conclusions can be drawn. Freezing injuries to the feet can sometimes be quite painful, and the pain -relief aspect of the epidural can be utilized to good effect. In addition, the sympathetic block that generally occurs with the epidural can be useful in re-establishing blood flow at least in the microcirculation of the skin. Stellate ganglion blocks have been used for freezing injuries to the hands.

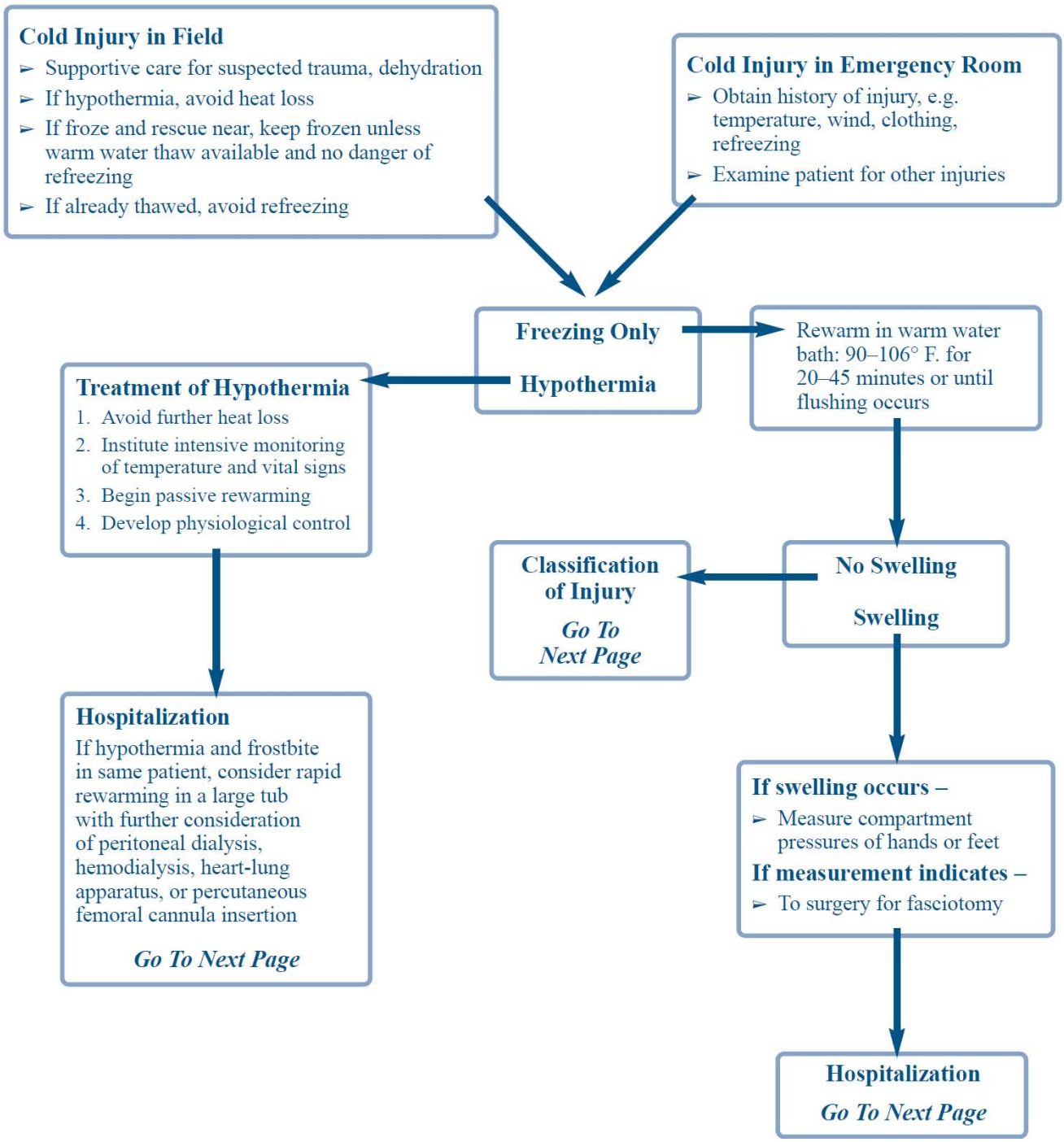
If, as the hospitalized patient's injury is evolving and it appears as though some tissue loss will occur, then the object of continued treatment is to allow demarcation of the injury as much as possible. This allows for a maximum length of an eventual amputation and maximum eventual function of the affected part. Continued daily clinical evaluation of the progress of mummification is essential. If it appears as though some tissue loss beyond the level of the digit or more proximal loss than the proximal M-P joint will occur, we involve all members of the treatment team, including a plastic surgeon, to evaluate the patient so that all future reconstruction options are considered. Any surgery beyond minor debridement of freezing injuries should take place late in the course of treatment. The one exception to this is when the entire distal extremity has been frozen and upon adequate thawing no circulation is seen. In these unusual cases, which in our experience mostly involve the feet, the patient is still allowed to demarcate his injury as much as possible.

The patient with no circulation to the extremity is carefully followed for signs of liquefaction necrosis and sepsis. If, and when, this occurs, and a hectic fever curve is evident, indicating sepsis, IV antibiotics are initiated early with broad spectrum coverage. The earliest amputations for frostbite are done in these cases of liquefaction necrosis with sepsis not responding to antibiotic treatment. Amputation done too early allows for retraction of the muscles and tissues and results in a shorter amputation than if the injury is allowed to demarcate.

Originally published in *Alaska Medicine* Volume 35, Number 1 Jan/Feb/Mar 1993. Used by permission.

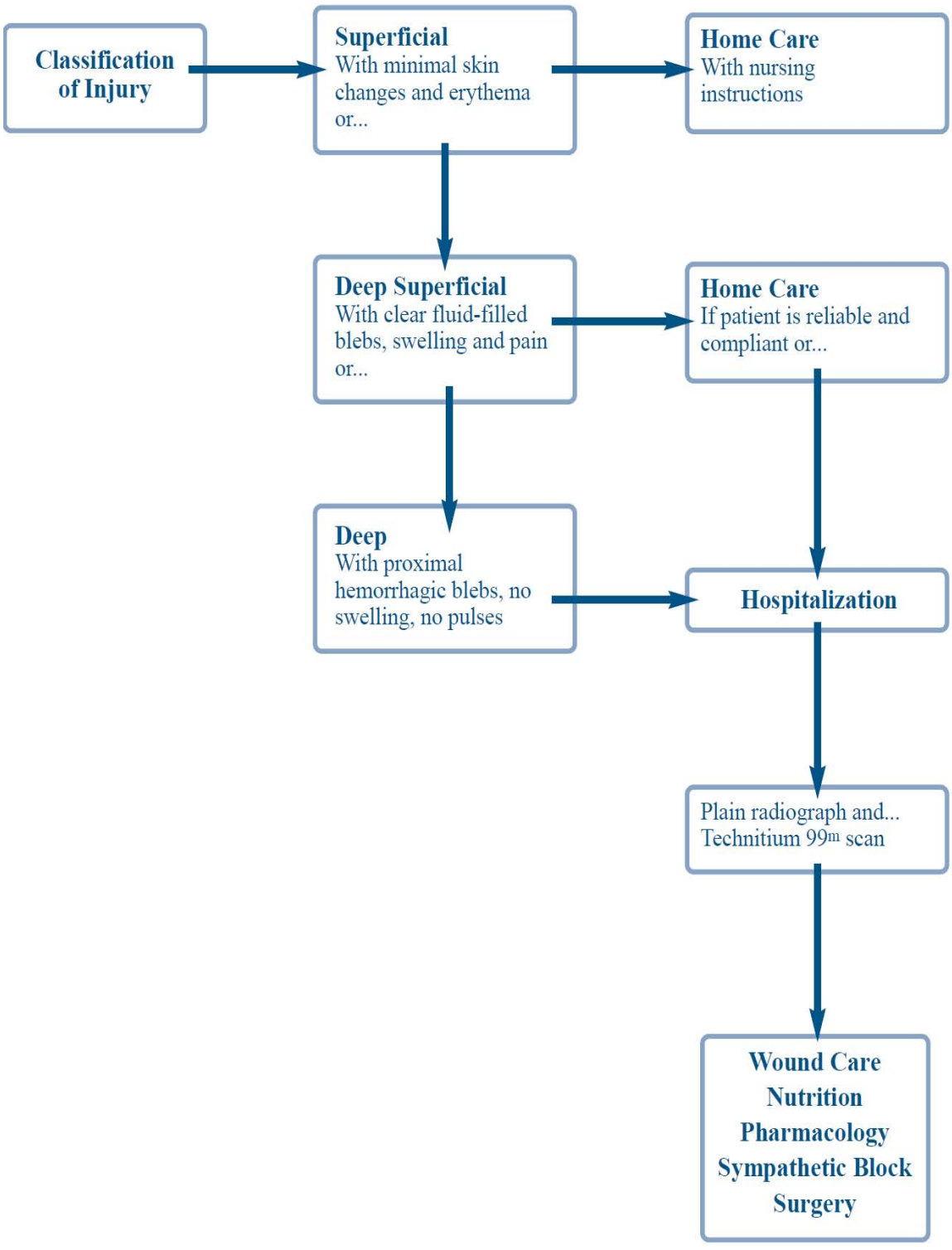
Algorithm for Treatment of Cold Injury

Adapted from The Alaskan Method



Algorithm for Treatment of Cold Injury Continued

Adapted from The Alaskan Method



Problems for the Thermal Nurse, if Patient Hospitalized

Nursing Goal: To strive for maximal patient compliance of the frostbite regime until optimal results are achieved.

