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SECTION MEETINGS

CLEVELAND, O.	
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DISTRICT OF COLUMBIA	
George Washington University	October 4, 1956
ILLINOIS	
Wesley Memorial Hospital, Chicago	November 13, 1956
IOWA	
State University of Iowa	October 30, 1956
MARYLAND	
Baltimore City Hospitals	November 2, 1956
PACIFIC COAST	
University of California, Berkeley	October 24, 1956
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University of California, Los Angeles	October 18, 1956
SOUTHWESTERN	
University of Texas Dental School	October 19-20, 1956
WESTERN NEW YORK	
University of Buffalo	November 3, 1956

Effect of Environmental Temperature in Traumatic Shock.* (22840)

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The question of effect of environmental temperature upon survival time in traumatic shock has not been definitely answered. Previous workers(1-5) report optimal temperatures ranging from 10° to 35°C. However, this discrepancy is no doubt due to the use of different methods of inducing shock as well as the use of different species of animals. Since control of the environmental temperature in treating human cases of shock is to some extent possible, the question is an important one. This paper is submitted as a contribution toward the study of this problem.

Method. Our method of inducing shock in the rat has been previously described(6). In brief, it consists in withdrawing the cecum

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through a mid-abdominal incision and pinching it at a rate of 3 times per second with a rubber-tipped hemostat for 15 minutes. Nembutal anaesthesia, in a dose of 50 mg/kg is employed and blood pressure recordings from the carotid artery are taken. Data from several hundred control animals used in previous studies have shown this to be a reliable method of inducing irreversible shock. In the present experiment a refrigerator equipped with a heating unit and small fan was used as the constant temperature chamber, the temperature being controllable to less than .5°. The preparatory surgery and infliction of trauma was done at room temperature, which, however, was always within the range of 20° to 25°. The body temperature was taken by means of a dial thermometer, inserted 4 cm within the rat's colon and read at 15 minute intervals. Nine temperatures, between 10° and 40° were studied, 10 rats being used in each group. Males of the University of Denver colony, weighing between 195 and 205 g were employed.

TABLE I. Effect of Environmental Temperature on Survival Time.

Env'l temp., °C	Mean survival time, min.	Stand. dev., min.	Avg body temp. at death, °C
10	63.1	13.0	19.2
15	59.3	12.0	19.2
17.5	127.8	49.2	19.6
20	212.9	57.6	20.2
22.5	171.3	82.8	23.6
25	153.0	22.4	25.5
30	128.8	26.3	30.0
35	91.5	17.4	31.9
40	58.7	16.7	33.6
	20° *vs* 17.5°,	t: 3.55, P <.005	
	22.5°,	1.30, not sig.	
	25.0°,	3.06, P <.01	

Results. The results for survival time and average body temperature at the time of death are shown in Table I; Fig. 1 illustrates body temperature behavior for 3 contrasting groups.

Discussion. 1. Survival time. Control groups of 10 non-shocked, anaesthetized rats

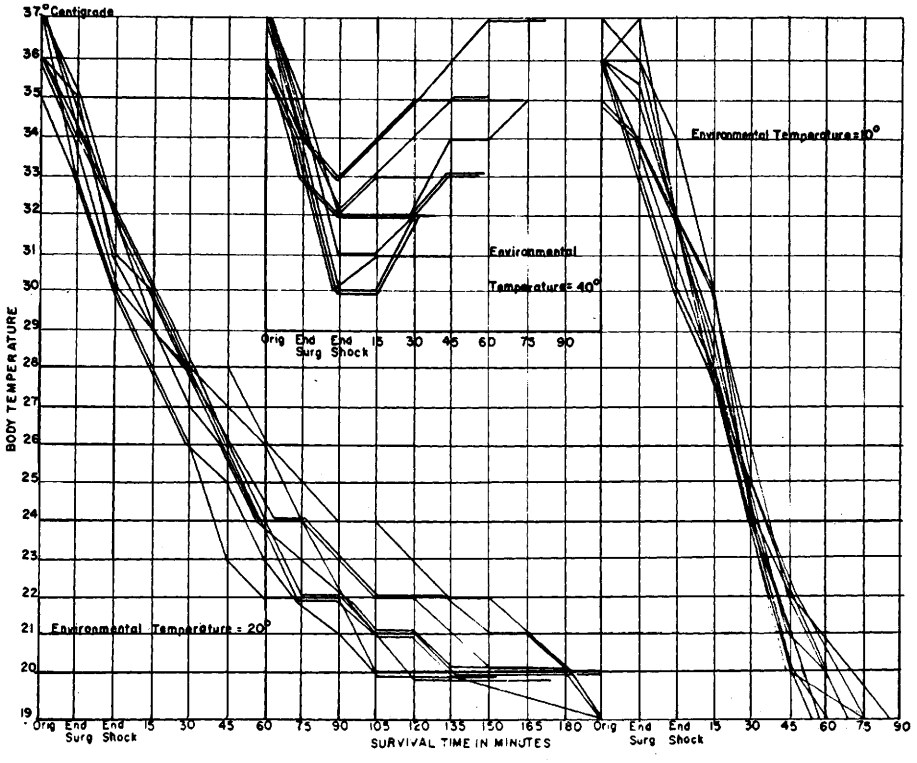

FIG. 1. Trend of body temperature fall of individual rats at environmental temperatures indicated, at 15 min. intervals.

were run at each temperature. At 10°, their mean survival time was 115 minutes, roughly twice that of the experimental group, and at 15°, it was 243 minutes or approximately four times that of the corresponding shocked animals. At all other temperatures, the non-shocked animals were alive and in good condition at the end of 300 minutes. It appears, therefore, that at the 2 lower temperatures, cold contributed directly to the death of the shocked animals, their short survival times not being entirely due to the effect of cold on the shock condition.

