Snakebite or Frostbite: What Are We Doing?

An Evaluation of Cryotherapy for Envenomation

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THE BITE OF A POISONOUS SNAKE can be a fatal injury. It creates instant terror in its victim and anxiety in his physician. The knowledge that his patient may lose part of his limb, or even die, spurs the physician on to use any treatment reported to be effective. It is not surprising, therefore, that cryotherapy,[1] a plausible recommendation, should achieve instant popularity in a medical community. This is especially so when it has been dramatically presented[2] by a positive proponent like Herbert Stahnke and widely publicized in the popular press. Its continuing popularity in the face of serious complications is due in part to the limited experience of any one physician with patients bitten by poisonous snakes. It is also due to the impossibility of critically evaluating the results of Dr. Stahnke's ligature-cryotherapy program in any clinical situation. One must consider on the one hand that a bad outcome in a snakebitten patient occurred in spite of the use of cryotherapy, or because cryotherapy was used incorrectly or too late, and on the other the possibility that the bad outcome might be because cryotherapy itself added to the damage caused by snake venom.

Since 1960 I have had occasion to repair and reconstruct a number of limbs which had first been bitten by poisonous snakes and then been treated by cryotherapy. My interest was aroused when I noted how similar the damage was to that caused by cold immersion as I had observed it during World War II.

The difficulty in clinically evaluating cryotherapy (or any other therapy) arises from the nature of snakebite itself. The seriousness of such a bite depends upon the amount of venom injected, the toxicity of that particular venom, and the location at which the venom was deposited. In any given patient none of these significant variables can be known or even guessed at until the poison has done its ultimate damage. Even the location of the fang marks gives no exact indication of where the poison is—it may have been placed intravenously. A widely used clinical classification of the degree of envenomation[3,4] is based on the observed condition of the patient 12 hours after the bite. But by that time therapy of some kind has been given in almost every case. Therefore, the observed condition of the patient is usually the net effect of the snakebite and its treatment. If a treatment is used which not only is ineffective, but which by itself can produce similar injurious effects or which can actually enhance the injurious effects of the venom, the picture is even further confused.

Collected statistics for California[4] as well as elsewhere, indicate that mortality due to snakebite is rare, about one-half of one percent. Permanent damage or loss of portions of limbs as a result of snakebite is common when any significant envenomation has occurred.[5] It is imperative, therefore, to know whether we are doing harm or good to the bitten limb when we ad-

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minister cryotherapy. In my opinion this question can only be answered by looking to experimental studies including envenomation with and without cold immersion and to studies of the effect of cold immersion without envenomation. Fortunately many such studies have been made.

According to Stahnke[1] the rationale of the ligature-cryotherapy treatment is as follows. Rapid absorption and systemic distribution of the snake venom and of tissue breakdown toxins can be prevented, first by the placement of a constricting ligature around the limb between the bite and the body, and subsequently by rapidly reducing the temperature of the tissues around the bite. Local destruction of tissues by the enzymatic actions of the venom can also be prevented by lowering the tissue temperature. If, in addition, the body generally is kept warm and circulation is supported, natural detoxification processes can cope with the small quantities of venom which are slowly absorbed and can ultimately protect the patient from both general and local destruction. Lastly Stahnke asserts that immersion of a limb in ice water, even for several days, is not in itself harmful if the rest of the body is kept warm and circulation is supported.

No one seriously questions Stahnke's first assumption that rapid systemic absorption of toxic substances can be prevented by the ligature and cryotherapy or that local destruction of tissues by the venom can be slowed or stopped by cold. However, experimental observations do not support his contention that ultimate detoxification without loss of life or limb can occur during and as a result of cryotherapy. Nor do they support his contention that cold immersion *per se* is not harmful. Let us examine the evidence.

Keating's monograph summarizes the literature of many years with respect to cold injury of the limbs.[6] He points out that "prolonged exposure to water colder than 15° C produces a characteristic pattern of changes that include lasting damage to nerve and muscle." . . . "Six hours' exposure seems to have been required to produce fully developed cases of 'immersion foot'." . . . "In a few cases of immersion injury blood flow never returned to some areas, apparently because of thrombosis of arteries supplying them, and these ischemic areas often became gangrenous." . . . "There was some indication that arterial thrombosis and gangrene occurred only when limbs had been exposed to trauma as well as cold."

