Seminar on frostbite*

Chairman:

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Participants (all faculty members except Drs. Knight and Martin):

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DR. HERMANN: This seminar is concerned with frostbite, a constantly recurring health hazard in the Rocky Mountain area. The case for discussion is a complex one, but does illustrate many problems encountered in diagnosis, treatment and prophylaxis of frostbite. Dr. Martin, you were the first physician to see the patient. Could the discussion be started with a presentation of the patient's history and appearance when you first saw him?

DR. MARTIN: The patient, aged 25, went mountain climbing on a Saturday in October. During the afternoon, he and his companion parted. Shortly he became lost in snow and decided to settle for the night. The following morning he attempted to walk out but again became lost. He ate snow but did not have any solid food. He remembers nothing about his actions subsequent to about

3:00 a.m., Monday morning. He was discovered by search parties about 10:00 a.m. on Tuesday morning. When found, he was conscious but not coherent. His fingers and elbows were stiff, but could be passively extended. His feet and lower legs were mottled. His respirations were 18 per minute; heart rate, 50. His blood pressure was unobtainable, as were his peripheral pulses. Rectal temperature was 25 degrees Centigrade. After emergency care, he was transferred to Colorado General Hospital.

DR. HERMANN: Dr. Paton, please tell us about the patient's appearance when you saw him in the emergency room at Colorado General Hospital.

DR. PATON: The patient was conscious but disoriented. Rectal temperature was 29.5 degrees Centigrade, blood pressure unobtainable, and his heart rate was about 65 with auricular fibrillation. The exposed parts of his face were bright red in color, with no areas of impending tissue loss. His hands were scratched and torn, but the color was reasonably normal. The part of his feet and ankles which had been inside his boots were purple and mottled proximally and dead white distally. There was no evidence of capillary filling. Blood chemistries drawn at this time revealed a blood urea nitrogen of 56 and evidence of severe acidosis with an arterial pH of 7.1. His urinary output initially was good, but in the next few hours decreased. Because his oliguria persisted over the next 72 hours, he was dialyzed. After evaluating his frostbitten legs, it was decided to treat this injury by the technic of rapid rewarming.

DR. HERMANN: Dr. Starzl, please discuss the place of rapid rewarming in the treatment of frostbite.

DR. STARZL: Despite enormous experience with frostbite, especially during recent wars, the basic physiology of this form of trauma has been poorly understood. Consequently, there has been ambiguity concerning the appropriate methods of therapy, particularly in the acute lesion.

Recent work has shown that the one single treatment of proved value is rapid rewarming of frozen members, the technic of which is explicit. The injured extremity should be immersed in water which is kept between 37 and 42 degrees centigrade until no further improvement occurs. When this patient's feet were placed in the warm water, the line of demarcation advanced toward his toes, rapidly at first, but as it moved distally,

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the rate of color return became progressively slower. At the time the feet were removed from the warm water, it was apparent that little, if any, tissue loss would occur.

Numerous other remedies, including sympathectomy, vasodilator drugs, heparin, or the systemic infusion of low molecular weight dextran have been recommended for immediate treatment of frostbite. None of these has been proved to be of consistent value.

DR. HERMANN: Dr. Starzl, please say a few words about the place of amputation in treatment of frostbite.

DR. STARZL: The degree of eventual tissue loss is almost always less than that which would be anticipated from the initial examination. Weeks, or even months, may be required before the true extent of tissue loss is accurately known. Therefore, definitive amputation should be delayed as long as possible.

DR. HERMANN: Dr. Paton, one of the intriguing aspects of this case was the generalized body hypothermia this patient evidenced. Would you enlarge on this subject?

DR. PATON: Unfortunately, detailed observations of people with accidental profound hypothermia are scant. The facts that we do know have been accumulated with controlled hypothermia. The major change noted with hypothermia is progressive decreases in the oxygen requirement and the oxygen consumption. For example, at 30 degrees centigrade, somewhat above this patient's temperature when he was first found, the oxygen consumption is only 50 per cent of normal.

