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STUDIES ON GANGRENE FOLLOWING COLD INJURY. VII. TREATMENT OF COLD INJURY BY MEANS OF IMMEDIATE RAPID WARMING[1]

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It has long been considered that the immediate application of warmth to frostbitten extremities should be avoided. In 1939 the Russian investigator, Ariev (1), reviewed the experimental and clinical observations upon which the accepted procedure of slow, gradual warming is based. He concluded, on the basis of his experimental results (cited below), that rapid warming in cases of cold injury should be subjected to clinical trial. Two human cases were reported in which no ill effects were observed to follow rapid warming of the feet and legs.

The older literature, cited by Sonnenburg and Tschmarke (2), Ariev (1), and Harkins (3), contains many observations which have been interpreted to indicate the danger of rapid warming of frostbitten parts. These observations are, however, uncontrolled and in many cases open to doubt. Perhaps the outstanding example is Barron Larrey's description of cold injury among the soldiers of Napoleon's army during the retreat from Moscow in the winter of 1812–1813 (4):

"Unfortunate was the fate of him, who, with his animal functions nearly annihilated, and his external sensibility almost destroyed by the cold, should suddenly enter too warm a room, or approach too nearly a large bivouac fire. The projecting parts of the body, grown insensible, or being frozen, and remote from the centre of circulation, were attacked with gangrene, which manifested itself at the same moment, and was developed with such rapidity, that its progress was perceptible by the eye, or else the individual was suddenly suffocated by a sort of turgescence, apparently invading the pulmonary and cerebral systems. He perished, as if in a state of asphyxia."

An experimental approach to the problem of slow *versus* rapid warming of frozen extremities was made in 1937 by Harkins and Harmon (5). Hind limbs of dogs were frozen with solid CO_2 and one leg was thawed at 42° C. for comparison with the other thawed at 2° to 12° C. One frostbitten ear of rabbits was thawed at 38° C. and the other at 2° C. In neither case was any essential difference reported between the rapidly and slowly thawed extremities, although the frostbitten legs were observed only for 18 hours. They reported that "if anything, the ears that were thawed in ice water showed a trace more gangrene."

Ariev (1) has reported the results of several series of experiments which were designed to compare the effect of slow and rapid warming of frozen ears and feet of rabbits. After freezing both ears of rabbits with ethyl chloride, one ear was warmed with hot compresses wet in water at 40° to 45° C. The frostbitten part of the slowly-warmed ear was ultimately lost in 5 animals while the rapidly-warmed ear showed only fibrosis in the distal part. Both feet of rabbits were frozen with ethyl chloride to the tibio-tarsal joint. One foot was then rapidly thawed by means of the application of compresses wet with water at 35° to 40° C. while the other was thawed in air. In the rapidly warmed foot, gangrene "only touched the most distal parts of the toes," while in the opposite foot "total gangrene spread over the entire region subjected to freezing."

The experiments reported here were carried out in order to determine the effectiveness of immediate rapid warming in preventing the occurrence of gangrene following controlled cold injury. The effect of rapid warming on local blood flow in frostbitten ears and feet following thawing has been examined by means of fluorescein and by measurement of skin temperature.

METHODS

The method used for the production of controlled cold injury has been described previously (6). In this series of experiments, rabbit ears were immersed at − 55° C. for 1, 1½ or 2 minutes; rabbit feet were immersed at − 55°

[1] The work described in this paper was done under a contract, recommended by the Committee on Medical Research, between the Office of Scientific Research and Development and Stanford University.

C. for 3 minutes and at − 15° C. for 60 minutes. The rapid warming was accomplished by immersion of the foot or ear, immediately after removal from the cold liquid, in a water bath at + 42° C. Ears were warmed for periods ranging from 15 to 120 seconds; feet were warmed for 2 minutes or 5 minutes. During immersion in the warm water the foot or ear was kept in constant motion to facilitate thawing.

The effectiveness of immediate rapid warming was evaluated on the basis of the extent of ultimate tissue loss, the time required for the development of wet and dry gangrene, and the extent of induration and scarring of the ear or foot several weeks after injury.

RESULTS

1. *Immediate rapid warming of rabbit ears following controlled cold injury.* The extent of tissue loss following frostbite of rabbit ears without treatment and with treatment in the form of immediate rapid warming is given in Table I. The

TABLE I

Tissue loss following severe cold injury in untreated animals and in animals treated by immediate rapid warming of ears

All ears frostbitten by immersion in liquid at −55° C.

