Alterations in Pulmonary and Peripheral Vascular Resistance in Immersion Hypothermia

By Leslie A. Kuhn, M.D., and John K. Turner, M.A.

The effects of immersion hypothermia on the systemic and pulmonary circulations were investigated in anesthetized, closed-chest, untreated dogs, cold-adapted dogs and in dogs receiving sympathomimetic and sympatholytic agents. Evidence is presented that both pulmonary and systemic vascular resistances are increased progressively during hypothermia, the former to a greater extent than the latter. It is felt that the primary causes for the increase in vascular resistance are progressive diminution in the cardiac output and the local effect of low blood temperature on the vasculature.

It was the object of these studies to elucidate the effects of hypothermia on the systemic and pulmonary circulations and to assess the effects of sympatholytic and sympathomimetic agents on the course of hypothermia.

Despite the use by several investigators of agents acting on the autonomic nervous system in the hope of favorably modifying the course of hypothermia, there has been relatively little investigation of alterations in pulmonary and systemic vascular resistance produced by hypothermia. The effects of low body temperature on peripheral resistance were initially studied by Bigelow and more recently by Sabiston and his associates. The pulmonary circulation during hypothermia in the intact animal has had scant investigation except for Prec’s studies of pulmonary artery pressure in eight dogs cooled to 28°C and the recent studies of Galletti and his associates performed under conditions of constant flow.

METHODS

Studies were conducted on 58 dogs anesthetized with 30 mg./Kg., of sodium pentobarbital intravenously. After insertion of an endotracheal tube a double-lumen catheter, introduced via an external jugular vein, was so placed that the distal end lay in the main pulmonary artery, the proximal lumen remaining in either the right ventricle or the right atrium. A second catheter similarly inserted was wedged into the periphery of the lung field to record pulmonary “capillary” pressure. A polyethylene cannula was inserted through a femoral artery into the descending aorta. Pressures were measured by Sanborn electromanometers and recorded simultaneously on a four-channel, direct-writing recorder.

Cardiac output was determined by the direct Fick principle utilizing venous blood extracted from the pulmonary artery and arterial blood simultaneously removed from the aorta for oxygen analysis by the manometric method of Van Slyke and Neill. Oxygen consumption was measured on a Sanborn metabolism machine. Hematocrits were determined according to the Thorn method.

After control observations were obtained, the animal was immersed in an ice-water bath, the head, neck and the ventral third of the thorax remaining above water. Pressures, cardiac output, electrocardiograms, eosinophils and hematocrits were determined immediately prior to immersion (after anesthesia) and at five-degree intervals thereafter. Deep colonic temperature was measured by means of a thermistor.

Artificial respiration was instituted via the endotracheal tube when colonic temperature reached 30°C, usually with room air. Effort was made to keep the amount of ventilation approximately uniform in all experiments. The degree of ventilation resulted in the maintenance of normal arterial oxygen saturation even at the lowest body temperatures reached.

Resistances were estimated according to the modified Poiseuille equation

\[
\text{resistance} = \frac{\text{pressure gradient}}{\text{rate of blood flow}}
\]

Ventricular work was estimated using the following formulas:

\[
W_r = \frac{(CO \times 1.055)(P_{Am}-R_{Am}) \times 13.6}{1000} \quad \text{Kg. M/min./Kg.}
\]

\[
W_l = \frac{(CO \times 1.055)(F_{Am} \times 5) \times 13.6}{1000} \quad \text{Kg. M/min./Kg.}
\]

where \(W_r\) = work of right ventricle; \(W_l\) = work
of left ventricle; CO = cardiac output, L./min./
Kg.; 1.055 = specific gravity of blood; PAaw =
pulmonary arterial mean pressure, mm. Hg; PAAw =
right atrial mean pressure, mm. Hg; PAaw =
femoral arterial mean pressure, mm. Hg; 5 = as-
sumed left ventricular diastolic pressure, mm. Hg;
13.6 = specific gravity of mercury.

Experiments were conducted on 36 untreated
anesthetized dogs. The results in these animals
were separated into two groups, those in which
ventricular fibrillation occurred at a temperature
of 22 C. or above and those in which there was
survival below 22 C. without fibrillation. The
dividing point of 22 C. was chosen because this
was close to the average temperature at which
ventricular fibrillation supervened in the untreated
animals. Five untreated dogs were carried to ter-
minus. These animals, in addition to the usual
procedure, received 5 per cent glucose in water
in 1,000 ml./min. so as to more accurately compare them with dogs receiving drugs
dissolved in a similar solution.

