EFFECT OF PERIDURAL HEART BLOCK ON THE THRESHOLD OF VENTRICULAR FIBRILLATION IN DOGS

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The threshold of ventricular fibrillation in animals with an intact heart and myocardial infarction was determined by injection of trimecaine into the peridural space at the T_1-T_2 level. The threshold of fibrillation was shown to be increased under these circumstances on account both of the resorptive action of trimecaine and the blockade of the sympathetic nerves to the heart.

KEY WORDS: denervation of the heart; peridural anesthesia; ventricular fibrillation; threshold of ventricular fibrillation; myocardial infarct; trimecaine.

Peridural anesthesia (PA) is finding ever-increasing use as a component of the protective measures in anesthesiology during operations on the heart. PA was first used in the writers' clinic for the relief of pain in acute myocardial infarction in 1972. It was observed that, besides its analgesic action, PA also has an antifibrillatory action. For instance, in more than 300 patients treated by PA during the first five days of the disease, primary ventricular fibrillation (VF) was never once observed although, according to records of the writers' clinic for many years, it arises in 2-4% of patients during this time period.

It was accordingly decided to make an experimental study of the effect of PA on the predisposition of the heart to VF, more especially because no data on this problem could be found in the literature. A predisposition of the heart to VF was established by determining the threshold of VF (TVF) during electrical stimulation of the heart.

EXPERIMENTAL METHOD

Acute experiments were performed on 36 mongrel male dogs weighing 18-29 kg. After intramuscular injection of listhenon in a dose of 1-2 mg/kg the animal was incubated and artificially ventilated throughout the experiment by means of the DP-8 apparatus on a semiopen circuit. Hexobarbital, 8 mg/kg, was injected intraperitoneally. Left-sided thoracotomy was performed. A negative electrode was inserted to the left of the interventricular artery at the level of its middle third into the substance of the myocardium, and a positive electrode into the soft tissues of the chest wall. In the experiments in which a myocardial infarct was produced in the dogs the positive electrode was inserted into the infarcted area. The electrodes were connected to an ÉSL-2 electrical stimulator, activated by means of a special device whereby a single electric pulse could be applied to the heart in the so-called vulnerable phase of the cardiac cycle – the ascending limb of the T-wave (Fig. 1) – by means of the synchronizer of the cardiomonitor. The fact that the pulse was applied in the "vulnerable" phase was verified by the ECG. To determine TVF, a single pulse 100 msec in duration was applied every 3 min to the heart; its voltage increased from an initial 1 V to a final 60 V. The voltage of the pulse at which VF appeared was regarded as TVF.

PA was carried out as follows. Two needles were inserted into the peridural space at the levels of T_4 and T_5. Confirmation that the needles had entered the peridural space was given by: 1) absence of any flow of warm, opalescent CSF from them, 2) a flow of colored, cold liquid, injected into one needle, from the other needle, and 3) the unobstructed passage of a catheter in the oral direction as far as the level T_1. The needles were removed and 2.5% trimecaine solution was injected through the catheter into the peridural space in a dose of 0.3 mg/kg.

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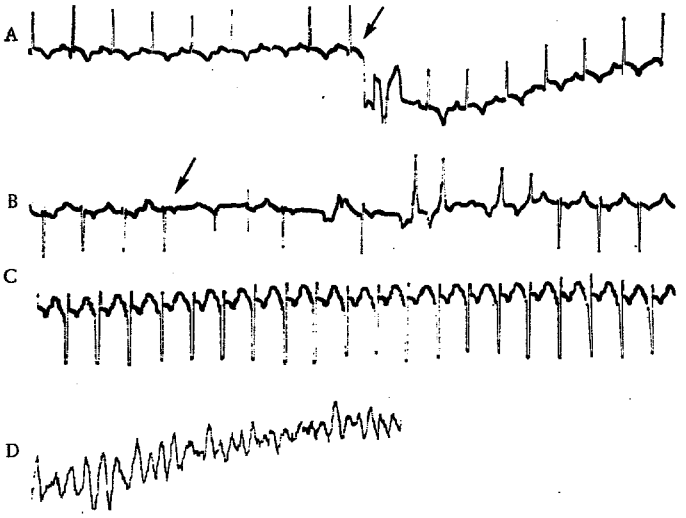

Fig. 1. ECG during determination of TVF. A) Initial ECG-arrow marks verification that pulse is applied in "vulnerable" zone; B) ligation of coronary artery (arrow); C) 10 min after ligation; D) VF after application of "threshold" pulse.