*Untreated control series**

Duration of exposure	Number animals	Extent of tissue loss
seconds		
60	11	Complete to level of immersion
90	24	Complete to level of immersion
120	2	Complete to level of immersion

Immediate rapid warming

Duration of exposure	Temp. of warm water	Duration of warming	Extent of tissue loss
seconds	*° C.*	*seconds*	
60	+41	15	None
60	+42	60	None
60	+42	120	None
60	+42	120	Complete to level of immersion
60	+42	120	Distal 1 cm.
60	+42	120	None
90	+42	30	None
90	+42	30	Distal 2 cm.
90	+42	120	Complete to level of immersion
120	+42	45	Complete to level of immersion

* These animals are the same as those included in paper I of this series (6).

entire distal part of the ear as far as the line to which it was immersed becomes gangrenous and is eventually lost when the ear is thawed in air at room temperature (untreated). After 60-second immersion at − 55° C. the procedure of immediate rapid warming resulted in saving the entire frostbitten part of the ear in 4 out of 6 animals, and in the retention of about 90 per cent of the injured region in 1 animal. With frostbite of longer duration (90- and 120-second immersion at − 55° C.) the procedure of rapid warming resulted in saving the entire area in 1 out of 4 animals and incomplete loss in 1 animal.

In the treatment of frostbite of rabbit ears by immediate rapid warming, thawing of the frozen ear occurs 10 to 15 seconds after it is placed in warm water. Following removal from the warm bath the ear is intensely hyperemic and usually tends to be somewhat cyanotic. Edema is evident upon removal from the warm bath and exceeds that observed in untreated frostbitten ears during the first 24 hours. Blisters are frequently seen. Exudation of protein-containing fluid from the surface of the ear occurs after 24 to 48 hours and results in the accumulation of a crust of protein which covers the entire frostbitten part of the ear. The crust begins to crack and fall from the ear after 10 to 15 days, revealing a pink surface covered by a thin layer of epithelium. The ear beneath contains a large amount of fibrous tissue which shrinks during the next few weeks. After several weeks the ear is shrunken and thickened, but shows no loss of tissue as the results of injury. The appearance of frostbitten ears treated by means of rapid warming is shown in Figure 1. The course of events following frostbite of a rabbit ear, with and without rapid warming, is outlined in Table II. The times required for development of wet and dry gangrene were quite uniform in the animals treated by rapid warming and in controls.

2. *Immediate rapid warming of rabbit feet following controlled cold injury.* The extent of tissue loss following frostbite of rabbit feet without treatment and with treatment in the form of immediate rapid warming is given in Table III. In general, rabbit feet frostbitten for 3 minutes at − 55° C. and permitted to thaw in air at room temperature (untreated) develop gangrene over the entire injured area and are usually lost to the level of immersion (Table III; Figure 2B and 2C). Following untreated frostbite produced by immersion of rabbit feet for 60 minutes at − 15° C., gangrene develops over the whole of the injured