Nine dogs received 1-norepinephrine (Levophed
bitartrate), 4 ml. of a 0.2 per cent solution in 1,000
ml. of 5 per cent glucose in water, at a rate of 0.5
ml./min.

Six dogs were given the adrenergic blocking
agent, phenoxybenzamine (Dibenzyline), 50 mg.
in 1,000 ml. of 5 per cent glucose in water intra-
venously at a rate of 3 ml./min. The rate of ad-
ministration was such that the animals received at
least 1.0 to 2.5 mg./Kg., a quantity sufficient to pro-
duce adrenergic blockade at normal body tempera-
ture.

Five dogs received tolazoline (Priscoline hydro-
chloride), an adrenolytic, sympatholytic and chlo-
linergic agent, in a dosage of 250 mg. in 1,000 ml.

| TABLE 1.—Average Peripheral Arterial, Pulmonary Arterial, Pulmonary "capillaries" and Right Atrial Pressures During Hypothermia |
|---|---|---|---|---|
| Anesthetized dogs | Femoral artery | Pulmonary artery | Pulmonary "capillary" | Right atrial |
| Degrees C. | 37 | 30 | 25 | 20 | 37 | 30 | 25 | 20 | 37 | 30 | 25 | 20 |
| Untreated | 121 | 112 | 95 | 58 | 11 | 14 | 11 | 9 | 3 | 5 | 4 | 2 | 3 | 4 | 8 |
| Tolazoline | 100 | 68 | 55 | 13 | 15 | 11 | 2 | 8 | 6 | 8 |
| Phenoxybenzamine | 120 | 116 | 86 | 51 | 10 | 19 | 17 | 9 | 1 | 9 | 13 |
| Cold adapted | 150 | 143 | 109 | 65 | 14 | 18 | 16 | 8 | 5 | 2 | 3 | 1 | 1 | 3 | 1 |

*Standard error. None significant at p < .05.

Results

Effect on Pressure, Flow and Vascular Resistance. The untreated animals showed the
gradual fall in peripheral arterial pressure
described by other investigators (table 1).
Although at 25 C. the average systemic blood
pressure of the norepinephrine group was
slightly higher and the average systemic pres-
sures of the phenoxybenzamine and the tolazo-
line groups were lower than the average of the
untreated animals, there was considerable
variation within each group and there was no
statistically significant difference among the
groups. The two cold-adapted dogs showed
slightly higher arterial pressure throughout
hypothermia than the untreated dogs.

Pulmonary artery pressure, unlike systemic, remained near the initial levels even after
very low temperatures were attained, and was
not appreciably affected by the administered
drugs. Pulmonary "capillary" pressure re-
ained within normal limits, as did right atrial pressure.
Table 2.—Average Cardiac Output and Stroke Volume During Hypothermia

<table>
<thead>
<tr>
<th>Anesthetized dogs</th>
<th>Number</th>
<th>Cardiac output (ml/Kg./min.)</th>
<th>Stroke volume (ml./beat)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Degrees C.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Untreated surviving below 22 C.</td>
<td>18</td>
<td>37 (14)</td>
<td>30 (12)</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>173 (36)</td>
<td>106 (18)</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>8</td>
<td>161 (119)</td>
<td>137 (12)</td>
</tr>
<tr>
<td>Phenoxycbenzamine</td>
<td>6</td>
<td>151 (31)</td>
<td>149 (12)</td>
</tr>
<tr>
<td>Tolazoline</td>
<td>5</td>
<td>172 (32)</td>
<td>222 (26)</td>
</tr>
<tr>
<td>Cold adapted</td>
<td>2</td>
<td>129 (14)</td>
<td>88 (17)</td>
</tr>
</tbody>
</table>

Cardiac output and stroke volume are tabulated in table 2. All groups showed the expected fall in cardiac output at 25 C. and 20 C. The variation among the groups at 37 and at 30 C. is probably attributable to individual variations in depth of anesthesia and varying degrees of shivering. At 25 C. shivering usually stopped and the different groups could be more accurately compared. Although at 25 C. the average cardiac output and stroke volume of the tolazoline-treated group were higher than those of the other groups, there was sufficient intra-group variability to render these differences not statistically significant at the p = .05 level.