It will be seen from the table that the environmental temperature at which survival time was longest was 20° (Mean: 212 min.), although this was not significantly greater than at 22.5° (Mean: 171 min.). It was, however, significantly longer than at 25° (Mean: 153 min.; $p<.01$) and also significantly greater than for the next lower temperature studied, 17.5° (Mean: 127 min.; $p<.005$). Ascending temperatures above 25° caused a progressive reduction of the survival time; at 10° and 15° the survival time was approximately the same (Means: 63 and 59 min.). These results agree with those of Rica *et al.*(3), insofar as 20° being optimal is concerned; we found somewhat lower temperatures less favorable and somewhat higher temperatures more favorable, than did they. The results are also in line with clinical opinion today, which holds that the overheating of shock victims, as practiced in the past, is deleterious. In fact, the difference in survival time at 20° as compared with 30° or more is so great as to suggest the desirability of the use of air-conditioned rooms for shock patients during very hot weather. It will also be noted that the individual variation is greatest at those temperatures where survival time is longest. (S.D. at 20°, 57.6 min. and at 22.5°, 82.8 min.). This suggests that the investigator in the field of shock would perhaps do well to choose a temperature somewhat above or below those figures. The necessity for controlling the temperature, as an important factor affecting survival time, is, of course, obvious.

2. Body Temperature. The average body temperature at the time of death is given in Table I. As shown in Fig. 1 for the 10°, 20° and 40° animals, the body temperature fell by about 5° during the half-hour spent in surgery and traumatization. Upon being placed in the constant temperature chamber, the fall continued precipitately for the low temperature groups, less rapidly for the intermediate temperature groups, and tended to rise for the high temperature group. The control (non-traumatized) groups at 10° and 15° behaved similarly. Their body temperatures at time of death were very nearly the same as those of the experimental group. As previously mentioned, the controls at all other temperatures survived for 300 minutes in good condition. Their body temperatures on termination averaged approximately 10° higher than those of the experimental groups. The results are not strictly comparable, however, since the controls, although given additional anaesthetic when indicated, were never as completely flaccid as were the experimental animals.

It is interesting to note that the survival time of the 2 low temperature groups and the high temperature group is approximately the same, in spite of the great difference in body temperatures, from which it would appear either that both conditions were equally harmful or that the body temperature itself was not the determining factor in causing death.

Summary. The effect of 9 environmental temperatures between 10° and 40° on survival time and on body temperature of rats in traumatic shock was studied. The results indicate: 1. that the optimum temperature is 20°. Survival time at this temperature is significantly better than at 17.5° or 25°; lower or higher temperatures are increasingly harmful. 2. Animals in shock appear to be poikilothermic, their body temperatures tending to approach the environmental temperature.

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Isolation of Intranuclear Inclusion Producing Agents from Infants with Illnesses Resembling Cytomegalic Inclusion Disease.*† (22841)

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On morphologic grounds various authors have suggested that the syndrome termed "generalized cytomegalic inclusion disease" by Wyatt *et al.*(1) or simply "inclusion disease" by Smith and Vellios(2) is viral in etiology and is a generalized form of salivary gland inclusion disease. The demonstration by Fetterman(3) that the infant with inclusion disease may excrete inclusion-bearing cells in the urine has resulted in evidence suggesting that the generalized process may be more common than hitherto suspected and may not have a fatal outcome(4). Information as to the nature of the causative agent is now being elaborated. Smith investigated the salivary gland viruses of rodents(5). From mouse salivary gland tissue an intranuclear inclusion producing virus was isolated in cultures of mouse tissues. Then, employing cultures of human myometrial cells, Smith obtained a cytopathic virus from human salivary gland material and recently isolated a similar agent at autopsy from kidney tissues of an infant with inclusion disease(6). Related viruses also have been recovered from adenoid tissues of children by Rowe and his coworkers(7). Our interest in the problem arose from the concurrent isolation of an agent from human liver biopsy material as has been noted briefly(7,8).

In this communication we are reporting the isolation of cytopathic intranuclear inclusion producing agents from 3 infants during life; each was ill with a syndrome resembling cytomegalic inclusion disease. One virus, derived from liver biopsy material, has been maintained for 20 months in serial passage. From the second child virus was obtained on 3 occasions from the urine, and once from liver biopsy material. Recently, an agent has been isolated from the urine of a third patient.

Materials and methods. Tissue cultures: Roller tube cultures of various human and animal tissues were utilized. The nutrient fluid at first consisted of bovine amniotic fluid (90%), beef embryo extract (5%), horse serum (5%), antibiotics, soybean trypsin-inhibitor (omitted in kidney cell cultures), and phenol red as previously employed(9); for the past 12 months Hanks'(10) balanced salt solution (45%) has been included and the amniotic fluid reduced proportionately. Changes of nutrient fluid were made at 3- to 5-day intervals. For histologic studies, tissues were planted on coverslips or prepared by the collodion technic of Cheatham(11); fixation was with Zenker-acetic or Bouin's and preparations were stained with hematoxylin and eosin. For serial cultivation, inocula consisting of 0.2 ml of coarsely ground tissue removed from the preceding set of cultures were employed. Control cultures were similarly inoculated and maintained in par-

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