Plan/Action:

1. Perform initial nursing history and physical assessment. Determine impact of cold injury.
2. Investigate patient's real and perceived needs. Introduce frostbite regime.
3. Deliver tailored care to satisfy identified needs. Provide support for maximum compliance.
4. Develop and implement discharge plan.

Nursing Concerns: Wound care/infection control; Nutrition; Hydration; Safety; Exercise; Relaxation; Mental Health; Cognitive, emotional and spiritual needs; Comfort; Adaptation.

Roles:
M.D. – Architect of Care
Nurse – Project Supervisor
Support Services – Care Contractors
Patient – Customer of care, Consumer

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Standard Orders for Admission, Thermal Unit

1. Routine vitals
2. I & O x 24 hours
3. Activity for
   - Upper Extremity: OOB prn hallway
   - Lower Extremity: Wheelchair or crutches
4. Foot cradle to bed, elevate feet on two pillows
5. Elevate hands on two pillows when down or elevate with stockinette slings
6. Dress extremity with frostbite booties or gloves (sterile stockinette)
7. High calcium, high protein diet
8. Push fluids p.o.
9. IV D5 1/2 NS 125 cc/hr x 24 hours
10. Td .5 cc im adult, DT .5 cc IM pediatric
11. Dibenzylene 10 mg p.o. qd x three days then 10 mg bid x three days
12. Vitamin C 500 mg tid
13. Stresstab with zinc 1 qd
14. ASA 325 mg p.o. qd x ten days
15. Dextran 40 (LMW) 25 cc/hr via IV infusion pump
16. Labs: CDC, SMA 12, UA
17. TCN Scan of affected part, plain film of affected part
18. Twice daily
    - whirlpool treatment, 20 minutes at 93–95º F. with Hibiclens
    - rinse and dry post whirlpool
    - feet may be open to air beneath bed cradle
    - culture open draining wounds
19. Consent to photograph
20. Consult variously:
    - Thermal Case Manager
    - Social Services
    - Internal Medicine
    - Psychiatry
    - PT for exercises and/or crutch training
    - Plastic Surgery

Originally published in *Alaska Medicine* Volume 35, Number 1 Jan/Feb/Mar 1993. Used by permission.

Freezing Cold Injury

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Introduction

Clinical studies and case study evaluations have shown, that depending on the degree of a freezing or non-freezing cold injury, both insults may result in acute and long term sequelae. Patients with long term sequelae often demonstrate signs of peripheral neuropathy, reduced circulation, cutaneous fat pad loss, scarring, intrinsic muscle contracture and nail changes. To understand one of the insults, freezing cold injury, it is important to know how it occurs.

Background

Historically, freezing cold injury was referred to as "frostbite," although terminology and diagnosis of freezing and non-freezing cold injury have often been confused. Freezing cold injury damage occurs in two segments: the cellular event, where cellular changes occur during the course and duration of freezing, and the thawing and post-thawing event where vascular injury may occur following freezing and during thawing as in reperfusion injury.

Freezing cold injury (FCI) may be confined to the skin or may involve the deeper tissues of the body when there is sufficient heat loss due to tissue freezing with formation of ice crystals in the extracellular spaces. The diagnosis in freezing cold injury is usually made as either superficial or deep cold injury. Frostbite generally develops when the skin temperature falls below -3° C/26° F or when the air temperature is below -12° C/10° F. It may occur at a temperature nearer the freezing point when other elements such as high winds, dampness, or chilling of the body are present. The onset of frostbite may initially cause a) pain, then b) tingling or stinging and eventually results in c) numbness or the loss of sensation to the point of anesthesia. Those early signs indicate that tissue freezing is a danger. When all pain or tingling has disappeared, leaving only anesthesia, the body part and the tissue have then become frozen.

The major driving force for physiological changes to occur is usually the change in the body's peripheral temperature, the heat loss of the skin and muscles. In the body's attempt to minimize thermal loss, certain reactions will occur causing changes in muscle tone,

metabolism, and behavior, depending on the degree of the drop of the peripheral temperature. In the event that these responses are not effective, the body's core temperature that of the brain, heart, and lungs, will begin to drop. This condition, hypothermia, is defined as a 2° C drop from the body's internal core temperature, normally 35° C (96° F.) in resting condition in a thermal neutral environment. As the body cools, the flow of blood from the core to the periphery becomes minimal, and allows the limbs, especially the digits, to become susceptible to cold injury, causing irrevocable loss of function and likelihood of anatomical destruction.

Definitions

Freezing cold injury (Frostbite) is true tissue freezing and occurs when there is sufficient heat loss in the local area to allow ice crystals to form in the extracellular spaces, and extract cellular water. The water then freezes, so that eventually cellular dehydration and cell collapse occur.

Occurrence

Freezing cold injury occurs by the following mechanisms:

1. True frostbite of superficial or deep tissue.
2. Mixed injury occurring when freezing follows a non-freezing injury. (This is very serious and often results in severe tissue loss.)
3. Freezing, then thawing by any means, with re-freezing. (This generally has a poor clinical outcome with total tissue destruction and early mummification of distal tissues that occur often within five to seven days.)
4. Hypoxia, high altitude environment injury, often with severe dehydration of tissues, due to general body dehydration and hypovolemia with extremity freezing. (The prognosis here is poor, especially if associated with compartment pressure syndrome.)
5. Extremity compartment compression, from any cause, followed by freezing. (This too gives very poor results if compartment pressures are elevated and are not relieved by fasciotomy.)
6. Extremity fracture or dislocation and superimposed freezing. (The results are often poor if the fracture or the dislocation is left unreduced. Best results appear to follow rapid rewarming techniques and open or closed wound care with treatment of any vascular impairment.)

Hypothermia

In hypothermia, associated with superimposed freezing injury of extremities, paramount importance must be given to the restoration of heat to the victim, under total physiological control and monitoring. Good results for a freezing injury can occur with tub rewarming of the hypothermia and simultaneous thawing in warm water of the frozen extremity. Perhaps even better, safer results occur with the application of peritoneal dialysis for the hypothermia at the same time the frozen extremities are rewarmed rapidly in a tub. The correction of hypothermia and freezing injury must be done with adequate monitoring. One of the dangers here is the sudden release of metabolites, especially the release of excess amounts of potassium from muscle degradation and injury. This may cause hyperkalemia and cardioplegia. The immediate balance of electrolytes and restoration of normal pH levels is imperative.

Stages of Freeze-thaw Cold Injury

The first event in the freeze-thaw injury, is the supercooling or freezing event. First, with exposure to cold, there is an early tissue response to cooling. This is often described as a cold-induced vasodilatation (CIVD), the “Hunting Response”. (One hypothesis holds that at the same time or as cooling continues, arteriovenous anastomoses develop with shunting of blood from the distal area.) Soon, after sufficient heat loss occurs to allow freezing, ice crystals form in the extra cellular fluid spaces and extracellular freezing occurs. It is possible that some structural damage may result from continued ice crystal growth. Extracellular osmotic pressure increases resulting in cell volume reduction and solute concentration in the extracellular spaces and in the interstitium.

As freezing continues, there is an elevated concentration of electrolytes, protein denaturation, intra- and extracellular pH changes, intra- and extra cellular dehydration, freezing of extracellular water, loss of protein bound water in cells, and destruction of essential enzymes. Cell membrane damage occurs during this period. Impairment of microvascular function and increased cell wall permeability, with critical endothelial cell injury, and endothelial separation from the internal elastic lamina of the arterial wall occur. During this time, severe injury to chondrocytes may also occur since cartilage, especially epiphyseal
cartilage, is susceptible to freezing damage. Further insult causes ultrastructural capillary damage, mitochondrial loss in muscle cells, and injury to other intracellular structures, such as nuclei.

Thawing (rewarming) and post-thaw events are vascular events. Depending upon the nature of thawing, post-thaw hyperemia, cyanosis, even total circulatory failure usually develops. Proximal or distal blebs (or no blebs) may appear. According to Meryman, the usual series of events is that of vasodilatation, edema, and stasis. Corpuscular aggregation begins with thawing, often associated with progressive ischemia or with hyaline plugs in the vascular tree. Occasionally, because of associated or combined injury, increased pressures may develop in soft tissue compartments. It is essential, if this does occur; that the compartment pressures be measured before irreversible damage occurs after the thawing. Changes related to reperfusion injury include formation of oxygen free radicals, neutrophil activation and other inflammatory events.

An early response in the thawing stage, and perhaps in cooling too, is the arachadonic acid cascade that liberates prostaglandins and thromboxane, which predisposes to vascular clotting. Production of proteolytic enzymes leads to increased membrane permeability. The utilization of five grains (325 mg) of aspirin at the time the patient is seen, and thereafter each day, is usually sufficient to block thromboxane formation.

Eventually vascular reconstitution and clot dissolution may begin, or capillary and peripheral vessel collapse may occur. When vascular collapse occurs, it is followed by micro and macro vascular thrombosis, venule and arterial obstruction by thrombosis, tissue ischemia, necrosis, and gangrene, which results in loss of the affected part or area.