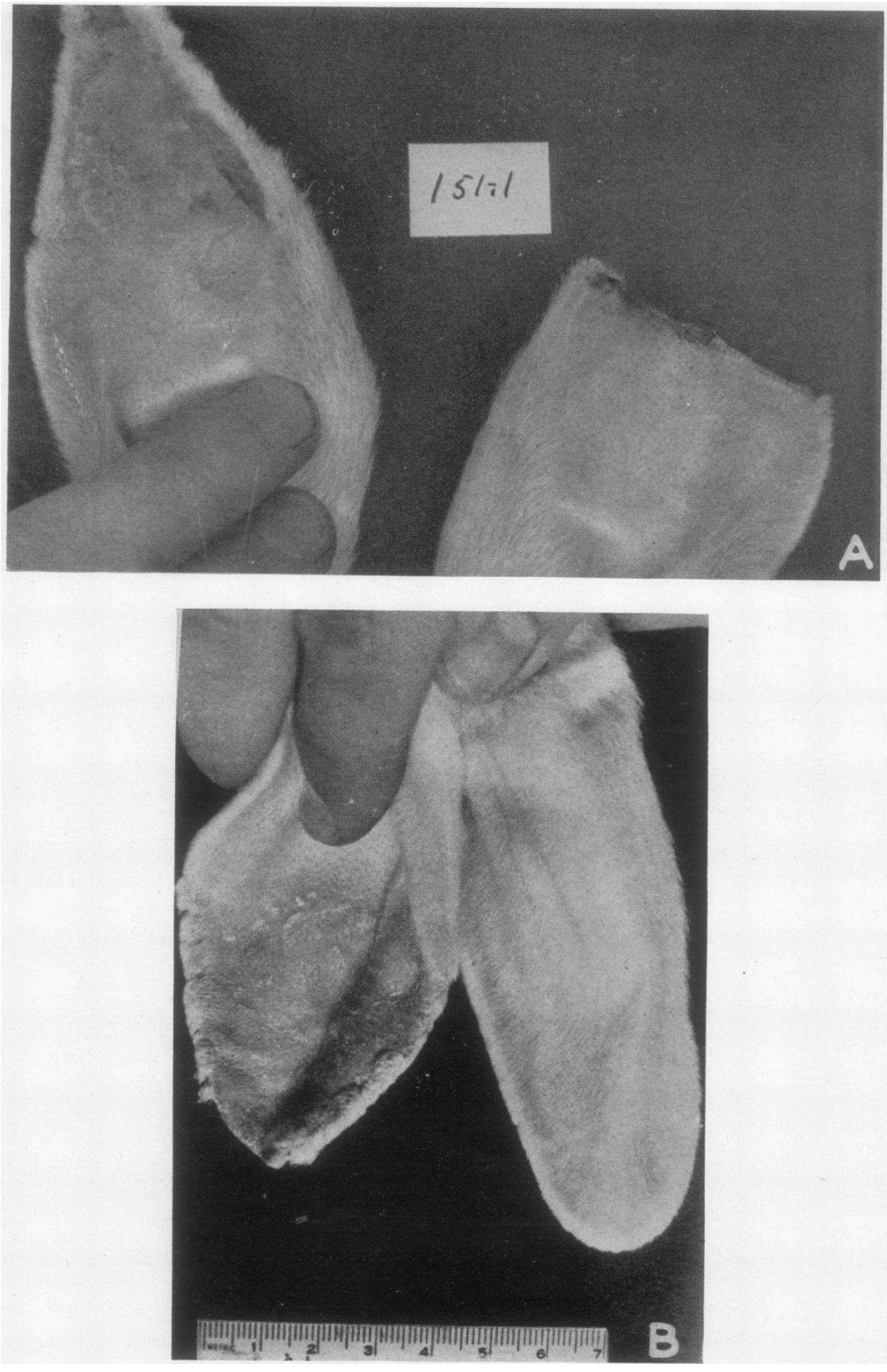

FIG. 1.

TABLE II

Effect of immediate rapid warming on the course of events following frostbite of rabbit ears

Time course of changes following severe cold injury of both ears of one rabbit. One ear treated by immediate rapid warming, while the other permitted to thaw at room temperature in air. Both ears injured by immersion for 60 seconds at −55° C. Right ear warmed in water at +42° C.

Days after injury	Right ear immediate rapid warming	Left ear untreated; thawed in air
1	Edematous. Warm. No blistering	Edematous. Cool. Moderate cyanosis. Large blister
2	Edematous. Warm	Edematous. Cool
3	Edematous. Warm	Weeping. Cool
5	Slight weeping. Warm	Weeping. Cool. Cyanotic. Wet gangrene on tip
6	Warm. Slight weeping. Encrusted with protein	Cold. Wet gangrene, but beginning to dry on tip
8	Encrusted with protein. Warm	Dry to level of frostbite
15	Crust peeling off. Frostbitten portion of ear intact	Spontaneous amputation at level to which immersed

area, and either the entire foot is lost to the level of immersion or a slender tongue of tissue on the plantar surface of the foot is retained.

Treatment of frostbite, following exposure at − 55° C. for 3 minutes, by means of immediate rapid warming results in the survival of all of the injured region except the toes (Table III; Figure 2D). Rapid warming following exposure at − 15° C. for 60 minutes reduces the extent of tissue loss so that the toes or parts of the toes only are lost (Table III; Figure 2E and 2F).

The course of events in rapidly warmed feet is similar to that in rapidly warmed ears. Thawing occurs after about 1 minute in the warm bath. Following removal from the warm water bath the foot usually appears cyanotic and very hyperemic. The edema is somewhat greater than in untreated feet. Exudation begins after about 2 days. Wet gangrene develops on the toes after 4 to 5 days, and drying follows within 1 to 2 days. The toes shrink, become mummified, and eventually separate. In some cases wet gangrene may develop on the dorsal surface of the foot, but this soon dries into a crust 1 to 2 mm. thick. This eschar separates after 10 to 14 days, leaving a pink, granulating surface which is soon covered by epithelium. The foot remains warm at all times in contrast to the fall in temperature which occurs in untreated feet after about 48 hours (7). In final appearance the foot is thickened, with moderate scarring especially on the dorsal surface (Figure 2, compare normal A with D, E, F).

TABLE III

Tissue loss of untreated frostbitten rabbit feet and of frostbitten rabbit feet treated by means of immediate rapid warming

One hind foot of rabbit immersed in cold liquid, at the indicated temperature, to the level of the tuberosity at the base of the fifth metatarsal

*Untreated control series**

Duration of immersion	Temp.	No. of animals	Extent of tissue loss
minutes	° C.		
3	−55	16	Complete to line of immersion (11).† Complete except plantar pad (4). All toes (1).
60	−15	5	Complete to line of immersion (2). Complete except plantar pad (2). None; foot did not freeze (1).

Immediate rapid warming

Temp. of warm water	Duration of warming	Extent of tissue loss
° C.	*minutes*	
	A. Feet exposed at −55° C. for 3 minutes	
+36	5	Complete except for 1 cm.
+43	2	Toes only
+42	5	Parts of toes: II-3; IV-3**
+42	5	Toes only
+43	5	Toes only
+42	5	Toes only
+41	5	Toes only
	B. Feet exposed at −15° C. for 60 minutes	
+42	5	Parts of toes: II-2,3; III-3**
+42	5	Toes only
+42	5	Parts of toes: II-3; III-3**
+42	5	None
+42	5	Parts of toes: II-2,3; IV-2,3**

* These animals are the same as those included in paper I of this series (6).