If cold immersion alone can produce tissue destruction, what will it do when combined with the trauma of snake venom, itself capable of producing tissue destruction? Stahnke recommends[1] placing the bitten member directly in iced water (0°C) for the first four hours and then protecting the surface with a layer of plastic sheeting and continuing the immersion in iced water for at least 12 hours and often for four to six days of continuous treatment. Tissue destruction would be expected in some patients so treated even if they had never been bitten at all. One patient referred to me after cryotherapy for snakebite had had his forearm and hand immersed in ice water for ten days. Yet the loss of his fingers and of the function of his forearm was attributed by his physician to the snakebite.

Obviously prolonged cold immersion, with or without a tourniquet obstructing blood flow, and with or without snake envenomation, has its hazards. Is there any real evidence that it ameliorates the lethal or the local effects of snake venom?

Since in humans the quantity of venom injected, its toxicity, and its exact locus are always unknown, the effects of therapeutic measures can only be evaluated in animal experiments in which all three of these factors can be standardized. McCollough and Gennaro[5] in 1963 reviewed the experimental studies to that date and added new studies of their own, using radioisotope tagged pooled rattlesnake venom and radioisotope tagged polyvalent antivenin crotalidae in anesthetized dogs. Measured doses of standardized venom per kilogram of animal produced consistent and reproducible toxicity. They, and previous investigators[7] clearly demonstrated under controlled conditions the following facts:

- Local tissue necrosis produced by rattlesnake venom is not decreased by local cooling and in most instances is greater than in untreated animals.
- There was no evidence that local hypothermia (cryotherapy) was of value as definitive therapy, since all animals treated by local hypothermia alone died following injection of lethal amounts of venom.

• If administration of antiserum was delayed for as long as eight hours after injection of venom, some increase in survival was obtained by local hypothermia; however, if antiserum was administered within four hours after envenomation, no benefit of local hypothermia was apparent. What increase in survival there was, was obtained at the price of increased local tissue destruction.

• Using I^{131} labeled antivenom administered intravenously, it is possible to demonstrate 85 percent of the administered dose of antivenom in the envenomated leg within two hours' time. However, cooling prevents or considerably reduces the amounts of I^{131} labeled antivenom which collects at the site of envenomation.

"Without the knowledge of these experimental facts, but with the favorable publicity in news and sports publications," McCollough and Gennaro said, "the rationale of croytherapy seemed logical, and many physicians" continue to use it. Clinical reports attesting its value continue to appear, but analysis of these reports is useless because the degree of envenomation is always unknown and cryotherapy is seldom used as the sole treatment. In light of the evidence that cryotherapy's only real value is in delaying damage when definitive therapy cannot be secured for more than four and less than eight hours after envenomation, and that even this limited benefit can be obtained only at the price of increasing the ultimate damage at the envenomation site, it appears strongly recommended that the method ought to be used rarely if ever. Reliance should be placed mainly on antitetanus, antibiotic and antivenom therapy—recognizing the while the not inconsiderable hazards of the antivenom also.

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FLAREUPS FROM X-RAY

Does the usual preparation of the colon for colonic x-ray possibly cause a flare-up of ulcerative colitis?

"It does. In the first place I don't think you should x-ray patients with severe acute ulcerative colitis or moderately severe ulcerative colitis. Frequently a preliminary film of the abdomen is all you need. If you need follow-up studies to exclude carcinoma, most of these patients are in a chronic state, so it's all right. But some of these patients do come in with severe diarrhea; we just put them on a liquid diet and give them the enema ourselves. The other problem is that many patients with mild and chronic ulcerative colitis have multiple fecal impactions in their colon because they've been taking all kinds of drugs to stop the diarrhea, and to x-ray these patients is a *tour de force.* You just have to be persistent—put them on liquid diets and multiple low enemas. I stay away from cathartics in preparing patients who have ulcerative colitis."

—RICHARD H. MARSHAK, M.D., New York City
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