Following thawing, should refreezing occur, intracellular ice formation is most probable, resulting in cell and vital organ destruction. Thawing in this usually deep injury, results in unrelieved thrombosis, stasis, and failure of cell repair. Loss usually occurs near or at the level of the second freeze.

The Sequelae of Cold Injury

Sequelae may be transient or long lasting. The transient sequelae include:

1. Hyperhydrosis
2. Hypesthesia or anesthesia of digits
3. Limitation of motion of the interphangeal and metatarsal or metacarpal joints
4. Joint swelling of the IP and MP joints
5. Edema of digits, hands, feet

6. Thin, fragile epidermis in involved areas
7. Nail loss
8. Intrinsic muscle atrophy
9. Fat pad loss of distal digital tips
10. Pain from injury to peripheral nerves and small vessels as a result of ischemia

The late or long-lasting sequelae are even more numerous and often increase over time. They include:

1. Deep fixed scars over the affected area
2. Atophy or fibrosis of the affected musculature
3. Flexion or extension deformity of distal joints, especially involving the toes, often with hammer toe or claw toe deformity
4. Volar fat pad loss of fingers and toes
5. Hyperesthesia of distal tips of digits with increased sensitivity to heat and cold
6. Residual hypesthesia of digital tips
7. Decreased proprioceptive sense of digital tips
8. Permanent nail bed deformity, as onychogryphosis, often with associated fungus infection (onchomycosis)
9. X-ray evidence of sub and peri-articular lytic destructive changes of bone and cartilage, especially in the phalangeal joint areas
10. Avascular necrosis of bone, especially in the phalanges, metatarsi and tarsi (ex, calcaneus)
11. In children or young adults, epiphyseal necrosis, or total destruction of physis or epiphysis, (growth plate destruction) with joints or phalangeal angulation, deformity or shortening
12. Chronic ulceration, infection or osteomyelitis in area of cold injury
13. Loss of fibro-cartilage of ears and external ear part loss
14. Decreased capillary perfusion by isotope examination, (indicative of endothelial damage), and cold sensitization

15. Rare findings of carcinoma (usually squamous cell) in long standing draining sinus tracts, or with chronic wound infection or osteomyelitis
16. Interphalangeal joint immobility, marked limitation of joint motion or fusion of IP joints
17. Carpal and tarsal tunnel syndrome (seen in acute stage too)
18. Intermittent or constant pain in hands or feet, often extending in lower extremities to levels above the apparent injury level
19. Variable findings of hyperhydrosis, hypohydrosis, anhydrosis
20. The ultimate in long standing sequelae, or the result of severe early cold insult, namely amputation of the involved part at any level

Relevance to Clinicians in the Department of Veterans Affairs

It is important that VA medical practitioners examining and caring for veterans who have experienced cold injuries, be familiar with the recognized long-term and delayed sequelae, including peripheral neuropathy, skin cancer in frostbite scars (which appear especially on the heels and earlobes), arthritis in involved areas, chronic tinea pedis, high arches occurring from intrinsic muscle contracture, stiff toes and hammer toes, nocturnal pain and cold sensitization. It is likely that these veterans' cold-related problems will worsen with age.

It must be acknowledged that there has been limited research on delayed effects following cold injuries and many unanswered questions exist (such as whether there are long-term or delayed effects on internal organs of individuals surviving acute cold injury). In addition, the diagnosis of the subtle sequelae of non-freezing cold injury that can also be very debilitating are sometimes difficult.

Patient Interview

An important part of the diagnostic process of caring for the delayed effects of a cold injury, is an interview with the patient to record all the pertinent data. This should include:

(a) Pertinent information regarding the original cold injury, including ambient temperature, wind if present, duration of exposure,

(b) Treatment if any, administered at the time of the injury, whether on the march, in the field or in base dispensaries or battalion aid stations or in shelters or vehicles along the way, and

(c) Progressive symptomatology. Some patients may underestimate the severity of their original injuries, and may not be aware that the symptoms they experience now are a result of their cold injury. The examining physician must then develop and follow a checklist of symptoms and establish chronology of onset of these symptoms.

Due to the lack of information in medical records at the time the cold injury occurred, the medical history of cold-injured veterans should include:

1. Description of the acute cold injury (e.g., circumstances such as ambient temperature, length of exposure, wet/dry conditions), signs and symptoms, diagnosis made, treatment provided). Individuals at higher risk for cold injuries include:

   • Riflemen of lower ranks

   • African Americans

   • Poorly nourished individuals

   • Those with concomitant injuries

2. Description of signs and symptoms in the interval following the acute cold injury, (amputations or other tissue loss, cold sensitization, Raynaud's-like phenomena, hyperhidrosis/excessive seating, neuropathy/disturbances of sensation, chronic pain resembling causalgia/reflex sympathetic dystrophy, weakness/reduced strength, sleep disturbances due to pain, recurrent fungal infections, breakdown/ulcerations of scars, disturbances of nail growth, skin cancer in chronic ulcers or scars, arthritis/joint stiffness/loss of range of motion in the affected limbs, reduction in mobility, swelling, pain/paraesthesia/numbness, changes in skin color, skin thickness, intermittent blisters and scaling of skin to the knee)

3. Medical treatments and other measures to alleviate symptoms (having to use multiple pairs of socks, sleeping with socks on, moving to a warmer climate, etc.)

4. Current symptoms and other subjective data, such as pain (including location, intensity, nocturnal pain, sleep disturbances), cold feelings (including differences between winter and summer), numbness, excessive sweating, tingling, and burning sensations

5. Information about smoking; current medications; other diseases such as diabetes, hypertension, etc.

Physical examination

The physical examination of cold-injured veterans should include:

1. Blood pressure – of the arm, the leg and the ratio of ankle blood pressure/brachial blood pressure that may be useful in evaluating occlusive arterial disease of the lower extremity.
2. Examination for loss of body parts/tissue and amputations. A number of Chosin veterans have had late amputations because of complications of peripheral vascular disease.
3. Examination of the skin for temperature, color, hair, sweating during examination, scars, ulcerations, fungal infections, skin cancer, and nail changes including onychomycosis.
4. Neurological examination for decreased strength, reflexes, pinprick, temperature sensation, discrimination, and light touch.
5. Joint examination for signs of arthritis and loss of range of motion.

Clinical Studies

Clinical laboratory tests should include nerve conduction studies for nerve velocity and function, if symptoms of motor or sensory loss are present. It is important to demonstrate whether permanent sensory loss of the involved digits or segments is present.

Laser Doppler studies may be useful for demonstrating vascular function, but are currently primarily research tools.

Radiographic examination should be compared with past examinations if those tests have been made, to determine progress of any bone pathology, or evidence of avascular necrosis of bone. The radiographic studies should include flat plate studies and even evaluation by CAT scan or MRI of the local area in some cases.

Infrared thermography has been used to document the pattern and rate of rewarming after mild cooling. Thermal threshold measurement studies to define loss of skin sensation to temperature have also been used in British studies. Technetium scans may be valuable for bone blood flow studies.

Biopsies of suspected neoplasia are indicated. Squamous cell carcinoma of draining sinuses might be found in long standing cases, although this is quite rare.

Periodic reevaluations may be necessary to document progression. These evaluations may include clinically indicated tests for diabetes (although not a result of cold injury, diabetes may complicate the sequelae of cold injury), arthritis, and evaluation of any peripheral vascular disease. These studies should include ultrasound Doppler or specialized testing using infrared thermography. It might be helpful to repeat technetium scans to demonstrate increase or decrease in pathology in distal areas.

Specialty consultations may be indicated to include podiatry, neurology, dermatology, and rheumatology.

It is important not to discount the contribution of cold injury to a veteran patient's symptoms, even when other conditions may exist, which also could explain the symptoms. Enrollment in VA Primary Care Programs should be encouraged by VA medical care practitioners. Treatment recommendations include foot care including involvement of podiatry for patients felt to be at high risk for foot complications, smoking cessation, control of lipid disorders, weight control, control of hypertension, and general preventive medicine measures appropriate for the age of the patient, (i.e., immunization against influenza and pneumococcus).

Medications such as tricyclic antidepressants, specifically amitriptyline in a nocturnal dose, or dibenzeline may be effective and offer help in controlling pain.

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Non-Freezing Cold Injury

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Summary

Non-freezing cold injury may occur when peripheral tissues, almost always the feet, are exposed to cold and/or wet conditions, but tissue fluids do not freeze. Very common among infantry soldiers, it typically produces only mild symptoms during exposure, but profuse and chronic problems for many years afterwards. Sequelae are protean, including cold sensitization, pain, and hyperhidrosis. Little treatment is effective, and sympathectomy is discouraged.

Introduction

Whilst cold injury in general has a long military medical history, it was not until the First World War that non-freezing forms were distinguished from those in which freezing of tissues had occurred. Even as recently as the Second World War and the Korean War, terminology and field diagnosis were confusing, for instance, lumping most cases of non-freezing cold injury (NFCI) with some of freezing cold injury (FCI) under the term “cold injuries, ground type.” What is clear is that NFCI has accounted for very large numbers of casualties in most infantry wars fought in cold-wet climates, including the First World War, the European and other Theatres towards the end of the Second World War, and most recently the Falklands Conflict. Conditions usually taken to imply NFCI include “trench foot” and “immersion foot,” although in strict historical usage the former should be applied to very severe mixed injuries resulting in massive tissue destruction.