** Roman numerals indicate digits. Arabic numbers indicate phalanges; 1 = proximal, 2 = middle, 3 = distal.

† Numbers in parentheses indicate number of animals.

FIG. 1. TREATMENT OF EXPERIMENTAL FROSTBITE BY MEANS OF IMMEDIATE RAPID WARMING

A. Both ears exposed at − 55° C. for 1 minute. The ear on the left was warmed for 2 minutes at 42° C. immediately after injury. The ear on the right received no treatment and was permitted to thaw in air at room temperature. Photographed 16 days after frostbite.

B. The ear on the left was exposed at − 54° C. for 1 minute and then immediately warmed in water at 42° C. for 2 minutes. The ear on the right is normal. Photographed 21 days after injury.

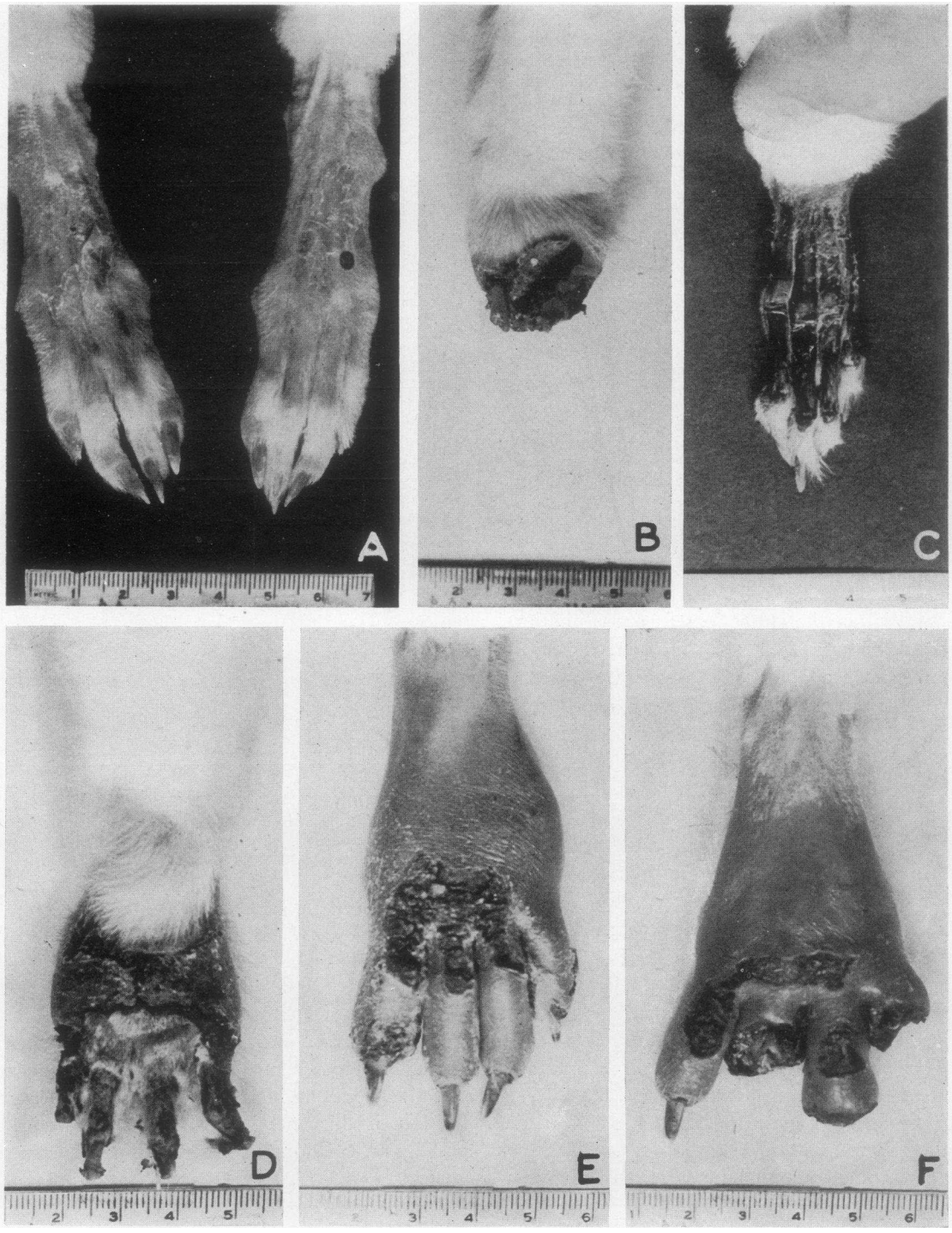

FIG. 2. TREATMENT OF FROSTBITE OF FEET BY MEANS OF RAPID WARMING

A. Normal rabbit feet clipped in preparation for frostbite.

B. Untreated frostbite 16 days after injury. Foot immersed at − 55° C. for 3 minutes. Spontaneous amputation at the level of immersion occurred 14 days after frostbite.