Table 3 illustrates alterations in pulmonary and peripheral vascular resistance occurring during hypothermia. At all temperature levels, the relative rise in pulmonary vascular resistance was greater than the rise in peripheral vascular resistance at the same rate of blood flow. Average peripheral vascular resistance was higher with norepinephrine and lower with phenoxycbenzamine and tolazoline than in the untreated animals, but the differences were not statistically significant nor were the differences in resistance between surviving and nonsurviving untreated animals. The apparently small differences in pulmonary resistance among the treated and untreated groups indicates a poor response of the pulmonary circulation to these agents during hypothermia.

Effect on the Electrocardiogram (Table 4). The animals studied exhibited changes in the electrocardiogram similar to those previously described. Death was usually associated with an immediately preceding ventricular fibrillation, generally heralded by multiple premature ventricular contractions, although in three instances (two with norepinephrine and one with tolazoline) death at very low body temperatures (9.5, 12.5, and 8.0 C.) was in asystole. It was not possible to delineate consistent difference in the electrocardiograms...
TABLE 4.—Average Q-Tc and Pulse Rate During Hypothermia

<table>
<thead>
<tr>
<th>Anesthetized dogs</th>
<th>Number</th>
<th>Q-Tc (sec.)*</th>
<th>Pulse rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Degrees C.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Untreated surviving below 22 C.</td>
<td>18</td>
<td>.12 (.01)‡</td>
<td>.25 (.04)‡</td>
</tr>
<tr>
<td>Untreated fibrillating above 22 C.</td>
<td>10</td>
<td>.14 (.01)†</td>
<td>.26 (.07)</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>9</td>
<td>.12 (.01)†</td>
<td>.22 (.06)</td>
</tr>
<tr>
<td>Phenoxybenzamine</td>
<td>6</td>
<td>.11 (.01)†</td>
<td>.28 (.10)</td>
</tr>
<tr>
<td>Tolazoline</td>
<td>5</td>
<td>.13 (.01)†</td>
<td>.24 (.04)</td>
</tr>
<tr>
<td>Cold-adapted</td>
<td>2</td>
<td>.13 (.01)†</td>
<td>.20 (.03)</td>
</tr>
</tbody>
</table>

*Q-Tc = Q-T x square root of R-R interval.
†Standard error.
‡Significant; p < .05.

in the untreated dogs as compared with those receiving drugs, except for the Q-T interval. All groups demonstrated a prolongation of Q-Tc as body temperature fell. Despite similar initial Q-Tc intervals, at lower body temperatures the Q-Tc of the norepinephrine group was significantly lower than that of the untreated group or the groups receiving other drugs (p < 0.05). The two cold-adapted dogs also demonstrated a lower Q-Tc at low body temperature than did the animals receiving drugs.

The expected gradual reduction in pulse rate was seen in all groups with little apparent difference among them.

Effect on Ventricular Work. The estimated work of the right and left ventricles is presented in table 5.* A reduction in the work of both ventricles occurred as body temperature was lowered. However, left ventricular work decreased to a comparatively greater extent than did right ventricular work. At 20 C. there was reduction in left ventricular work to approximately 7 per cent of that at normal temperature, whereas the work of the right ventricle had diminished to only 20 per cent of the control value. There was no statistically significant difference among the groups in regard to ventricular work.

Effect on Oxygen Consumption and A-V Difference. Oxygen consumption fell progressively during hypothermia from an initial level of about 6.0 cc/Kg/min. to 1.0 cc/Kg/min.

TABLE 5.—Average Alterations in Right and Left Ventricular Work Occurring During Hypothermia Expressed as per cent of Variation from the Preimmersion Level

<table>
<thead>
<tr>
<th>Anesthetized dogs</th>
<th>Ventricular work (Kg. M./min./Kg.)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Left ventricle (%)</td>
</tr>
<tr>
<td>Degrees C.</td>
<td>30 25 20</td>
</tr>
<tr>
<td>Untreated surviving below 22 C.</td>
<td>18 77.3* 41 7.3</td>
</tr>
<tr>
<td>Untreated fibrillating above 22 C.</td>
<td>10 58.6 41 11</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>9 69 28 11</td>
</tr>
<tr>
<td>Phenoxybenzamine</td>
<td>6 59 32</td>
</tr>
<tr>
<td>Tolazoline</td>
<td>5 128 41</td>
</tr>
<tr>
<td>Cold-adapted</td>
<td>2 117 26</td>
</tr>
</tbody>
</table>

*No statistically significant difference among the groups.