Clinical and laboratory studies of NFCI have increasingly shown its overlap with similar conditions. These include "paddy foot," described in soldiers whose feet have been immersed in warm water for long periods, and "shelter limb," which occurred in Londoners taking refuge in the Underground system during the Blitz, whose feet were neither wet nor cold, but remained dependent and immobile for long periods. There are also remarkable similarities with conditions including "deprivation hands and feet," which appears to be NFCI in infants, and "reflex sympathetic dystrophy," a relatively recently described group of disorders involving chronic pain and vascular instability following diffuse trauma. In view of this, it is perhaps best to consider NFCI as part of a spectrum of diseases which are marked by ischaemia at the time of the primary injury, hyperaemia on cessation of that insult, and chronic neurological and vascular consequences – "ischaemia, hyperaemia, neuro-vascular" or IHNV syndromes.

In distinction to FCI, and in common with other IHNV disorders, the early evolution of cases of NFCI is usually unimpressive, and often even sub-clinical; this contrasts with the long-term consequences, which are frequently sustained and more severe. Even when victims have been provided with a high standard of accessible medical care at the time of the primary injury, it is usual for them to present many weeks, months or even years afterwards, complaining of sequelae. The clinician may then be posed the problem of trying to reconstruct dimly-remembered events from the distant past in an effort to establish whether current signs and symptoms result from NFCI, or whether they reflect a more conventional Raynaud's disorder.

Definition

Non-freezing cold injury is defined as being an injury of peripheral tissues resulting from exposure to cold and/or wet conditions, such that the tissue fluids do not freeze at any time.

In cases where an adequate history can be obtained, the key diagnostic criterion is of a sensory neurological disturbance during the period of exposure. This usually amounts to local anaesthesia, and is distinct from sensations of prolonged or extreme cold.

Occurrence

NFCI is most commonly reported in military personnel who are exposed to suitably cold-wet environments for sufficiently long periods, typically infantry soldiers in static positions, such as in defensive trenches. Surveys undertaken of personnel involved in the Falklands Conflict showed that front-line personnel almost invariably sustained NFCI, whilst it was

much less common in headquarters, logistic support, and similar staff; however rear echelon personnel working in water and mud for prolonged periods were more liable, and anyone who becomes soaked during an amphibious operation is clearly at risk. Certain ethnic groups, particularly Afro-Caribbeans, show increased incidences, as do tobacco smokers. There is insufficient information to comment on gender differences, although the low-grade peripheral vascular problems common in women suggest that they should be more prone.

Civilian personnel do suffer from NFCI, but seldom present; when they do, the absence of overt primary injury and the nature of sequelae often cause confusion. In the extreme, such patients may have had long careers following a relentless progression from vascular surgeon to neurologist, ending with psychiatrist. Establishing a physical diagnosis may then result in immense relief to the patient, and restoration of self-esteem, even though they have been warned that little treatment is available for the condition. NFCI may be not uncommon in mountaineers, participants in water-sports, divers, and year-round outdoor workers.

The feet are almost universally the affected periphery, although occasionally the hands may be involved. Anecdotal reports of NFCI to the male genitalia appear not to have been investigated. Whilst exceptional injuries may occur to any area of skin, non-freezing injuries of the face or ears seem very rare indeed, in contrast to FCI.

Clinical Course

Although the grading of cold injuries by severity is becoming increasingly deprecated, the progression in time of NFCI is usefully divided into stages. Most generally accepted are those originally employed by Ungley, in his classic description of immersion foot during the Second World War. It is strongly recommended that this staging is adopted for all descriptions of NFCI.

Stage One: Ischaemia

Prior to the start of the first stage, the periphery feels cold, but sensation and function remain essentially intact. This stage is marked by loss of sensation in the affected part, to a varying degree. The majority of patients admit to complete loss of feeling in the most distal extremity (toes), although some employ more colorful and distinctive descriptions, including the feeling that their feet were made of cotton wool, or that they were “wearing someone else’s feet.” The latter phrase is almost characteristic, and neatly summarizes the

consequences of loss of proprioception as well. If the affected part is examined at this time, it is very cold indeed, but unfrozen (e.g. there is no ice in the socks), white, and apparently ischemic. This stage ends once rewarming commences; such rewarming may be as little as that occurring during massage and routine foot care, in which case the feet may cycle between stages one and two several times before final rewarming.

Stage Two: Early reperfusion

Once rewarming has started, the periphery undergoes transition from ischaemia to hyperaemia, exhibiting a fleeting intermediate which, although rarely observed, is distinctive. The return of color starts with a blue-mottled appearance, with swelling beginning, but rarely prominent. Tissue temperatures start to rise, and that rewarming is accompanied by pain in the face of the numbness remaining from stage one. In some more severe cases, the skin remains wet, as if from profuse sweating. This stage ends once hyperaemia commences, which is normally within a few minutes or hours of starting rewarming.

Stage Three: Hyperaemia

The most overt signs of NFCI are paradoxically the hyperaemia, swelling, and pain which ensue soon after the extremity is rewarmed. As tissue temperatures continue to rise during rewarming, the skin flushes and remains pinker than usual for the remainder of this stage. Pulses become full and bounding, and slowed capillary refill can be demonstrated (rest a fingertip on the skin to blanch it – on removal, the blanching is slow to disappear). Early swelling which may have appeared during stage two continues to grow, to the point where the patient may be unable to wear conventional footwear. Pitting oedema is not normally present, although the most severe cases may develop blistering not unlike that of FCI.

Early loss of sensation usually remains, to be accompanied by pain, which may be exquisite and severe. In milder cases, the pain is mainly nocturnal and confined to the region of the metatarsal heads, under the sole of the foot, and may mimic metatarsalgia. This commonly prevents or disrupts sleep, and is exacerbated by contact with bedclothes and footwear. More severe pain is uncommon, but when it does occur it may be unremitting and resistant to all conventional analgesics (including narcotics, which simply remove the distress which the pain was causing), although it can be abolished by regional analgesia such as spinal or epidural administration of local anesthetics. At its worst, such pain can be relentless, and last for several weeks.

Other sensory abnormalities are less common, but distinctive when they do occur. Some complain of formication, or other similar dysthesiae. Painful paraethesiae are more common but usually transient. Conventional assessment of gross motor function and reflexes are normal.

The hyperemic stage usually lasts several days or weeks, and gradually gives way to the final stage.

Stage Four: Sequelae

For an apparently minor initial injury, NFCI is notorious for its severe and long-lasting consequences. Long-term follow-up of small numbers of patients from the Second World War and many more from recent conflicts suggests that some suffer from life-long sequelae, whilst others recover spontaneously from three months to 12 years after the injury. Resolution is unpredictable, although serial thermography can provide tentative prognosis.

The most common and significant consequence is the symptom of cold sensitivity, which is an indicator of the presence of cold sensitization. Patients complain of an increased sensitivity to cold exposure in the cold-injured extremity, in particular, that once it has become cold, it is very slow to rewarm. It is not uncommon for a mild cold stress (such as immersion in 15º C. for two minutes) to require more than six hours of exposure to warm air before the patient feels that the toes have fully rewarmed. Objective evidence of such protracted vasoconstriction has been provided by both infra-red thermography (see below) and laser Doppler blood flow measurement. As a consequence, those who are cold sensitized may be unwilling to go outdoors during the winter, may require to heat their homes excessively, and sometimes even during the summer, and undergo social and occupational restrictions. Although sometimes described as a secondary Raynaud's condition, it is sufficiently distinctive to accord it the name of "cold sensitization." A typical and almost diagnostic remark is that a sensitized male's female partner complains of his cold feet in bed, rather than him complaining about hers.

Pain and other disturbances of sensation (ranging from numbness to hyperaesthesia) are also common, and may be associated with cold sensitization and episodes of cooling. Patterns are very variable, and range from random shooting pains higher in the foot or ankle, to dull aches on exposure to the cold. They may mimic causalgia, and some patients appear to be suffering from allodynia. Partial or complete loss of sensation usually resolves slowly, but certain modalities may never recover. Two characteristic instances are a rare permanent loss of proprioception, which in turn alters gait and may severely limit walking, and almost complete loss of warm thermal sensation. The latter can be confirmed by the measurement of thermal sensory thresholds.

Many who have suffered from NFCI complain of excessive local sweating, or hyperhidrosis. The high evaporative heat loss from affected feet, coupled with profound resting vasoconstriction, can lower skin temperatures well below ambient, down to as low as 22º C. when conditioned to 30º C. air. Sustained sweating leads to the accumulation of sweat within footwear: this causes social problems because of odor, can rot leather footwear (some sufferers have to throw their shoes away every three months), and may lead to further NFCI. In contrast, the skin may become dry and cracked, particularly along skinfolds, with fissures opening and weeping periodically. Stubborn, deep-seated and recurrent fungal infection may take hold, aided by the moist environment resulting from hyperhidrosis. This can affect the nails too, leading to thickening and in the worst cases onychogryposis. Other patients complain that they shed and regrow toe nails two or three times a year.

Other sequelae are more unusual, and may include joint involvement similar to "frostbite arthritis," although this does not appear to have been studied in those who have had NFCI alone. The most severely injured may undergo amputation and suffer problems common to those with the worst freezing injuries.

Special Investigations

Taking a careful history and performing a thorough medical examination should enable the non-specialist physician to arrive at the correct diagnosis in the great majority of cases. Equivocal findings, disputes, and when there is a need to assess change (perhaps in response to treatment, or to offer a prognosis) merit the greater rigor of investigation. Although there is as yet no single measure which can act as a diagnostic criterion, two techniques have shown themselves to be of value in assessing cold sensitization and thermal sensory impairment.