C. Untreated frostbite 27 days after injury. Foot immersed at − 55° C. for 3 minutes. Foot mummified to level of immersion but not separated.

After immediate rapid warming of feet exposed for 60 minutes at − 15° C., induration is more severe than in those feet frostbitten at − 55° C. for 3 minutes. Partial resolution of the induration occurs over a period of about 3 weeks, but sufficient periarticular fibrosis remains to cause spreading of the foot to 1.5 to 2 times its normal width. The fibrosis is particularly marked about the metatarsal-phalangeal joints (Figure 2E and 2F).

3. *Local status of the circulation in frostbitten ears and feet following immediate rapid warming.* Investigation of ears and feet of rabbits subjected to immediate rapid warming following cold injury has shown that certain important differences can be established between these rapidly warmed extremities and those which did not undergo treatment. Reference should be made to the previous papers in this series, cited below, for the details of the methods used.

a. SKIN TEMPERATURE. Skin temperature measurements of rabbit ears, which were immediately warmed for 2 minutes at + 42° C. following 60-second exposure at − 55° C., were made during the first few hours after injury and at intervals up to 5 days. Immersion of the frozen ear in warm water results in a rise in skin temperature to between 25° and 30° C. A further rise in temperature occurs during the next 30 minutes so that the maximum temperature does not differ markedly from that of untreated frostbitten ears at this time (7). During the first 5 to 6 hours after injury the skin temperature of the injured part of the rapidly warmed ears falls several degrees. The most striking differences in temperature between rapidly warmed ears and those thawed in air (control animals) are observed after 24 hours (Table IV). By this time the decline in temperature of the frostbitten untreated ear has become marked, while the frostbitten, rapidly warmed ear is at approximately the same temperature as the normal ears. Forty-eight hours after injury the temperature of the untreated frostbitten ear has fallen to that of the environment; the skin temperature of the rapidly warmed ears remains at 35° to 37° C. Skin temperature measurements of the ears of 3 untreated animals and 3 animals in which the frostbitten ears were rapidly warmed are given in Table IV. It is quite clear that blood flow through the frostbitten, rapidly warmed ears is maintained at a high level at all times during the period following injury.

TABLE IV

Effect of immediate rapid warming upon the skin temperature of frostbitten ears

Injured ear warmed at +42° C.				Injured ear thawed in air			
Animal number	Time after injury	Skin temperature		Animal number	Time after injury	Skin temperature	
		Injured ear	Normal ear			Injured ear	Normal ear
	hrs.	*° C.*			*hrs.*	*° C.*	
113	1 to 4	37.2	37.8	83	1 to 5	35.9	36.8
	20	35.5	35.0		29	24.4	37.3
	44	36.3	37.2		48	22.6	37.3
	68	35.2	36.0		95	24.3	34.4
					120	24.0	32.3
120	1 to 5	32.0	38.6				
	28	38.0	37.8	94	1 to 2	32.4	37.1
	47	34.3	34.5		27	35.8	37.2
	72	34.0	38.3		45	29.2	36.2
	96	37.3	37.5		118	24.5	37.9
121	1 to 3	31.8	33.6	93	1 to 6	35.1	37.7
	24	38.4	38.8		24	30.8	38.2
	49	35.9	37.1		31	28.5	34.9
	72	31.5	25.7		54	27.4	32.7
	119	35.7	37.2		72	27.2	37.2
					98	26.0	36.8
					150	24.3	37.6

b. DEMONSTRATIONS WITH FLUORESCEIN OF LOCAL CIRCULATION IN FROSTBITTEN EARS TREATED BY RAPID WARMING. The adequacy of the local circulation in untreated frostbitten ears and in frostbitten ears treated by immediate rapid warming has been examined by measuring the intensity of fluorescence under ultraviolet illumination in the injured region after the intravenous injection of fluorescein (8).

In 6 animals the distal half of the ear was frostbitten at − 55° C. for 60 to 90 seconds and im-

D. Frostbite treated by rapid warming. Photographed 26 days after immersion at − 55° C. for 3 minutes. Treated by immediate rapid warming in water at 42° C. for 5 minutes.

E. Frostbite treated by rapid warming. Photographed 16 days after immersion at − 15° C. for 60 minutes. Treated by immediate rapid warming in water at 42° C. for 5 minutes.

