THE INFLUENCE OF EPIDURAL ANAESTHESIA ON CARDIAC EXCITABILITY IN PROFOUND HYPOTHERMIA

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IN NON-ANAESTHETIZED PATIENTS who are subjected to a lethal level of hypothermia, the cause of death is believed to be related to autonomic reflexes which might induce ventricular fibrillation. It has been shown that surgical sympathectomy,² sympathectomy with drugs,³ and deep general anaesthesia⁴ decreased the occurrence of hypothermic ventricular fibrillation. In this laboratory we demonstrated that with total epidural anaesthesia, animals can be cooled to lower temperatures before their hearts fibrillate.⁴⁻⁶

The purpose of this investigation was to ascertain the relationship between the observed protection of sympathetic block and ventricular excitability during hypothermia.

METHOD

Twenty mongrel dogs were used in this series of experiments. Thiopentone 20 mg/kg was administered intravenously for induction. After endotracheal intubation, the animals were ventilated artificially with 100 per cent oxygen using a Bird respirator (Mark 8). Succinylcholine 10 mg was administered intravenously every 20-30 minutes. Hypothermia was obtained by extracorporeal blood cooling using a technique described in detail elsewhere.⁶ In brief, the femoral artery and vein were cannulated, blood was taken from the femoral artery (10 cc/kg/minute), cooled to 20°-25° C, and returned to the femoral vein. This shunt was sustained until the oesophageal temperature reached 27°-30° C. At this temperature, the direction of the shunt was reversed, the blood flow was increased to 20 to 30 cc/kg/minute, and maintained until an oesophageal temperature of 10° C was achieved. The extracorporeal system was primed with blood obtained from donor dogs. Oxygen 100 per cent was used in the oxygenator. Artificial respiration was discontinued below 25° C of oesophageal temperature. Lead 2 of the ECG, the fronto-occipital lead of the EEG, the oesophageal temperature, the arterial blood pressure, and the venous blood pressure were recorded with a Sanborn 150 polygraph. Statham transducers were used for pressure determinations. Five millilitres of arterial blood were withdrawn at normal temperature, and at 35° and 5° intervals thereafter for pH and pCO₂ determinations. The experiment was discontinued when ventricular fibrillation occurred or the oesophageal temperature reached 10° C.

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To determine the excitability of the ventricle, the chest was opened and a bipolar electrode was placed in the left ventricular wall. An electrical stimulus of 2-msec duration and variable current was applied to the ventricular myocardium to obtain a premature ventricular contraction. The strength of the current varied according to its temporal relationship with the preceding R wave and the excitability of the left ventricle, this technique is described elsewhere.⁵

For control determinations, hypothermia was induced in 15 succinylcholine-paralysed and lightly anaesthetized animals. Determinations of cardiac excitability were made throughout the cooling period.

In five animals, epidural anaesthesia with lidocaine 5 mg/kg of 1 per cent solution was administered through a catheter placed in the epidural space and similar determinations of cardiac excitability were performed. Hypothermia was induced as in the control group.

RESULTS

The ventricular diastolic threshold (minimal electrical current needed to produce an extrasystole during the electrical diastole) of the 15 control animals had wide oscillations. It was higher than control at temperatures above 25° C. It decreased sharply below this temperature in some experiments. In contrast, this threshold was stable in the epidural animals, with a small increase under 25° C (Fig 1, Table I).

