Cardiac Physiology in Dog During Rewarming From Deep Hypothermia

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The influence of body temperature on cardiac activity in the dog under conditions of immersion hypothermia has been described (1). The present communication continues those studies through the stage of rapid rewarming from temperatures which approximate the lethal. They were undertaken to supplement general knowledge on the subject of hypothermia, and with the thought that additional information might be of practical importance. Investigations to date on this aspect of hypothermia in both dog and man have been concerned mainly with electrocardiographic analyses. Hook and Stormont (2) and Prec et al. (3) have shown that the normal cardiac rhythm as well as normal P-R and QRS time relations are restored by rewarming from moderate hypothermia in the dog. In hypothermic man the most characteristic feature of cardiac behavior is auricular fibrillation, which appears when the body temperature has been lowered to approximately 29° to 30° (4) and this too is reversible (5-7) as are the altered T waves, rhythm, etc.

Bigelow et al. (8, 9) have rewarmed dogs from rectal temperatures of 19° to 20° and report measurements in individual experiments with which the data to be presented below are generally in agreement. The differences concern mainly the mean low temperature from which successful rewarming may be anticipated and the incidence of idioventricular rhythm in uncomplicated hypothermia.

In the course of the present investigation it became apparent that the conditions for some of the experiments introduced a previously unrecognized artefact, upon the basis of which an hypothesis to explain death in hypothermia was presented in the aforementioned paper (1). The nature of the artefact has more recently been presented and the hypothesis retracted (10). In brief, some of the measurements reported were from experiments involving resort to ventricular catheterization. Although the measurements reported are not invalidated by the mode of measurement, the catheters used were found to initiate idioventricular ectopic beats at temperatures approaching 20° C., and these in turn, uncontrolled, precipitate ventricular fibrillation, thus terminating many experiments at a temperature which most uncatheterized dogs tolerate well.

METHODS

The pre-immersion preparation, mode of cooling and methods of recording have been presented (1) as have the rewarming procedure, justification for use of artificial

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respiration at low temperatures etc. (10). Mongrel dogs were used ranging from 7 to 16 kg. The temperature of the blood in the right ventricle was measured directly simultaneously with rectal temperature in 6 dogs throughout cooling and rewarming. The relation of the former to the latter at all stages was tabulated or graphed, and served as a basis for estimating 'heart' temperature in experiments in which only rectal temperature was recorded (1). All temperatures hereafter refer to the heart, and are expressed in degrees Centigrade.

The overall study involved 24 experiments which were completed through the rewarming stage. The experiments are divided into 3 groups on the basis of the procedures involved. In group I, 10 dogs were cooled to a mean temperature of 16.2° ± 1.0 and rapidly rewarmed in a 42° to 45° bath. Electrocardiograms (standard limb leads), mean carotid pressure, and pulse rate were recorded at regular intervals. Six dogs comprise group II. The right ventricle was catheterized, the catheter bearing a thermocouple at its tip for registration of the ventricular temperature. Other measurements were those described for group I. The remaining 8 dogs (group III) carried catheters in both ventricles for registration of pressure cycles. Electrocardiograms and pulse rates were also recorded, but not blood pressure. The latter groups (II and III) were cooled to mean temperatures of 18.0° ± 1.0 and 19.1° ± 1.1, respectively.

RESULTS

The data are summarized in figures 1, 5 and 6. A comparison with figures 1, 6 and 8 of the previous report (1) reveals that cardiac function during rewarming mirrors that seen in cooling. Only the curve for peak left ventricular pressure (LVP) appears not to fit into the general scheme, and this will be discussed more fully.

The rewarming pulse rate curve of figure 1 differs from that in cooling mainly by being somewhat lower, i.e. shifted toward the higher temperatures. The curve is the average for all 24 dogs. The deviations percentage-wise are greatest at the highest temperatures, where the rate range is 120 to 200 beats/minute. At the other extreme the range is 1 to 24.

The curve for mean arterial pressure in figure 1 is the composite of the data for
groups I and II, exclusive of 2 which reverted to circulatory shock in the course of rewarming. The greatest deviations from this curve are at the highest temperatures where the range is from 94 to 158 mm. Hg. The very rapid initial recovery of blood pressure is somewhat surprising in view of the mode of rewarming which might be expected to broaden the vascular bed, thus neutralizing the influence of increased heart rate. Recovery of vasomotor reflexes at the lowest temperatures, where the blood pressure rise is steepest does not seem likely, although data on this point are lacking. Nor can the abrupt rise be related to shivering, since this reflex is not called forth in this type of rewarming, (see also Penrod, II). That flow through the superficial tissues is increased immediately on immersion in hot water is indicated by the more rapid rise in heart temperature compared to that of the rectum. Peripheral blood flow studies under these conditions have not been made, and are clearly indicated.

Also plotted in figure 1 are the P-R intervals and electrical systole (Q-T) for all rewarming temperatures. The curves are from the records of 16 dogs in groups I and II. The reversible character of the temperature influence on conduction and repolarization throughout the temperatures range is indicated, as was previously shown for higher temperatures (3). The T-wave and S-T segment undergo the most dramatic changes with temperature of any portion of the EKG, the former sometimes being nonexistent in a given lead. The greatest deviations from the plotted Q-T curve did not exceed 14 per cent. In figures 2 and 3 are records taken from uncatheterized dogs during rewarming, which reveal the changes in character of the several portions of
the EKG and the characteristic time relations. Attention is called to the fact that a P-wave is not necessarily present in all three simultaneously recorded leads.