F. Frostbite treated by rapid warming. Photographed 14 days after immersion at − 15° for 60 minutes. Treated by immediate rapid warming in water at 42° C. for 5 minutes.

mediately warmed in water at + 42° C. Sodium fluorescein, 75 mgm. per kgm., was then given intravenously. In some cases, a second injection of fluorescein was made after 24 or 72 hours. The fluorescence of the rapidly warmed ear was compared with that of the ear of another animal subjected to the same type of cold injury but permitted to thaw in air at + 23° C. The results in all animals were similar. When the fluorescein was given immediately after injury the entry into the rapidly warmed ear was more rapid than in the untreated ear and the maximum fluorescence was greater in the rapidly warmed ear. The differences became more marked when fluorescein was given at successively longer intervals after injury. Figure 3 shows the difference between a rapidly warmed ear (No. 18) and an ear thawed in air (No. 19) 18 hours after frostbite and 40 minutes after the administration of fluorescein. In the untreated ear, fluorescence is apparent only around the central artery and near the tip, in the region of large vessels. In the rapidly warmed ear fluorescence is fairly uniform.

Fluorescein injected 72 hours after injury usually fails entirely to enter the frostbitten untreated ear, while the rapidly warmed ear glows brightly.

In one animal both ears were frostbitten for 60 seconds at −55° C.; one ear was then rapidly warmed in water at + 42° C. Fluorescein was given immediately, and again after 48 hours. The curves showing the intensity of fluorescence as a function of time in both ears are given in Figure 4. After both injections, the fluorescence was more intense in the warmed ear. Forty-eight hours

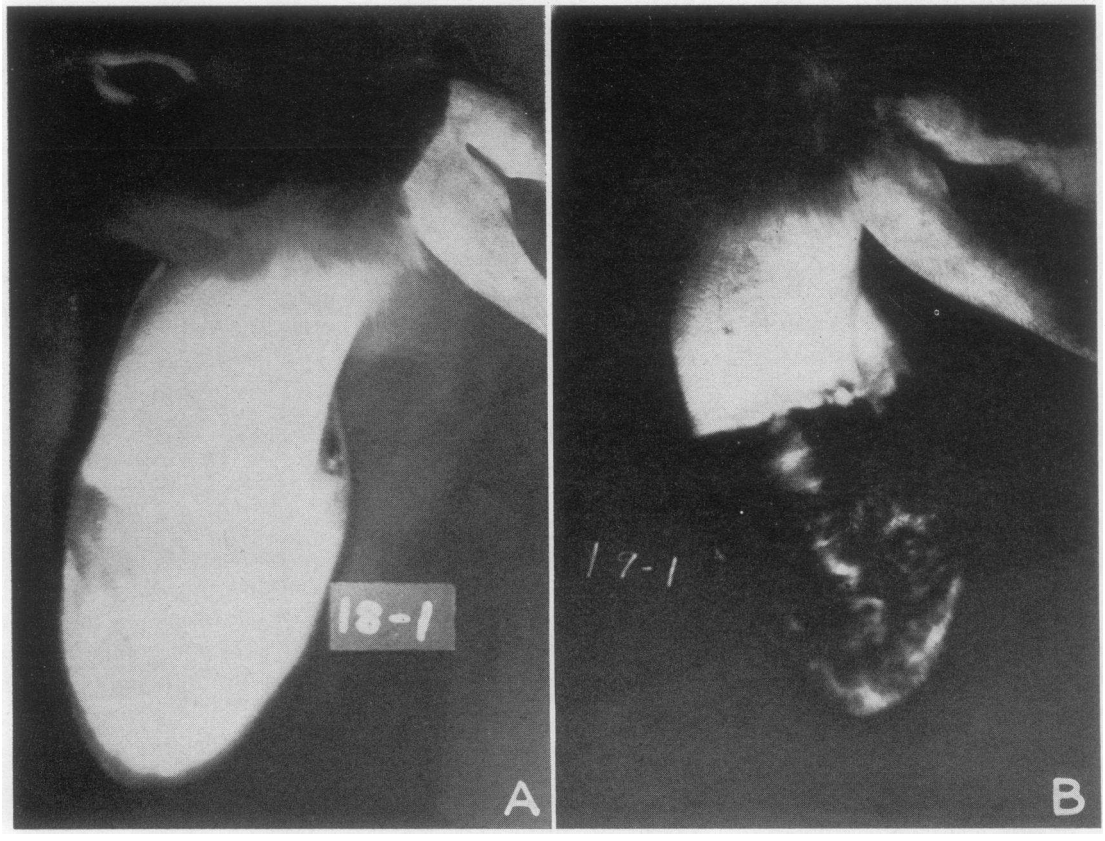

FIG. 3. FLUORESCENCE IN FROSTBITTEN RABBIT EARS AFTER FLUORESCEIN ADMINISTRATION

Fluorescence in rabbit ears 18 hours after frostbite and 40 minutes after the intravenous administration of 75 mgm. per kgm. sodium fluorescein. Ears frostbitten by immersion at − 56° C. for 90 seconds.
A. No. 18. Ear warmed in water at 42° C. for 2 minutes immediately after removal from freezing mixture.
B. No. 19. Ear permitted to thaw in room air (control).