In all control animals, ventricular fibrillation was started when testing with

FIGURE 1 Relative and absolute changes of the various strength-interval curve periods as a function of temperature

TABLE I

	T R P (msec)		T R P (%)		V D T (mamp)		H R (B/min)	
	C	E	C	E	C	E	C	E
Control	170	200	45.9	50	0.10	0.125	162	150
Epidural	—	204	—	37.1 ^o	—	0.124	—	109 ⁺
35° C	222 ⁺	298 ⁺	49.3	42.0 ^o	0.13	0.116 ^o	133 ⁺	85 ⁺
30° C	288 ⁺	356 ⁺	57.6	41.4 ^o	0.14	0.120 ^o	119 ⁺	70 ⁺
25° C	396 ⁺	442 ⁺	60.9	39.8 ^o	0.15	0.136 ^o	93 ⁺	54 ⁺
20° C	471 ⁺	720 ⁺	62.0	46.8 ^o	0.13	0.132 ^o	79 ⁺	39 ⁺
15° C	533 ⁺	783 ⁺	47.2	39.2 ^o	0.14	0.16 ^o	53 ⁺	30 ⁺

T R P (msec)—total refractory period in milliseconds

T R P (%)—total refractory period as a percentage of the heart cycle

V D T (mamp)—ventricular diastolic threshold in milliamperes

H R (B/min)—heart rate, beats per minute

C = control, E = experimental

^o $P < 0.02$, experimental group against control group

⁺ $P < 0.02$, for the same group

diastolic threshold levels of current applied during the last 20 msec of the relative refractory period (vulnerable period⁷). It occurred at a higher temperature (24° C to 15° C) in those animals with lower ventricular diastolic threshold.

All dogs in the control group suffered ventricular fibrillation at temperatures between 24° C and 11° C. In five animals of the epidural group only two fibrillated (13° C and 11° C).

The epidural group had a steady decrease in heart rate between 35° C and 20° C (3 beats per degree). The decrease was sharper (6 beats per degree) for the first two degrees (37° to 35° C) and slower for the last five (20° to 15° C). The control group had marked heart-rate changes of no definite pattern (Fig 2, Table I). This rate was higher than in the epidural group at any given temperature. The mean blood pressure had a slow hypothermic drop in the control group, but was rapidly decreased in the epidural group.

The length of the QRS, at any given temperature, showed no difference between the control and the epidural group.

Cooling the epidural group had no effect on the percentage of the heart cycle components—absolute and relative refractory periods, and diastolic period. They were greatly modified in the control group (Fig 1).

DISCUSSION

The protection offered by epidural anaesthesia against hypothermic ventricular fibrillation was partial, and it was not related to the ventricular diastolic threshold changes despite its stability in the epidural anaesthesia group when compared to the controls (Table I).

Goldberg reported the occurrence of hypothermic arrhythmias at higher temperatures with sympathetic block. Israel,⁹ using total spinal anaesthesia, found no protection against cardiac arrhythmias induced by the administration of epinephrine. In the former study, a veno-venous shunt of extracorporeal cooling was used without circulatory support, while in the latter, the administration of epinephrine as a test of cardiac excitability has questionable meaning.

FIGURE 2 Heart rate and blood pressure changes as a function of temperature

Sympathetic block would be expected to protect against the endogenous release of catecholamines but not against their administration

Two principal causes of hypothermic ventricular fibrillation may be considered those initiated by external stimuli, and those occurring spontaneously. The former, or induced, may originate in a surgical manoeuvre, an electrical stimulation, or a reflex mechanism and is reflected in the ventricular fibrillatory threshold. The latter, or spontaneous, may originate from a metabolic change¹⁰ and can be studied through ventricular diastolic threshold determinations. Both causes imply modifications in cardiac excitability.

The ventricular diastolic threshold (VDT) and the fibrillatory threshold (FT) are affected differently by hypothermia. The former (VDT) was unchanged or increased at temperatures over 20° C and decreased below this temperature, while the latter (FT) decreases with decreasing temperature.

Szekeres,¹¹ comparing thresholds and arrhythmias in the heart *in situ* with those in the heart-lung preparation, also suggested the existence of two kinds of hypothermic ventricular arrhythmias. Hypothermic dogs under epidural anaesthesia behave similarly to heart-lung preparations. The V D T, seems to be related to the spontaneous hypothermic ventricular fibrillation and the F T to the induced hypothermic ventricular fibrillation.