*Since blood viscosity increases during hypothermia, the conventional formula for determining ventricular work is not strictly applicable as the specific gravity of blood is greater than that listed in the formula. However, the error that this introduces presumably does not interfere with comparisons of the work of the two ventricles at the same temperature.
TABLE 6.—Average Alterations in Hematocrit and Eosinophil Counts Occurring During Hypothermia

<table>
<thead>
<tr>
<th>Anesthetized dogs</th>
<th>Number</th>
<th>Hematocrit</th>
<th>Eosinophils (per mm.³)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Degrees C.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Untreated surviving below 22 C.</td>
<td>18</td>
<td>43</td>
<td>48</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(1)*</td>
<td>(1)</td>
</tr>
<tr>
<td>Untreated fibrillating above 22 C.</td>
<td>10</td>
<td>43</td>
<td>48</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>9</td>
<td>43</td>
<td>48</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(1)</td>
<td>(1)</td>
</tr>
<tr>
<td>Phenoxybenzamine</td>
<td>6</td>
<td>40</td>
<td>38</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(1)</td>
<td>(1)</td>
</tr>
<tr>
<td>Tolazoline</td>
<td>5</td>
<td>40</td>
<td>47</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(1)</td>
<td>(2)</td>
</tr>
<tr>
<td>Cold-adapted</td>
<td>2</td>
<td>44</td>
<td>51</td>
</tr>
</tbody>
</table>

*Standard error.

Kg./min. at 20 C. Below 25 C. the dogs in all groups demonstrated similar oxygen consumptions.

The arteriovenous oxygen difference remained generally unchanged throughout the course of hypothermia and there was no significant difference between the drug-treated and the untreated groups. Arterial oxygen saturation also remained normal throughout hypothermia in all groups.

Effect on Hematocrit and Eosinophils. The hematocrit values in all groups (table 6) except the one which received phenoxybenzamine showed an initial rise of 10 to 12 per cent which was evident at 30 C. Thereafter, the hematocrit fell slightly so that at 20 C. it was equal to or slightly above the initial level.

There was a sharp drop in eosinophils in all groups shortly after immersion, then a leveling off between 30 and 25 C. and a secondary fall in those surviving to 20 C. No significant alteration of this pattern could be delineated in any of the groups.

Effect on Rate of Cooling (Table 7). The rate of cooling was not uniform throughout hypothermia since to a certain extent it was dependent on the depth of anesthesia and the degree of shivering. Cooling at body temperatures below 25 C. was considerably slower than it was during the initial stages despite the frequent occurrence of shivering between 37 and 30 C. The norepinephrine group initially showed a slightly slower average rate of fall and the phenoxybenzamine and tolazoline groups a more rapid fall than did the untreated animals. Below 25 C. the average rate of temperature fall was slightly more rapid in the norepinephrine group (0.58 min./degree C./Kg.) than in the untreated animals (0.71 min./degree C./Kg.), but the differences were not statistically significant. The two cold-adapted animals cooled slightly more slowly below 25 C. than did the other groups.

Effect on Survival. The average temperature at death (table 8) of the untreated dogs was 21.9 C. as compared with Hegenauer's average of 21.3 C. for dogs with one ventricular catheter and 23.3 C. for dogs with two intracardiac catheters. In the absence of intracardiac catheters, it might be expected that the dogs in each of the groups would survive body temperatures lower than those recorded here.

