Effect of Artificially Controlled Heart Rate on the Incidence of Ventricular Fibrillation in Hypothermia

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ABSTRACT

TORRES, J. C., E. T. ANGELAKOS AND A. H. HEGNAUER. Effect of artificially controlled heart rate on the incidence of ventricular fibrillation in hypothermia. Am. J. Physiol. 195(2): 437-439, 1958.—Five dogs were subjected to auricular and five to ventricular driving throughout the period of cooling. All animals in both groups succumbed to ventricular fibrillation at mean temperatures of 21.3°C and 20.3°C, respectively. These temperatures do not differ significantly from those for dogs with spontaneous heart action. Thus, abnormalities in S-A nodal rhythm or A-V conduction appear not to be contributory to fibrillation, since the one is eliminated by auricular driving and the other by ventricular driving. Intraventricular conduction times were assumed equal to the QRS duration. In both groups the conduction time increased linearly over the temperature range studied, and the slopes were parallel, suggesting that the limiting, temperature-sensitive process in conduction is the same for Purkinje system and myocardium.

METHODS

Ten healthy mongrel dogs of both sexes, ranging from 8 to 15 kg in weight were anesthetized by intraperitoneal sodium pentobarbital (33 mg/kg). In five dogs the driving electrode was in the tip of a catheter which was passed to the right auricle via the jugular vein. Correct placement was assured when a monitoring oscilloscope showed an auricular driving configuration. The indifferent electrode was planted under the skin in the precordial area. In the other five dogs the driving electrode (bipolar) was sutured to the pericardium overlying the left ventricle. Chest closure and reduction of the pneumothorax permitted resumption of spontaneous respiration. Blood pressure was recorded in all dogs from the carotid artery manometrically.

Hypothermia was induced by immersion in an iced bath as previously described (4) and artificial respiration was initiated in all dogs at 26°C. Temperature measurements were made thermoelectrically by means of an esophageal thermocouple placed at the level of the auricles. Rectangular monophasic pulses of 1 msec. duration were delivered to both ‘auricular’ and ‘ventricular’ preparations from Grass stimulators at voltages just sufficiently intense to control the rhythm. Lead II was recorded on a Visocardiette and monitored via an oscilloscope.
HEART RATE
(beats/min.)

FIG. 1. Cardiac driving rates vs. heart temperature during progressive hypothermia. A, auricular and V, ventricular driving. Slopes parallel spontaneous rhythm but at slightly higher rates.

RESULTS

Auricular Driving. In this group of experiments driving via the right auricle precluded the possibility of S-A nodal irregularities. The driving rate (which was of necessity slightly faster than the spontaneous rate) was maintained at a level compatible with an adequate blood pressure, and decreased in parallel with the spontaneous decrease which results from the temperature drop (fig. 1). All five dogs succumbed to VF at a mean temperature of 21.3°C and mean terminal rate of 67 b.p.m. No isolated ectopic action was observed or recorded prior to fibrillation.

Ventricular Driving. This presumably eliminated any possibility of A-V conduction irregularities. Again all five dogs terminated in VF at a mean temperature of 20.3°C and from a preterminal rate of 67 b.p.m. Ectopic activity of both ventricular and supraventricular origin was recorded in each of three dogs.

QRS durations were measured in all experiments as indices of ventricular conduction times. As anticipated, the ventricular conduction time in ventricular driving was greater than in auricular driving.

DISCUSSION

The mean lethal heart temperature in 110 dogs subjected to hypothermia has been reported by one of us (5) to be 20.1°C. The values obtained in this study (auricular driving 21.3°C, ventricular driving 20.3°C) are not significantly different from the control values. It would appear, therefore, that whatever the factors which precipitate VF, they do not include arrhythmic impulse discharge to the ventricles from the S-A node, nor impaired A-V transmission.

The duration of the QRS complex is generally regarded as an index of intraventricular conduction time. With a ventricular pacemaker the path of excitation is predominantly along undifferentiated myocardium, whereas under auricular driving the impulses spread via the Purkinje network to the myocardium. If this be true then the plot of figure 2 indicates that conduction is the same for the Purkinje system and the myocardium.

In a review of the cardiovascular effects of hypothermia, Brown (6) mentions that Stephens et al. (7), employing artificial pacemakers, maintained heart action in dogs cooled to 12°C. This, however, is misleading since only one of seven dogs so treated survived, not to 12°C but to 14.7°C. The remainder all succumbed to VF at higher but unstated temperatures. In all seven animals the driving was initiated only after asystole had occurred at the abnormally high mean temperature for asystole of 21.4°C.

FIG. 2. Duration of QRS complex vs. heart temperature during progressive hypothermia. A, auricular and V, ventricular driving.
REFERENCES