Assessing cold sensitization

Many different techniques have been employed to try to demonstrate the prolonged vasoconstriction which results from exposure to a cold stress. For a method to be successful, the stress must be sufficiently mild as to avoid causing too many false positives, the method of assessing change must be reproducible, and the whole test conducted in an environment warm enough to support resting vasodilatation in normal subjects.

The current method used at INM has been developed from the original work of Golden, Gallimore and Francis in 1982. Testing is conducted in an environmental chamber providing still air at a dry-bulb temperature of 30º C., in which patients are rested recumbent for

at least 30 minutes before starting. A control infra-red thermogram is then taken, of the sole of an affected foot (or palm of hand). The extremity is put into a plastic bag (which keeps it dry during immersion) and plunged into a waterbath maintained at 15º C. for two minutes. On removal, the plastic bag is discarded, and a second thermogram taken. Five minutes after removal from the water, the third and final thermogram is taken.

Normal and mildly cold-sensitized subjects show warm (surface temperatures above 32º C.) extremities in the first infra-red image, whilst those with moderate and severe degrees of sensitization have digits at around 30º C. and below. The third image is the most discriminatory: in normal subjects, skin temperatures have returned to normal, warm pre-immersion values. The mildly cold-sensitized will only have achieved 29º–30º C., moderate cases around 27º C., and severe cases 25º C. and below. The distribution of temperatures usually gives insight into the digits which are worst affected, and may sometimes show demarcated deficits, although these are more usual in FCI, vibration injuries, etc.

The requirement to use a tightly-controlled environmental chamber limits the use of this technique to specialist centers. Variant methods have been used by some workers in their attempts to perform assessments elsewhere; some have focussed on trying to follow cold-induced vasodilatation during longer cold exposures, whilst others have tried short periods of alcohol spraying. Although these may be of value in experienced hands, they can also produce very high false positive rates, and are not as easy to interpret as results from the INM protocol. Other methods of assessing blood flow have been used; Francis tried photoplethysmography, whilst Oakley has more recently performed many studies using laser Doppler systems, but they are more demanding and only of value as research tools.

Assessing sensory impairment

Anecdotal reports of impaired warm sensation (see above) were married in 1986 with Fowler's development of simple apparatus to measure thermal sensory thresholds in those with diabetic peripheral neuropathy. Oakley demonstrated that those with NFCI of the feet commonly had unrecordably high (> 6º C.) warm sensory thresholds in the toes, whilst cool thresholds were little altered (< 3º C.) from normal. Since then Fowler's Middlesex Hospital Thermal Testing System has been used to assess those presenting with NFCI. Although it is not a direct measure of cold sensitization, this differential pattern of damage remains a common objective finding in stage four. Because the measurement of thermal sensory thresholds is much less demanding of facilities, and several commercial systems are available and in use in neurological departments worldwide, it is a more accessible means of investigation than infra-red thermography.

Treatment

The very limited understanding of the pathophysiological mechanisms responsible for NFCI and its sequelae have severely limited its treatment. Current understanding focuses on three main causes of tissue damage: exposure to cold, prolonged ischaemia accompanying that exposure, and the action of free radicals during reperfusion. There are as yet no proven treatment regimes for any of these modes of injury.

Acute management

The critical difference in management between FCI and NFCI is that non-freezing injuries should only ever be rewarmed slowly. Laboratory and clinical evidence has shown that the rapid rewarming preferred for freezing injuries will at the very least result in more severe symptoms and signs during stage three, if applied to non-freezing injuries. Beyond that, management is conservative, with topical anti-bacterials, avoidance of further trauma including any further cold exposure, and early mobilization.

Pain relief is usually the greatest problem during stage three. Mild analgesics and non-steroidal anti-inflammatory drugs are usually completely ineffective; as remarked above, even narcotic analgesics do not alleviate the pain, but just affect the reaction to pain. Oakley found that nocturnal doses of quinine salts (e.g. quinine sulphate 200 mg rising to 400 mg hs) were effective, but subsequent experience has been unfavorable. More recent approaches have been based on amitriptyline (50 to 150 mg hs) which does afford most patients some degree of relief without significant side-effects. It can also be used in those suffering residual pain in stage four.

Amelioration of sequelae

Once the hyperaemia of stage three changes into the vasoconstriction of stage four, the administration of vasodilators may have a sounder rationale. Unfortunately, experience has shown that results are disappointing. The only approach which has so far shown any promise is the long-term use of sustained-release preparations of nifedipine. These need to be given for several weeks or months before there is demonstrable benefit, but side-effects are sufficiently common and significant that this is of no use in those remaining in the military, or otherwise leading an active life. Thymoxamine has not been of any value.

There is much controversy surrounding the use of sympathectomy. Whilst it can undoubtedly lead to short-term improvements, after six months most patients have worse symptoms than before. It also lacks any sound pathophysiological basis. Surgical sympathectomy therefore should never be considered, and chemical techniques should be avoided.

Keeping the feet warm and dry, through the use of suitable footwear, remains the mainstay of long-term treatment. Military and other personnel who want to return to cold and wet environments should be strongly discouraged from doing so until symptomatic cold sensitization has resolved. Serial thermographic assessment can be particularly valuable in deciding when it is safe to allow re-exposure, although the severity of symptoms does not always accord with the assessed severity of sensitization. Recurrent minor injury is common, and may result in the most severe degrees of cold sensitization; FCI may also occur in those who are cold-sensitized, readily causing massive tissue loss and high levels of amputation.

Prevention

Although NFCI can be postponed by good foot care and hygiene, and careful choice of footwear, fieldcraft and equipment appear unable to overcome fundamental physiological limitations to cold/wet exposure. Much of the success achieved in prevention has resulted from measures such as troop rotation, which were first applied during the First World War.

Training injuries are a particularly intransigent problem. The arguments for training realism might need to be tempered with increased risk of NFCI. On the other hand, the value of a very high standard of fieldcraft, as can only be achieved by long and demanding training, is also apparent. Provided that the objective is always training and not selection by natural wastage, better training could still result in less severe and obtrusive NFCI.

Future Developments

Efforts continue to try to unravel the complex pathophysiology of this protean group of conditions. Recent hypotheses that cold sensitization results from sympathetic denervation supersensitivity have not been borne out by the latest research. Instead, attention is being focussed on changes in the vascular endothelium, and in trained reductions in peripheral perfusion capacity. Experimental models have shown that small local blood vessels are quite severely damaged following NFCI, and groups are now examining neuro-endothelial function. It is also possible that cold sensitization is the reverse of the neovascularisation seen as a result of physical training in the heat, leading to a loss of peripheral vessels. If this is the case, sending patients to the tropics during stage three may prevent the appearance of cold sensitization.

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Journal Article Summary*

Reference: Sequelae to Cold Injury in One Hundred Patients: Follow-up Study Four Years After Occurrence of Cold Injury. *Journal of the American Medical Association. 1957 Apr;163(14):1203-1208.*

Authors: *Lieut. Col. Joseph R. Blair (MC), U.S. Army; Richard Schatzki, M.D., Boston; Lieut. Col. Kenneth D. Orr (MC), U.S. Army*

Introduction

About 5,600 victims of cold injury were evacuated from Korea during the winter of 1950-1951. Most of these patients were considered to have cases of frostbite rather than trench foot. A group of 100 of these patients, selected as a representative sample of the total number evacuated from Korea, were observed on average six days after the occurrence of their cold injury. Additional studies carried out four years later on these 100 patients form the basis for this report.

Material and Methods

Throughout this study, cold injury has been classified into four degrees of severity: first degree involves only hyperemia and edema; second degree, hyperemia with vesicle formation; third degree, necrosis of the skin and subcutaneous tissue; and fourth degree, complete necrosis and loss of tissue.

Of the 100 young men with cold injuries, 89 reported to medical installations where especially complete data, including a history, statement of symptoms, photographs of healed lesions, roentgenograms, circulatory measurements, and other physical findings were obtained.

Symptomatology

The six most common symptoms of these former patients were cold feet, pain, excessive sweating, numbness, abnormal color, and symptoms in joints. All symptoms, with the exception of excessive sweating, were much more severe during the winter months than during the summer months. The pain and cold feet experienced in cold weather were the most bitter complaints expressed by the patients with frostbite.

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Clinical Observations

The report on clinical observations was restricted to those 50 patients examined and studied personally by the authors. All symptoms were more severe than organic physical findings would indicate, but to what degree these complaints may have a psychogenic basis could not be determined.

In third- and fourth-degree frostbite cases residual pathology could always be detected four years after the injury. The most common physical findings were tissue loss, scars, abnormal nails, hyperhidrosis, abnormal color, and stiff joints. Tissue loss did not occur in second-degree cold injury, but did occur in all fourth-degree cases. Third-degree injury was a tissue defect (rather than a loss of a part), a "punched-out" area resulting from sloughing of the overlying eschar and subsequent granulation of the lesion. Scars were absent in patients with ill-defined conditions of the feet and could not be detected in any patients with second-degree frostbite. Scars were usually present at the site of injury in cases of third-degree injury and always extensive in cases of fourth-degree cold injury.