Both the group receiving norepinephrine and the group receiving tolazoline had a lower average temperature at death than did the untreated group. When subjected to statistical analysis, these differences are significant only at the 10 per cent level of probability. Although this is somewhat unconvincing from the statistical standpoint and the number of experiments is small, the impression remains that in some cases norepinephrine did pro-
TABLE 7.—Average Rate of Temperature Fall

<table>
<thead>
<tr>
<th>Anesthetized dogs</th>
<th>Number</th>
<th>Rate of temperature fall (min./degree C./Kg.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Degrees C.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Untreated surviving below 22 C.</td>
<td>18</td>
<td>.41 (.02) .44 (.03) .71 (.06)</td>
</tr>
<tr>
<td>Untreated fibrillating above 22 C.</td>
<td>10</td>
<td>.4 (.04) .38 (.03)</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>9</td>
<td>.44 (.08) .52 (.08) .58 (.08)</td>
</tr>
<tr>
<td>Phenoxymezamine</td>
<td>6</td>
<td>.35 (.04) .53 (.07)</td>
</tr>
<tr>
<td>Tolazoline*</td>
<td>5</td>
<td>.28 (.08) .28 (.08) .43 (.07)</td>
</tr>
<tr>
<td>Cold adapted</td>
<td>2</td>
<td>.49 (.03) .47 (.03) .85 (.07)</td>
</tr>
</tbody>
</table>

*Standard error.

TABLE 8.—Average Temperature at Death with the Range and Standard Error Indicated.

<table>
<thead>
<tr>
<th>Anesthetized dogs</th>
<th>Number</th>
<th>Average temperature at death (C.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Untreated</td>
<td>5</td>
<td>21.9 (19.0-25.0) (.3)</td>
</tr>
<tr>
<td>Norepinephrine*</td>
<td>8</td>
<td>18.0 (9.5-24.0) (1.8)</td>
</tr>
<tr>
<td>Phenoxymezamine</td>
<td>6</td>
<td>23.9 (23.0-26.6) (.8)</td>
</tr>
<tr>
<td>Tolazoline*</td>
<td>5</td>
<td>17.5 (8.0-23.0) (2.0)</td>
</tr>
<tr>
<td>Cold adapted</td>
<td>2</td>
<td>22.5</td>
</tr>
</tbody>
</table>

*The difference between average survival temperatures of the norepinephrine and tolazoline groups and the other groups is significant at the 10 per cent level of probability.

long life to a certain extent. Thus, one dog receiving norepinephrine lived to 9.5 C. and another to 12.5 C., low temperature levels for dogs with intracardiac catheters without artificial support of the circulation. The average temperature at death of neither the phenoxymezamine-treated nor the cold-adapted dogs was as low as that of the untreated dogs, but the differences were not statistically significant.

DISCUSSION

The peripheral resistance measured in these experiments is the resultant of the many factors affecting the circulation. From these data it is not possible to analyze precisely most of these. In hypothermia several forces are at work which tend to increase viscosity and hence the vascular resistance.

The rise in hematocrit, the diminished flow and the cooler temperature of the blood should all act in this manner. The studies of Lynch and Adolph on the rat mesoecum and hamster cheek pouch support the concept that the increase in resistance during hypothermia is due to alterations in the blood and not in the vessel wall. However, in our experiments the increase in resistance cannot be attributed principally to hemoconcentration because peripheral resistance continued to increase at low body temperature despite a return of the hematocrit toward pre-cooling levels. In addition, the efficacy of phenoxymezamine in reducing the peripheral vascular resistance in some animals also favors the supposition that vasoconstriction was at least partially responsible for the rise in peripheral resistance during hypothermia in certain animals. In other animals, however, and in the group viewed as a whole, there did not appear to be a neurogenically controlled vasoconstrictive factor of significance. In these experiments, the marked diminution in cardiac output appeared to be of major importance in accounting for an increase in peripheral vascular resistance, which apparent despite a fall in systemic pressure. However, the lack of uniform response of peripheral arterial pressure to autonomic agents may have been because maximal vasoconstriction occurred during immersion in the ice-water bath. The effect of low body temperature on the responsiveness of vessels to dosages which produce adequate response at normal body temperature should also be considered. Although there are no data on this problem as far as these experiments are concerned, quantitatively different responses at low body temperature as compared to those at normal body temperature have been observed with other drugs. 7, 8
The action of norepinephrine in rendering some animals more tolerant to hypothermia is not clear. It is difficult to state whether the benefits are derived from raising the coronary pressure head, improving the force of myocardial contractions, shortening the activity period of the cardiac cycle, decreasing ventricular excitability or a direct action on the coronary vessels. Although Berne and Sabiston imply that coronary flow remains adequate during hypothermia, Ross and Edwards have achieved beneficial results with perfusion of the coronaries.