The curve of figure 1 labelled ‘Activity phase’ expresses the duration of combined systole and isometric relaxation as a function of increasing temperatures. This is the composite of data from 8 dogs of group III. The records from which the data were taken are typified by those in figure 4. The prolonged period of contraction, ejection and isometric relaxation at the low temperatures could be the result of altered viscous-elastic properties of the myocardium or to a slower rate of energy transformation and liberation. The close parallelism between this curve and that of the Q-T segment suggests dominance of the metabolic factor.

The ratio ‘activity phase’: cycle duration, expressed as percentage, is plotted in figure 5 as a function of pulse rate, and indirectly as a function of temperature. The upper curve is from data for the left ventricle and the lower for the right. It is to be expected that, since pulse rate and activity phase bear the same relation to temperature during rewarming as in cooling, the ratios expressed in figure 5 should also be the same. With a rising pulse rate from lowest temperatures, the activity phase quickly occupies the major portion of the cycle (70–75%) apparently without dangerously influencing coronary flow or cardiac energy liberation. This is attested by the complete rewarming of all dogs, including those which reverted to circulatory shock with consequent low coronary pressure head. This result seems to emphasize the error expressed previously (1) that the prolonged activity phase through its influence on coronary flow might adversely affect the O₂ supply to the heart.

The rewarming LVP curve of figure 6, in view of the recovery of arterial pressure shown by groups I and II, raises another question regarding the influence of ventricular catheters on the hypothermic state. Whether or not the catheters are instrumental in preventing the full blood pressure recovery cannot be ascertained from these experiments alone. That some special aspect of the experimental technique is the causative factor in the poor recovery of pressure rather than the hypothermia as such is attested not only by the results obtained in groups I and II, but also by the success in rewarming from equally low heart temperatures achieved by Haterius and Maison (12). The rewarming deaths encountered under the conditions of these experiments have been discussed elsewhere (10) and it was shown that the deaths were in all cases due to ventricular fibrillation with no evidence that shock was a primary
factor. For the present the answer to the question raised must await further investigation.

**DISCUSSION**

The complete reversibility of cardiac function and blood pressure after hypothermia indicates that the deviations from normal are entirely on a functional basis rather than pathological. Temperature appears to determine function only through its regulation of the rate of the underlying metabolic processes, and on this basis it dictates ‘normal’ function at all levels of hypothermia. The only gross pathology thus far encountered in dogs is the rather frequent occurrence of subendocardial hemorrhage in one or both ventricles irrespective of the presence of catheters. These vary in extent from scattered petechiae on the trabecular eminences to areas of 2 to 4 cm. Microscopic examination reveals that the hemorrhages are confined to the subendocardium without extension into the myocardium. They are apparently without prognostic importance, for they occurred in 60 per cent of the dogs of these 3 series, catheterized and uncatheterized alike; whereas among those catheterized dogs which failed to survive cooling and rewarming (10) were many with no visible evidence of hemorrhage, or at most a few petechiae. Similarly these hemorrhagic areas are not necessarily the source of the ectopic beats characteristic of the catheterized state, for uncatheterized dogs with large areas of hemorrhage were free of cardiac arrhythmias to the lowest temperatures. In the absence of more information on causes, further discussion at this time will be fruitless. Reference to it is made only to indicate that the altered physiology described is the same in its presence or absence. This pathology has to the writer’s knowledge not previously been described in hypothermia.

Several features of canine cardiac activity in hypothermia seem to distinguish it from man in particular. 1) Auricular fibrillation is characteristic in human subjects, yet it has not appeared in over 100 hypothermic dogs for which EKG records are available. 2) The lethal temperature for the dog is near 16°C. Few records exist of man’s recovery from temperatures below 22°C. A notable exception is a recent case of accidental hypothermia not yet reported save in the daily press. This patient when found had a rectal temperature of 10°C, and survived rewarming. 3) Abnormalities of ventricular rhythm are relatively infrequent in dogs. Of 24 noncatheterized dogs cooled to lethal or near lethal temperatures, only 2 showed marked ventricular rhythms during cooling. The rest maintained a sinus rhythm throughout or produced occasional ectopic beats.
The question of cause of death in hypothermia remains. The results of experiments on the dog presented previously (10) and above are to some extent in accord with the postulate made by Lutz (4) to the effect that death is due primarily to progressive prolongation of diastole which ultimately becomes infinite. This in turn is ascribed to the slowing of metabolic processes underlying impulse formation until the rate of energy accumulation becomes inadequate. Such an hypothesis seems to apply in those cases where the normal course of hypothermia is not interrupted by a strategically timed ventricular ectopic beat which precipitates ventricular fibrillation.

Fig. 6. RELATIONSHIP OF LEFT VENTRICULAR PRESSURE TO HEART TEMPERATURE. Peak left ventricular pressures during progressive hypothermia (upper solid curve) and rewarming (lower solid curve). Included are curves from 3 individual experiments. The variability may reflect the influence of cardiac catheters.

SUMMARY

Dogs subjected to immersion hypothermia may be successfully rewarmed from a mean heart temperature of 16.2° C. (approximately the lethal temperature) when placed in a 45° bath. The return to normal of the pulse rate, blood pressure, P-R, Q-T and the duration of systole and isometric relaxation follow courses with increasing cardiac temperature which mirror almost exactly those observed in cooling. This complete reversibility suggests that within the temperature limits described the cardiac phenomena which obtain in cooling represent merely a temperature influence on metabolic rate rather than disfunction through pathology. There is from the experiments reported no evidence that the subendocardial ecchymoses seen at autopsy influence the course of either cooling or rewarming. The hypothermic dog has been compared and contrasted with the hypothermic human subject.

REFERENCES