One of the most characteristic physical findings was that of abnormal toenails. In more than two-thirds of the cases in which the injury was severe enough to involve full-skin thickness (third-degree of severity), the nails were markedly thickened, heavily ridged, contracted at the front margin, and the nail beds often had a cyanotic appearance. No abnormalities of the nails, however, were observed in cold injuries of less than third-degree severity.

The determination of hyperhidrosis or excessive sweating was qualitative. One question that could not be answered was whether the excessive sweating was a cause or effect of cold injury. Because a hyperhidrotic person is a likely candidate for cold injury, the hyperhidrosis may have been present before the frostbite occurred and a major factor in causing the frostbite.

The finding of abnormal color was different for Caucasian patients as compared to the Afro-American patient. Abnormal color was primarily an erythrocyanotic appearance in Caucasian patients and probably associated with peripheral circulatory disturbance. In Afro-American patients abnormal color was caused by skin depigmentation which occurred in all patients with third- or fourth-degree frostbite.

Damage to the joints was more frequent and more extensive in patients with more severe cold injury. In fourth-degree frostbite some of the stiffness of the joints could be attributed to heavy, contracted scar tissue surrounding the joint rather than pathology within the joint itself. In patients with pathological findings in the joints of previously frostbitten extremities, there was no evidence of arthritic changes or changes in the joints elsewhere in the body.

Roentgenographic Studies

None of the patients showed late occurrence of osteoporosis four years after cold injury. Trabecular changes as noted in a few of the four-year films consisted of coarsening of the trabeculae as seen in patients with changed statics. In those cases where a complete follow-up was possible for soldiers with partially mutilated terminal phalanges, it appeared that a loss of overlying soft tissue and exposure to air caused the mutilation. There was no convincing evidence that cold injury itself had caused mutilation of bone without loss of the overlying soft tissue. Of particular interest were the punched-out defects of the surfaces of the joints of toes and fingers. An exact comparison between the degree of frostbite and the occurrence of lesions of the joint was not possible.

Comment

In cases of first- and second-degree injury, there were almost no physical findings present in patients with cold injury after four years. However, when cold injury is full-skin thickness (third- or fourth degree of severity), certain findings become evident and relatively constant:

1. an area of scar formation and skin thickening;
2. abnormal condition of the nail;
3. tissue deficit or actual tissue loss in fourth-degree injuries;
4. color changes, especially depigmentation in Afro-American patients;
5. joint pathology, demonstrable by physical examination and roentgenograms; and
6. growth disturbance when cold injury occurs before closure of the epiphyses.

Summary

Symptoms after frostbite, in order of frequency, are excessive sweating, pain, cold feet, numbness, abnormal color, and symptoms in the joints. Characteristic physical findings are tissue loss and scarring, abnormal nails, discoloration and depigmentation, hyperhidrosis, and joint abnormalities. Roentgenographic changes include early transient osteoporosis, mutilation of terminal phalanges, cyst-like defects of the bone near the joint surfaces of fingers and toes, early transient periosteal new bone formation, and growth disturbance in patients with open epiphyses.

Journal Article Summary*

Reference: Frostbite Arthritis. *CMA Journal. 1981 Nov;125:1128-1130.*
Author: *Robert J.R. McKendry, M.D., F.R.C.P.(C)*

Introduction

Delayed damage to bones and joints is a recognized sequela of frostbite. Harsh Canadian winter conditions can cause frostbite and the increasing popularity of winter sports has increased the risk of this injury. Frostbite arthritis will develop many months or years after the original injury in approximately half of the cases. The characteristic clinical and radiologic features, which resemble those of osteoarthritis, are illustrated in this case report.

Case Report

A 29-year-old man had suffered a frostbite injury to his hands at the age of 16 after remaining outside for four-and-a-half hours on a night when the temperature was -27º C. When he was found, semiconscious, and taken to a local hospital, he underwent skin grafting to his hands because the frostbite injury was so severe and extensive.

The patient's hands were essentially free of symptoms for seven years until swelling and stiffness developed in the proximal interphalangeal (PIP) joint of the right second finger, followed by similar symptoms in the PIP joint of the left index finger. Six years after these symptoms, his hands appeared osteoarthritic with an unusual degree of flexion of the terminal phalanges. Xeroradiographs taken at this time showed flexion of most of the distal interphalangeal joints, loss of bone in some distal phalanges and subchondral cysts, but relatively little narrowing of the joint spaces or evidence of adjacent osteosclerosis.

At the time of this report (fourteen years after the injury), the patient had very little joint pain, but described interphalangeal joint stiffness and some loss of dexterity. His hands were unusually sensitive to cold: exposure produced a purple discoloration and a feeling of discomfort. Laboratory findings, including the complete blood count and the erythrocyte sedimentation rate, were normal; tests for rheumatoid factor gave negative results.

Discussion

The initial phase of frostbite arthritis can be categorized according to four degrees of severity. Bone and joint changes may occur weeks, months or year later. The radiologic changes are most pronounced at the distal ends of the digits and may be asymmetrically distributed. Symptoms of arthritis secondary to frostbite seem to vary from episodic attacks of quite severe joint pain to very mild pain and stiffness, as in the case reported here. Other chronic cold-induced sequelae have been described. This patient reported that his hands were so sensitive to cold that they became purple after brief exposure. Theories that have been proposed to explain the pathophysiologic features of frostbite fail to explain why progressive joint damage may occur many years after the initial injury. Both primary osteoarthritis and frostbite of the hands characteristically produce bony enlargement, discomfort and restricted movement of the distal and, to a lesser extent, the proximal interphalangeal joints. Although there are substantial differences between these two conditions, the similar clinical and radiologic features suggest the possibility of common pathophysiologic mechanisms. Perhaps chondrocyte damage, due to cold injury in frostbite arthritis and to biochemical or mechanical factors or both in primary osteoarthritis, is responsible for the similar clinical and radiologic features.

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Journal Article Summary*

Reference: Squamous Cell Carcinoma of the Heel Developing at Site of Previous Frostbite. *Journal of the Royal Society of Medicine. 1982 Sept;75:715-718.*

Authors: *C.G. Rossis, M.D., M.C.; A.M. Yiacoumettis, M.D., F.IC.S.; J. Elemenoglou, M.D. (Plastic and Reconstructive Surgery Unit, 401 Military Hospital, Athens, Greece)*

Introduction

During the Second World War the Greek army fought a defensive war against the invading armies of Italy and Germany in the mountainous region of Northern Greece. Many soldiers suffered severe frostbite. A rough estimate of the number of amputations due to frostbite lesions alone was approximately 25,000. A number of frostbite victims escaped amputation but, over the years, have borne various sequelae of their healed frostbite, these being mainly scars and contractures. In this article, ten cases of squamous cell carcinoma of the heel previously affected by frostbite are reported.

Methods and Results

Ten patients with similar histories and all veterans of the Second World War were referred with lesions on the heel at the site of previous frostbite. At examination, seven patients had macroscopically fungating tumours and three had ulceration. From the histories, it appeared that five of the seven who presented with fungating lesions had started with ulceration. In the three patients in whom healing had not occurred, the time to referral since breakdown of the lesions was about two years. In the seven patients with recurrent breakdowns, it was difficult to determine how long it had taken for the tumour to develop since their last breakdown.

Each patient had a biopsy of the lesion at the margin of the tumour and, subsequently, an excision. In four cases some bone had to be resected in order to obtain clearance, and in these a local skin flap was used to cover the defect. For the other cases a thick split skin graft sufficed. Histological examination of all specimens showed the tumours to be Grade I and Grade II squamous cell carcinomas. Following surgery there were only minor problems with management of the operated areas. Six patients were followed up for five years after surgery, and four patients for lesser periods.

Discussion

Squamous cell carcinoma of the lower leg is usually the result of chronic skin conditions and persistent irritation. Unless protection is provided, breakdown may result. All the tumours in this series developed on the heel rather than on other frostbitten areas of the foot, which may be explained by the greater pressure and irritation sustained by the heel.

Histology showed no specific changes that could explain the genesis of malignancy of old frostbitten areas. This type of carcinoma is well differentiated and therefore of low malignancy, which suggests that it might be treated less radically than has been recommended by others. Amputation seems unnecessary; lymph node block dissection should be reserved for proven lymph node metastases. In this series, none of the patients developed regional lymph node metastases. The authors concluded that squamous cell carcinoma developing on unstable, previously-frostbitten heels appears to be the result of chronic irritation, is of low malignancy, and should be treated by surgical excision.

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Journal Article Summary*

Reference: Late Neuropathic Sequelae of Cold Injury. *The Journal of Foot Surgery. 1987;26(3):213-216.*

Authors: *Morton I. Altman, D.P.M. and Steven J. Hutton, D.P.M.*

Introduction

Although injuries such as frostbite, trench foot, and immersion foot were a major disability among fighting forces during World War II and the Korean Conflict which prompted a wealth of medical research on this problem during the 1940's and 1950's, very little has been written describing the late sequelae of cold injury. The permanence of symptoms is never specifically addressed in any of these reports and, indeed, an implicit assumption has been made that a gradual improvement in symptoms can be expected.

American soldiers sustaining frostbite, trench foot, or immersion foot during World War II are now entering their 60s. Many are presenting to Department of Veterans Affairs (VA) clinics complaining of persistent symptoms in their lower extremities dating from a previous cold injury. Most of these injuries are now 40 years old. The purpose of this report is to present five patients with chronic, lower extremity, neurologic symptoms that appear directly related to previous cold exposure and to identify a permanent symptom complex as cold exposure neuropathy.