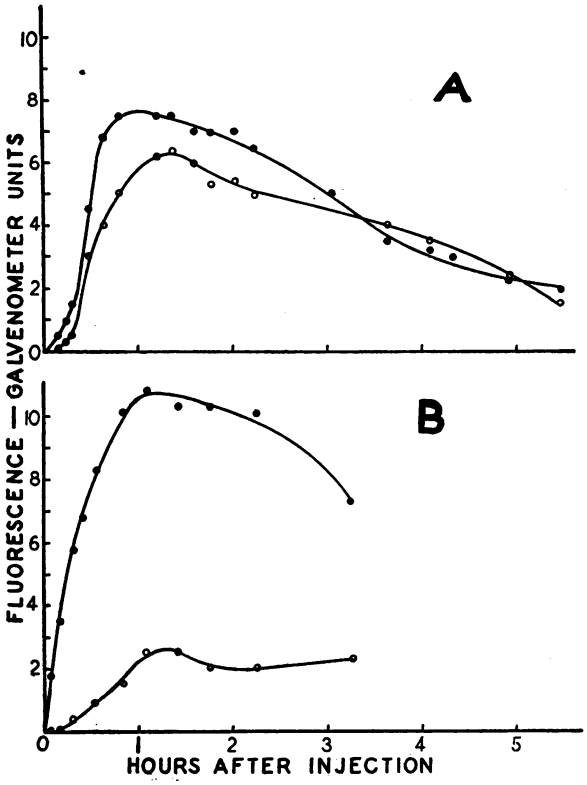

FIG. 4. EFFECT OF IMMEDIATE RAPID WARMING ON THE FLUORESCENCE OF FROSTBITTEN RABBIT EARS AFTER INTRAVENOUS ADMINISTRATION OF FLUORESCEIN

The results of a single experiment in which both ears of a rabbit were frostbitten by immersion at − 55° C. for 1 minute. Sodium fluorescein (75 mgm. per kgm.) given intravenously immediately after frostbite (A) and again 48 hours after frostbite (B).

O—O Ear thawed in air (control).

●—● Ear treated by immediate rapid warming by immersion in water at + 42° C. for 2 minutes.

after injury the maximum fluorescence of the rapidly warmed ear was 4.4 times that of the untreated ear.

The more rapid entry of fluorescein into rapidly warmed ears than into untreated frostbitten ears indicates that circumstances are present in the treated ears which permit the rapid establishment of equilibrium between the plasma and interstitial fluid in the ears. It is probable that this difference is a measure of the greater area through which fluorescein is able to diffuse into the rapidly warmed ear than the area through which it can pass in the untreated ear.

c. EFFECT OF RAPID WARMING UPON THE DEVELOPMENT OF STASIS AS DETERMINED WITH CARBON INJECTION. These data have been presented in detail previously as part of the description of local circulation following severe cold injury (9). Rapid warming of frostbitten ears had the effect of delaying the onset of complete stasis as determined by injection of carbon particles and direct observation of the trapping of carbon in the minute vessels of the ear. When stasis did develop in the rapidly warmed ears it coincided in time with the period of maximal swelling.

4. *Comparison of immediate rapid warming and cooling following severe cold injury.* Cooling of injured extremities has been recommended following cold injury of the trench foot or immersion foot type (10). In the treatment of frostbite, cooling in the form of cold baths (10° C.) and even rubbing with snow have often been recommended (11). From a theoretical point of view the maintenance of a low tissue temperature, and consequently of a low metabolic rate of the tissues, is desirable under circumstances in which blood flow is reduced.

In 2 animals the effect of cooling on the course of events following frostbite was studied for comparison with the results obtained by immediate warming. Both animals were frostbitten by immersion of one foot at − 54° C. for 3 minutes. The foot was then permitted to thaw in air at room temperature and was then cooled. The foot of one animal was suspended on a cradle inside a copper coil 3 inches in diameter, through which liquid at about + 5° C. was circulated. Air temperature inside the coil was maintained at 15° C. for 4 hours. The foot of the second animal was encased in rubber drainage tubing and cooled in cracked ice for 7 hours. In both animals the foot was lost to the level to which it had been immersed in the freezing mixture. The course of events leading to loss of the foot was similar to that in untreated animals.

DISCUSSION

Since any direct injury to the tissues resulting from the action of low temperature *per se* has already occurred by the time treatment can be instituted, any therapeutic measure, to be effective, must be directed toward prevention of the occurrence of the secondary changes which follow exposure to cold. S. S. Girgolav (12), director of a

bureau established in Leningrad under the Commissariat of Public Health, expresses a similar point of view in summarizing the Russian investigations: "From a clinical and pathologic standpoint, tissue necrosis in the body does not result immediately from the effect of cold, but appears as a secondary complication. . . . The course of necrosis is determined primarily by interference with or eventual complete stoppage in blood circulation."