As in the systemic circulation, it is not possible from these data to determine the exact causes of the raised pulmonary vascular resistance in hypothermia, but the relative lack of response of pulmonary artery pressure to phenoxybenzamine or to norepinephrine would support the view that nervous factors are not of major importance. The experiments of Galletti and his associates have shown increases of pulmonary vascular resistance in open-chest dogs at low body temperatures when a constant blood flow is maintained, attributable to local action of cool blood causing increased pulmonary vasomotion. The relatively greater degree of increase in pulmonary vascular resistance during hypothermia in these experiments as compared to those of Galletti may be due to the fact that flow is progressively reduced during hypothermia in the intact, close-chest animal. Changes in local pH which might affect pulmonary vascular resistance were not measured in our experiments, although Galletti and his associates believe that an increase in local pH due to blood cooling would, if anything, tend to cause lower pulmonary vascular resistance and hence cannot be considered as major factors in the observed rise in pulmonary vascular resistance. As in Galletti's experiments, the relatively greater rise in pulmonary, as compared to systemic, vascular resistance during hypothermia supports the concept of local action on the pulmonary vasculature.

There is suggestive evidence that the progressively increasing pulmonary vascular resistance in hypothermia may exert a deleterious effect. It has been seen in these experiments that although the work of both ventricles declines, the work of the right ventricle is proportionately much greater than that of the left at low body temperatures. This relatively greater work of the right ventricle is attributable to the high pulmonary vascular resistance against which it must pump and the ultimate conclusion may be failure of the right heart. There is some evidence that right heart failure actually does play a role in the pathologic-physiology of hypothermia. Autopsy findings consistent with right heart failure have been reported by several observers. Both digitalis and venesection have been used with apparently favorable results in prolonging life in hypothermia. Berne, however, by studying intracardiac pressure pulses, concluded that there was no evidence for myocardial failure in hypothermia. In the experiments reported here a slight rise in right atrial pressure, which did not progress as body temperature was lower, was generally noted in all the groups, but the influence of artificial respiration on the right atrial pressure was not clearly defined.

The two cold-adapted dogs demonstrated no increased resistance to hypothermia with respect to the temperature at which ventricular fibrillation occurred, a finding consistent with Adolph's results in rats, but in contrast to the conclusions of Covino and Beavers who studied a larger number of dogs than reported here and exposed them to lower environmental temperatures.

**Summary**

The effects of immersion hypothermia on the systemic and pulmonary circulations were investigated in untreated, anesthetized dogs and in anesthetized dogs receiving sympatholytic and sympathomimetic agents. Both pulmonary and systemic vascular resistance increased progressively during hypothermia, the former to a greater extent than the latter. It is felt that the primary causes for the increase in vascular resistance are progressive diminution in the cardiac output and the local effect of blood of low temperature on the vas-
VASCULAR RESISTANCE IN IMMERSION HYPOTHERMIA

Norepinephrine caused a moderate, variable increase in peripheral vascular resistance and lowered slightly the average temperature which could be safely attained whereas the sympatholytic agent, phenoxybenzamine, caused a variable fall in peripheral vascular resistance and death from ventricular fibrillation at slightly higher average temperature than in the untreated animals. Attempts to significantly alter pulmonary arterial pressure and resistance during hypothermia by sympatholytic and sympathomimetic agents were unsuccessful. The high pulmonary resistance was associated with maintenance of a relatively high degree of right ventricular work at low body temperatures, whereas the work of the left ventricle declined to a greater extent than did that of the right ventricle. Cold adaptation for two months did not protect against ventricular fibrillation in hypothermia.

ACKNOWLEDGMENT

The technical assistance of Donald E. Schuler, SP2, and Cleveland P. Hickman, Jr., Cpl., is gratefully acknowledged.

REFERENCES

10. Berne, R. M.: The effect of immersion hypo-


Alterations in Pulmonary and Peripheral Vascular Resistance in Immersion Hypothermia

LESLIE A. KUHN and JOHN K. TURNER

Circ Res. 1959;7:366-374
doi: 10.1161/01.RES.7.3.366

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1959 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circres.ahajournals.org/content/7/3/366

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation Research can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation Research is online at:
http://circres.ahajournals.org/subscriptions/