Coincidental with progressive decrease in the demand for oxygen is lowering of blood pressure and slowing of pulse. There is also an increasing irritability of the myocardium. Spontaneous respiration usually ceases about 32 to 30 degrees, but in isolated instances as in this patient, respirations may be maintained at lower temperatures. There are progressive electroencephalographic changes with decreasing temperature. At about 20 degrees, all electrical activity in the brain ceases.

It can be seen, therefore, that this patient was extremely lucky in that, although his body temperature had fallen to the range where ventricular fibrillation occurs where spontaneous respiration usually ceases, he managed to survive.

DR. HERMANN: Dr. Holmes, this patient developed renal shut-down as part of his illness. Was this due to the effect of cold per se on the kidneys, or was it secondary to prolonged hypotension?

DR. HOLMES: It is difficult to state whether this patient's renal lesion was a result of prolonged hypotension or of hypothermia itself. We do know that the former condition can lead to lower nephron nephrosis. We also know that, although proteinuria has been associated with exposure to cold, there is little reference in the literature to occurrence of prolonged oliguria or acute tubular necrosis secondary to cold injury. Although induced hypothermia causes reductions in glomeru-

lar filtration rate and renal blood flow, rewarming rapidly restores these values to normal.

This patient was dialyzed in keeping with our policy of early and frequent dialyses in all trauma patients with associated oliguria. He was dialyzed on the third hospital day. By this time, his BUN had risen to 180 mg. per cent. The dialysis produced notable clinical improvement and after going through a brief diuretic phase, the BUN dropped to 18 mg. per cent by the fifth post-treatment day. There was no further evidence of renal dysfunction.

DR. HERMANN: Thank you, Dr. Holmes. Dr. Blount, as a cardiologist with interest in cardiac surgery and controlled hypothermia, would you comment upon this patient's auricular fibrillation and the relationship of cold to the functioning of the myocardium?

DR. BLOUNT: The auricular fibrillation which this patient exhibited when first seen is a well known response of human myocardium to hypothermia. In the human, auricular fibrillation is the first abnormal rhythm manifested with decreasing temperatures. As the temperature drops, this will be followed by ventricular fibrillation and eventually cardiac standstill. Somewhere between 28 to 30 degrees centigrade, most patients will develop auricular fibrillation. With temperatures below 28 degrees, ventricular fibrillation supervenes with increasing frequency. In this patient, as has been the case using controlled hypothermia, the cardiac rhythm spontaneously assumed a normal beat, coincidental with rewarming.

Because of the possibility of ventricular fibrillation during hypothermia, a continuous visualized monitoring of a patient's cardiac rhythm is necessary. Specialized equipment should be in the immediate vicinity for treatment of ventricular fibrillation, should it occur.

DR. HERMANN: Probably the most important aspect of this discussion is prophylaxis or prevention of frostbite. Dr. Knight, you have had a wide experience with this problem in Great Britain. Would you outline basic principles for protecting one's self from cold injury?

DR. KNIGHT: The most fundamental and primary means of protection, of course, is proper clothing—multiple layers of light woolen material and a windproof outer garment. Still air is a poor heat conductor, so multiple layers of clothing which entrap air in its intersticies act as an ideal insulator. Proper head gear and foot wear are also necessary. Gloves should be worn at all times, particularly when hands must come in contact with metallic objects.

Since heat is generated into the body by its basal metabolism and by muscular effort, physical activity will provide warmth for the body to some extent. Violent exercise, on the other hand, is injurious. Although it generates heat, it increases the water and heat loss from the lungs. Large

amounts of both heat and water may be lost in this way. In addition, sweating, which is an accompaniment of violent exercise, will cause a more rapid loss of heat due to increased evaporation.

Lastly, it has been my experience that the majority of cases of frostbite occur when people become fatigued, with resultant carelessness. When exposure to prolonged cold is inevitable, one must refrain from becoming overtired. An adequate

diet is likewise extremely important, both in preventing fatigue and in supplying fuel for the metabolism of the body.

DR. HERMANN: Thank you, Dr. Knight. Everyone who is exposed to temperatures which may cause frostbite must heed those basic precepts of self-care. Realization that frostbite implies a long term illness with possible economic and physically disastrous consequences must be impressed on all peoples residing in cold regions. •

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