Materials and Methods

Five male patients between the ages of 59 to 66 years old were interviewed and examined at VA Medical Center in Albuquerque, New Mexico. Each was originally injured while on active duty during World War II. Four of the injuries could be classified as trench foot caused by the patient's exposure to prolonged moisture from wet socks and boots at temperatures above freezing. Another of the injuries was an apparent frostbite from prolonged standing and marching in thin foot gear on frozen surfaces. Four of the five cold exposures involved a single episode with each of these occurring during combat conditions. The fifth patient had multiple, relatively short episodes of cold exposure that occurred during
practice drilling on a frozen parade ground. Length of exposure for each patient varied from two to three hours up to seven days.

Discussion

Onset of symptoms in all of these patents could be traced to the time of previous cold injury. The severity of symptoms has not changed appreciably in any of the patients with one exception. Four of the five in this series were troubled by recurring pain in the area of cold injury. The next most common subjective complaint was a sensitivity or intolerance to cold, reported in three of the five patients. Numbness and tingling were reported by two of the five. The only other symptom recorded in more than one patient was a burning sensation (two of five). One notable difference between previously published reports and the small series presented here is the incidence of postinjury hyperhidrosis. Only one patient of five reported this finding, compared with previous reports showing a consistently high incidence.

A qualitative neurologic examination was performed on four of the five patients in an attempt to identify a correlation between abnormal physical findings, severity of exposure, and/or severity of chronic symptoms. Although the size of this series made statistical analysis impractical, the results were examined in an effort to identify any patterns. Based on the length of the exposure, length of hospitalization, and acute signs/symptoms, patients one and three sustained the most severe initial injury. Abnormalities were noted in several of the tests on these patients. However, upon examination of patient four with a relatively short exposure and no hospitalization, abnormal test results were also obtained that were not remarkably different from the two or more severely injured patients.

The comparison of severity of symptoms with severity of physical examination abnormalities also showed no consistent trend. Each patient complained of a different complex of symptoms, making it difficult to classify them in order of severity. None of the patients reported a marked limitation in their activities of daily living because of chronic symptoms, even though a wide range in the severity of injury was noted.

Nerve conduction and electromyographic testing provide the possibility of objective identification of nerve damage. In this series, three of the five patients sustained NCV/EMG testing. Two of the patients demonstrated no abnormality in conduction or muscle innervation. Patient four showed a slowing of conduction velocities, which was interpreted as being consistent with a diabetic neuropathy rather than secondary to cold injury.

All five patients in this series reported onset of symptoms shortly after their cold injury. Four of these five denied any progression of symptoms over the years. Patient four, however, did report an exacerbation of painful symptoms over a recent two month span. His past medical history was positive for adult onset diabetes mellitus. A definite slowing was noted in his NCV studies, consistent with diabetic neuropathy, notable because of the progression of the symptoms. Diabetes must always be considered when attempting to determine the etiology of a cold exposed patient's peripheral symptoms.

Conclusion

Although the investigated population was limited, certain points are apparent based on the findings in this report. The nature of the symptoms and physical signs in each of these patients suggest a neurologic deficit. The severity of these symptoms does not necessarily correlate with the severity of either the initial injury or the physical findings. It appears that the pathology of the sequelae of cold injury, while still debated in the literature, can be considered permanent and without apparent regression, even many years after the original injury. The clinical presentation after injury of the frostbite, trench foot, or immersion foot type is characteristic and can be considered a cold exposure neuropathy.

The neurologic sequelae of these injuries, including pain, numbness, tingling, hyperhidrosis, and cold sensitivity of the extremities are being seen with greater frequency because of large numbers of World War II veterans entering their 60's who are now presenting to VA facilities, clinics, and private office. The need for further detailed work on a much larger series of patients with cold exposure neuropathy should be apparent.

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Journal Article Summary*

Reference: Nerve Conduction Velocity in Human Limbs with Late Sequelae After Local Cold Injury. *European Journal of Clinical Investigation. 1996 Jun;26(6):443-50*

Authors: *A. Arvensen, J. Wilson, L. Rosen*

Introduction

Cold-induced neuropathy may play a dominant role in the long-term sequelae with cold sensitivity after local cold injuries (LCIs). The aim of this study was to determine nerve conduction velocities (NCV) and somatosensory function in the distal part of extremities of subjects with late sequelae after local cold injuries. Possible associations between degree of cold hypersensitivity and nerve conduction were investigated.

Patients and Methods

The authors had registered 40 soldiers who suffered LCI during their service in the Norwegian army. In a previous study, they found that thirty-eight of these soldiers had sequelae with hypersensitivity to cold; thirty-one were willing to participate in this study three to four years after the primary cold injury. Somatosensory functions were assessed and NCV and motor distal delay (MDD) were measured in the limbs of the subjects. NCV measurements were performed in 24 lower and 16 upper extremities.

Results

Of those subjects in the lower extremity group with a disturbed cold sensation, three had a delayed perception, four were not able to differentiate between cold and hot water, and another four experienced a hot or burning sensation. Among those with disturbed perception of the warm stimulus, three felt a delayed, one a paradoxical and seven an indifferent sensation. In the upper extremities four had a disturbed sense of cold. One had a delayed response, two felt a warm sensation and one was unable to differentiate between cold and heat. In response to heat stimulus, one felt cold and three could not differentiate. Oedema was not seen in the late phase in any of the extremities. Joint movements and tendon reflexes were normal. One reported decreased power of finger flexion, which was confirmed by the examiner.

NCV was related to degree of overall subjective complaints quantified by means of a visual analogue scale (VAS). Lower extremities: MDD in previously injured limbs was pathologically increased compared with controls. Motor (MNCV) and sensory conduction velocity (SNCV) were significantly decreased, but to a lesser degree. Upper extremities: MDD in the ulnar nerves of patient limbs was increased, but this difference did not attain significance on the right side. MNCV in the ulnar nerves of the forearms was significantly decreased. MNCV in the median nerves was also affected, but not significantly reduced
in the median and ulnar nerves compared with control subjects. SNCV in the forearms showed the same tendency, but differences were not significant. NCV in the uninjured limbs of eight patients were normal.

Discussion

This study's data show that, three to four years after the primary cold injury, NCV was still markedly decreased in the nerves of previously cold-injured limbs. Disturbed nerve function could represent a predisposition; however, these patients had no symptoms of cold intolerance or sensory disturbances before the injury. NCV was decreased compared with uninjured young men of the same age, and normal nerve conduction was found in the unaffected extremities. Decreased NCV was most prominent in the lower extremities. It was somewhat puzzling that a minor cold injury in fingers or toes may lead to impaired NCV proximally in the seemingly uninvolved forearm or calf respectively.

NCV recordings mainly reflect the spatial summation of action potentials from large myelinated fast-conducting fibres. However, hyperhidrosis, dystrophic skin changes and especially cold pain, as seen in this study, indicated damage also to thin myelinated and non-myelinated C-fibres. Altered or absent sense of temperature, pain and touch and increased latency of distal nerve segments (MDD and SNCV, distal segment) indicated a peripheral nerve damage. NCV and the clinical examination revealed that different types of nerve fibres and not only the thick myelinated fibres were affected.

Increasing VAS associated with decreasing NCV might have been anticipated but, unexpectedly, the opposite was found: a significant positive correlation of clinical importance. This is probably due to a functional disability of the more extensively damaged nerves. Conversely, the more functional nerves have greater pain-transmitting properties.

In the lower extremities MNCV and SNCV were significantly impaired. In the forearms, however, differences in MNCV and SNCV were non-significant compared with control subjects, with the exception of MNCV in the ulnar nerves. However, SNCV in the hands

was significantly decreased compared with controls. A more profound cooling of the calves than of the forearms may explain these findings: the lower extremities are especially prone to deep cooling during periods of immobilization, especially in humid conditions during military manoeuvres. Footwear and weightbearing may impair skin microcirculation and local thermoregulation by mechanical pressure. Additionally, deep nerve temperature depends on blood flow and heat exchange in the limb.

Within the control group cold-induced vasodilatation (CIVD) started sooner in the fingers than in the feet during ice water immersion. A more rapid or long-lasting CIVD in the forearms would maintain a higher nerve temperature than in the lower legs and consequently protect more effectively against cold injury. During hypothermia CIVD will disappear. Some of the patients may have been mildly hypothermic during the military manoeuvres, although for obvious reasons this was not registered. Consequently, in the pathogenesis of hypothermic nerve injuries, both environmental factors and individual factors may partly explain the great individual variations in NCV observed in this study.

The observed difference in MDD between the right and left ulnar nerve is difficult to explain. The ulnar nerve may be more susceptible to cold injury than the median nerve owing to a more superficial anatomical location. Additionally, structural or functional differences between the right and left hand in right-handed persons may render the right nerves less vulnerable. Finally, a false significant difference (type 1 error), despite an acceptable power of the study, cannot be excluded.

In conclusion, NCV, distal latencies and somatosensory functions were impaired in the extremities three to four years after the primary LCI. In the lower limbs SNCV and MNCV were significantly decreased, possibly as a result of a deeper and more pronounced cooling in the calves than in forearms. Peripheral sensory nerve function was impaired, but the thick myelinated motor fibres were also affected. NCV and distal latencies were normal in the uninjured limbs. Reduced NCV in the extremities seems to be consistent with cold-induced neuropathy after frostbite and trench-foot. NCV measurements can provide objective findings in a group of patients with few or no overt clinical symptoms.