The diastolic threshold reflects a cellular property. Cardiac arrhythmias originating from its modifications may be considered as of spontaneous occurrence. This threshold was higher in moderate hypothermia (over 25° C), and agrees with previous observations in which the incidence of hypothermic spontaneous cardiac arrhythmias was decreased,^{12,13} or even disappeared after its induction.¹⁴

The fibrillatory threshold is the myocardial response to a potent artificial stimulus applied towards the end of refractoriness—the vulnerable period.⁷ In normothermia this threshold is high (10–20 mamp) and the vulnerable period is short (5–40 msec). In hypothermia this period becomes longer and simultaneously the threshold falls to diastolic levels explaining the ease of induced hypothermic ventricular fibrillation.¹¹

The coexistence of a lower fibrillatory threshold, a longer relative refractory period, and an ectopic focus as a triggering point, sets the stage for the higher incidence of hypothermic-induced ventricular fibrillation. These ectopic foci have been explained by various mechanisms such as uneven cooling of the myocardium,¹⁵ uneven oxygenation, cold stress, anoxia, or hypercarbia. Shumway¹⁶ reported the existence of particular areas of the myocardium in which the ventricular diastolic threshold was lower under special circumstances. Epidural anaesthesia protects against ventricular fibrillation subsequent to cold stress or hypercarbia, by preventing a reflex sympathetic stimulation.

Lower metabolism implies disequilibrium of the intracellular/extracellular relationship of electrolytes, which may account for the higher incidence of spontaneous ventricular fibrillation under 20° C. Quinidine is supposed to protect at this level. Probably epidural anaesthesia provides no protection against this type of ventricular fibrillation.

The total refractory period was increased, percentage wise, in the control group, while practically no change was found in the epidural group. In absolute values, the total refractory period increased with the decrease in temperature in both groups. Covino¹⁷ stressed the importance of the ratio speed of conduction in the myocardium/ventricular refractoriness (QRS/TRP). In our study, the epidural group had a proportional increase in refractoriness and QRS duration and the same proportional decrease in heart rate for any given temperature. In the control group, the changes were unpredictable. This harmony in the epidural group seems to be a manifestation of the protective mechanism reported by Covino.

SUMMARY

The effect of total sympathetic block by epidural anaesthesia was studied during profound hypothermia. It protected against the incidence of ventricular

fibrillation over 20° C This protection seemed to be mediated through a steady equilibrium of the heart rate, duration of QRS, and the length of refractoriness (TRP) The ventricular fibrillatory threshold became lower and equal to the ventricular diastolic threshold in the animals that fibrillated This threshold (VDT) had wide changes in the control group and decreased sharply below 25° C in the experiments in which ventricular fibrillation occurred at higher temperatures However, ventricular diastolic threshold alone does not explain the protection obtained from sympathetic block Sympathetic block protected against reflex factors in induced ventricular fibrillation Total epidural anaesthesia during hypothermia produced marked arterial hypotension

RÉSUMÉ

Nous avons étudié l'effet du blocage sympathique total durant l'hypothermie profonde Il a protégé contre l'incidence de fibrillation ventriculaire au-dessus de 20° C Cette protection semblait être obtenue par un équilibre stable de la vitesse du cœur, d'après le complexe QRS, et par la durée de la réfraction (TRP) Chez les animaux qui faisaient de la fibrillation, le seuil de la fibrillation ventriculaire s'abaissait et devenait égal au seuil diastolique ventriculaire Ce seuil (VDT) variait beaucoup pour le groupe de contrôle et s'abaissait subitement au-dessous de 25° C, lors des expériences où la fibrillation ventriculaire se produisait à des températures plus élevées Cependant, le seuil diastolique ventriculaire seul n'explique pas la protection obtenue par le blocage sympathique Le blocage sympathique protégeait contre les réflexes dans les cas de fibrillation ventriculaire provoquée L'anesthésie épidurale totale durant l'hypothermie a produit une hypotension artérielle importante

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