The treatment of experimental frostbite by immediate rapid warming is an effective means of preventing the occurrence of the secondary changes following injury. The beneficial effects are probably not the result of shortening the length of time during which the part remains frozen. Thawing in water at + 36° C., rather than at + 42° C., was not effective in saving tissue even though the foot thawed within 2½ minutes after removal from the freezing bath. At 42° C. thawing requires approximately 2 minutes. In one experiment a foot frozen for 3 minutes at − 53° C. was then maintained in the frozen state at − 2° C. for 30 minutes. It was then thawed rapidly in water at + 42° C. for 5 minutes. The resulting tissue loss—toes only—was comparable to that obtained by rapid warming immediately after removal from the − 55° C. bath.

In confirmation of the work of Ariev (1) the immediate rapid warming of frostbitten feet and ears by immersion in water at + 42° C. has been shown to result in less extensive gangrene and smaller tissue loss than in untreated animals. Those ears and feet which were treated by this method underwent stages of hyperemia, massive edema, and exudation of serous fluid equal to, or exceeding, those of untreated animals. In spite of these changes, the extent of tissue loss was reduced after rapid warming. Evidence has been obtained to indicate that this beneficial effect of rapid warming is brought about by maintenance of blood flow through capillaries. The high skin temperatures of frostbitten parts treated by rapid warming are indicative of high rates of blood flow through the tissues. The accelerated rate of entrance of fluorescein from the blood stream into the interstitial fluid of frostbitten ears following rapid warming was observed in contrast to the slow entrance of the dye in frozen untreated ears. Since evidence has been presented which indicates a direct relationship between rate of penetration and available diffusion area (8), the above observation may be interpreted to indicate the preservation of a larger number of functional capillaries, and hence larger diffusion area, in the rapidly warmed ear. Delay of capillary stasis following rapid warming of frostbitten ears has been observed with the microscope in transilluminated ears, and persistence of blood flow has been verified with the aid of injected carbon.

In a recent publication of the Red Army on cold injury Ariev (13) states: "The fundamental goal of treatment in the pre-reactive period is elevation of tissue temperature, that is, warming of the cold-injured extremity, leaving aside the question of the advisability of rapid warming as under war conditions it does not have a great practical importance." Girgolav (12) also concurs in this view: "It has been definitely ascertained through a number of experimental investigations on different animals, that the more rapidly the chilled animal or any of its parts, such as the ear or extremity, is warmed, the more effective is recovery. From numerous experiments of this type, it has been clearly demonstrated that warming is the most satisfactory procedure." [2] If immediate rapid warming is as effective a measure in the prevention of gangrene following cold injury in man as it appears to be in experimental animals it would be highly desirable to develop methods for the institution of this treatment in the field. In spite of Ariev's statement (13) that conditions for warming are seldom available at evacuation stations at the front and that therefore the question of rapid or slow warming is devoid of practical interest, it seems possible that equipment for rapid warming could be developed if circumstances warrant its use.

Although immediate rapid warming of frostbitten feet and ears results in far less tissue loss

[2] Although this paper by Girgolav is titled "Modern Data on Frostbite" it is apparent that he is also concerned with hypothermia. For example he speaks of "frozen animals" treated by rapid warming and describes the minimum body temperatures tolerated. Part of this confusion has undoubtedly arisen as the result of translation. In view of the results obtained by Ariev (1) with rapid warming of rabbit ears which were frozen beyond doubt, it appears probable that Girgolav intends, in the quotation cited above, to include both frostbite and hypothermia. Treatment of hypothermia by means of rapid warming is considered by Sheinis (14) and by Alexander (15).

than in untreated extremities, the ear or foot which is retained is always thickened by the presence of dense fibrous connective tissue. Later, contraction of the scar is particularly evident in ears, which become shortened, narrower, and somewhat distorted in appearance (Figure 1). With the aim of reducing the extent of this fibrosis which may lead to loss of function, rapid warming should be supplemented by measures designed to control edema. The application of pressure dressings or of closed plaster or plastic casts to feet are satisfactory methods of accomplishing control of the edema following experimental frostbite. These methods of treatment will be described in detail in another paper (16).

SUMMARY

Controlled cold injury of rabbit feet and ears, produced by liquid immersion, was treated by immediate rapid warming of the injured part. The rapid warming consisted of immersion of the ear or foot in water at + 42° C. for 2 and 5 minutes respectively, immediately after withdrawal from the freezing mixture. In the majority of cases this treatment resulted in complete preservation of the frostbitten ear after immersion at − 55° C. for 1 minute, a severity of injury invariably leading to gangrene and loss of the injured part in untreated animals. Gangrene was in most cases prevented, or very greatly reduced in extent, and the amount of tissue loss was greatly reduced by rapid warming of rabbit feet injured by immersion at − 55° C. for 3 minutes or at − 15° C. for 60 minutes. Evidence is presented to indicate that the beneficial effect of rapid warming is brought about by alteration in the pattern of local blood flow in the frostbitten part.

Several weeks after injury the treated ears were shrunken and somewhat thickened and the treated feet showed evidence of periarticular fibrosis sufficient to cause spreading of the feet and toes. It is suggested that rapid warming should be supplemented by measures designed to control edema.

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