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Journal Article Summary*

Reference: Induced Vasodilation as Treatment for Raynaud's Disease. *Annals of Internal Medicine. 1982 Nov;97(5):706-709.*

Authors: *Jared B. Jobe, Ph.D.; James B. Sampson, Ph.D.; Donald E. Roberts, Ph.D.; William P. Beetham, Jr., M.D.; Natick and Burlington, Massachusetts*

Introduction

The authors explored the effects of Pavlovian conditioning on increasing blood flow as measured by an increase in the response of digital temperature to cold in persons with primary Raynaud's disease. To elicit increased temperatures, an unconditioned stimulus of warm water (43º C.) to the hands was paired with a conditioned stimulus, exposure of the whole body to cold (0º C.). After repeated pairings, exposure to cold air alone should elicit vasodilation in the hands (conditioned response).

Methods

Thirty-six male and female volunteers from the local population and other laboratories were studied. All were interviewed, medical histories taken, and physical examinations given. Eight persons with Raynaud's disease and seven normal persons each received 27 simultaneous pairings of hand immersion in warm water (43º C.) for ten minutes with exposure of the whole body to cold (0º C.). A second group of seven normal persons and nine persons with Raynaud's disease received no treatments. The temperature response of the fingers to cold air before and after treatment (or no treatment) was used as the dependent variable. Treatments were given on Mondays, Wednesday, and Fridays, three per day for three consecutive weeks. The week before treatments began, all subjects were tested to determine their digital temperature response to cold by being exposed to a single, ten-minute period of cold. The observed response of this cold test was the digital temperature recorded during the last minute of exposure. The cold test was repeated the week after the conclusion of treatments to establish changes in the response to cold exposure of both treated and untreated groups.

Results

Subjects with Raynaud's disease who received treatments showed significant increases in digital temperatures (2.2° C.) during the cold test compared with the values of untreated subjects with Raynaud's disease ($p < 0.01$). Normal subjects who had received treatments showed no difference from those who had not. Digital temperatures of subjects with Raynaud's disease after treatment increased to levels approaching those of normal subjects although they showed lower digital temperatures during initial exposure to cold ($p < 0.01$).

Treatments appeared to result in long-lasting benefits. The most frequent comments were that hands returned to normal much faster after episodes of vasospasm and felt much warmer. Others stated that they no longer wore gloves to shovel snow, to drive on cold days or to go outdoors in the cold. Still others reported that their attacks now occurred less frequently and were less severe.

Several subjects were retested up to four months after the treatments. All maintained improvement in their response to cold. Follow-up questionnaires were sent to all subjects with Raynaud's disease nine to 12 months after they completed the program. Nine of the 16 subjects who returned the questionnaire reported continued positive effects of the treatment. Five subjects stated that their attacks were less severe; six, that their attacks were less painful; and seven, that recovery time from an attack was shorter. One subject reported she had recurrent superficial ulcerations on her fingertips consistently for five consecutive years, but that she had none since the treatments.

Discussion

Persons with Raynaud's disease showed substantially lower digital temperatures at room temperature than normal persons. As a result of simple Pavlovian therapy, persons with Raynaud's disease showed beneficial increases in digital temperature response to cold that approach those of normal subjects. These results suggest that classical conditioning therapy is a feasible alternative to the more traditional medical management techniques. Conditioning therapy avoids the problem of side effects of drugs and the loss of nerve function secondary to sympathectomy.

Follow-up data indicate that the treatment has long-lasting effects, and if periodic reinforcement conditioning appears necessary, it can likely be accomplished at home using naturally occurring ambient cold and hot tap water. Although treating the hands did not appear to result in improvement to the feet (based on subjective comments), there is no reason to believe that the feet could not be as easily conditioned as the hands, either separately or simultaneously.

These results compare favorably with results obtained using biofeedback with several important advantages:

1. subjects with Raynaud's disease showed improved digital temperatures during exposure to cold of 0º C., whereas most biofeedback studies report results under relatively mild temperatures;
2. improvements in episodes of Raynaud's disease reported in biofeedback studies are largely subjective, with little or no objective measures to verify positive results in response to cold;
3. biofeedback usually requires "suggestive" subjects implying a placebo effect; and,
4. Pavlovian conditioning seems to be more applicable to a wide range of patients and has a more prolonged effect than other therapies.

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Instructions for Home Treatment of Raynaud's Disease

Provided by: Murray P. Hamlet, D.V.M., Chief, Research Support Division, U.S. Army Research Institute, Natick, Massachusetts

Note: A decision to try this treatment should be based on discussion with a physician.

You will need:

- Two insulated containers (such as Styrofoam or another type of cooler).
- One cooler will be placed in a heated indoor room and the other in a cold outdoor location such as a porch, balcony, or garage where your torso can get quite chilled while your hands are immersed in hot water.
- Thermometer
- Hot tap water to fill your containers (104-108º F. OR 41-43º C.). *Be sure to keep the water at the correct temperature.*

You should wear:

- Normal indoor clothing such as a long sleeve shirt and pants. Overdressing will **defeat** the purpose of the treatments; however, ***avoid exposure to severe cold!*** The torso must get uncomfortably cold.
- Gloves ***(Be sure to protect your hands at all times!)***

Remember these important points:

- **Do not** perform the treatment every day. Do the treatment cycles every other day.
- You will need to complete about 50 cycles total.
- You will need to complete three to six cycles per day, every other day. It is best to do the cycles sequentially, that is, after one cycle wait approximately 20 minutes and then begin another cycle.
- Some people respond more quickly and completely than others.
- Some require training yearly, but most have relief of symptoms for a number of years.
- The training may not be effective in autoimmune patients.

Now you are ready to begin. This is one cycle:

- Begin at your warm indoor location. Re-warm your hands in your container of hot water for two to five minutes. Dry your hands and put on gloves.
- Go to your cold outdoor location. Remove gloves and immerse both hands in hot water for ten minutes. Dry and cover your hands and return indoors.
- Place hands in hot water again until torso has warmed up (two to five minutes).

Independent Study Test for CME Credit

Directions

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   Medical Forum, Suite 500
   950 North 22nd Street
   Birmingham, AL 35203-5300

   If you have attained a passing score of 70% or higher, a certificate will be mailed to you approximately 6-8 weeks after your test has been graded. The test may be retaken.

   Please note: These Scantron Forms cannot be copied.

   You may also submit the Registration/Independent Study Test Answers and Program Evaluation responses by using the VA website if you have access to Internet Explorer 4.0 or Netscape 4.0 or higher. **For expediency**, you may wish to **register**, take the CME test, and complete the program evaluation **using the VA Intranet**. The address is:

   http://vaww.sites.lrn.va.gov/vhi

   After you take the test, you will receive immediate feedback as to pass or fail. You will be allowed to retake the test one time. Upon passing the test and completing the program evaluation, you will be able to immediately print your certificate according to instructions.

   For extra copies of this independent study or for other VHI Independent Study Modules, please contact your facility education contact person.

Cold Injury: Diagnosis and Management of Long Term Sequelae

Independent Study Test Questions for CME Credit

Using the **Independent Study Participant Registration/Answer Sheet**, please circle the letter corresponding with the correct answer.

1. Freezing Cold Injury (FCI) is:
   a) An injury of subcutaneous tissue and muscle.
   b) An injury to nerve and skin.
   c) A two stage injury involving cellular damage during freezing, and vascular injury during freezing and the warming event.
   d) All of the above.

2. During Freezing there is:
   a) Sufficient heat loss for the ice crystals to form in the extra cellular spaces.
   b) Cellular dehydration as fluid is extracted from the cells following extra cellular ice formation.
   c) Particular damage to the vascular endothelial lining.
   d) All of the above.

3. Non freezing cold injury (NFCI) usually occurs:
   a) Under wet, cold non-freezing conditions.
   b) In stages that are described as pre-hyperemic, hyperemic, and post hyperemic.
   c) When the soldier is immobile, extremities are in the dependent position, and socks and footgear are damp or wet.
   d) All of the above.

4. Post warming pain, swelling, and edema, and neuro-vascular changes of the extremities are found most often:
   a) In superficial frostbite.
   b) In deep frostbite.
   c) In freeze-thaw-refreeze injury.
   d) In non-freezing cold injury (NFCI).

5. Thawing of the frozen extremity is best accomplished by:
   a) Spontaneous warming.
   b) Ice and snow packs with massage of the part frozen.
   c) Oven heat or wood fire heat in bivouac.
   d) Rapid rewarming in warm water between temperatures of 90º to 100º F. (32º to 38º C.).

6. Cold injury sequelae can be:
   a) Transient only.
   b) Long lasting only.
   c) Both of the above.
   d) None of the above.

7. Cold injury sequelae:
   a) Do not change after six months following the initial injury.
   b) Do not involve bones, joints, or ligaments.
   c) Do not result in amputations.
   d) Often increase in number and worsen as the victim of cold injury ages.

8. Post thawing pain and residual pain are most common in:
   a) Freezing cold injury (FCI).
   b) Non-freezing cold injury (NFCI).
   c) Isolated hypothermia.
   d) None of the above.

9. Non-freezing cold injury and freezing cold injury sequelae may include:
   a) Hammer or claw toe deformity.
   b) Volar fat pad loss of the digits.
   c) Permanent nailbed deformity (onchogryphosis) and often fungus infection of the nail area (onchomycosis).
   d) All of the above.

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