Altogether two series of experiments were carried out, with three groups in each series. In series I the effect of PA on TVF was studied in dogs with an intact heart, in series II in dogs with an experimental myocardial infarct caused by ligation of the large left branch of the interventricular artery. In the first control groups of each series of experiments (on 6 dogs in each group) TVF was studied in the absence of any additional pharmacological or other procedures on the animals. In the second groups of each series (6 dogs in each group) TVF was studied starting from 30 min after intraperitoneal injection of trimecaine. It was assumed in this case that 30 min was a long enough time for trimecaine to be absorbed through the peritoneum into the blood stream and to exhibit its resorptive action. In the third groups of each series (6 dogs in each group) TVF was determined 15 min after PA. This period was chosen because, when trimecaine is injected into the peridural space, a sympathetic block and blocking of hot and cold sensation develop initially. Complete blocking of pain sensation is observed after 15-30 min [3]. The local anesthetic, when absorbed by the small vessels – terminal arterioles, capillaries, and venules – appears within a few minutes in appreciable amounts in the blood stream, where it reaches its maximal concentration between 15 and 30 min [4-6]. The general resorptive action of the anesthetic, on the one hand, and PA on the other hand, are associated with these processes.

EXPERIMENTAL RESULTS

In animals with experimental myocardial infarction TVF was lowered by more than 80%, i.e., as was to be expected, the electrical instability of the myocardium was increased (Table 1). Trimecaine, when injected intraperitoneally, exerted a resorptive action which was manifested as an increase of 40% or more in TVF both in intact animals and in animals with a myocardial infarct (Table 1). Local anesthetics are known to prevent the passage of Na and K ions through membranes. It is on this property of local anesthetics that the blocking of sensory nerves is based. Local anesthetics have a similar action on myocardial cells. In the modern view trimecaine, like lidocaine, depresses spontaneous depolarization of Purkinje fibers, reduces automatism, and lengthens the refractory period of heart muscle [7-9], and these effects are probably manifested as an increase in the level of TVF.

Comparison of the results obtained in the second and third groups of each series shows that a high PA raises TVF both in intact animals and in animals with myocardial infarction by 2-5 times, i.e., much more than intraperitoneal injection of trimecaine. This effect of PA on TVF is brought about, as these experiments show, in two ways: 1) through the resorptive action of trimecaine and 2) through actual blockade of the spinal nerve fibers.

Data in the literature on the effect of surgical denervation of different parts of the heart on the frequency of experimental VF are somewhat contradictory. According to some reports [1] in experimental myocardial

TABLE 1. Effect of PA on TVF in Dogs with Intact Heart and with Myocardial Infarction

Series	Characteristics of group	TVF in each experiment, V						$M \pm m$	P
I. Dogs without myocardial infarction	Control	20	35	30	35	35	30	30,83±5,9	
	Intraperitoneal injection of trimecaine	45	40	45	40	50	35	42,5±5,2	$P_{1-2}<0,001$
	PA	60	50	60	55	60	55	56,66±4,0	$P_{2-3}<0,05$
II. Dogs with myocardial infarction	Control	5	6	2,5	7	3	10	5,58±2,8	$P_{I_1-II_1}<0,05$
	Intraperitoneal injection of trimecaine	10	14	12	14	12	14	12,66±1,6	$P_{1-2}<0,05$
	PA	17	20	14	18	35	45	24,83±12,3	$P_{2-3}<0,02$

infarction stellate ganglionectomy and resection of T_1-T_5 increased the frequency of VF. Conversely other workers [7] reported that after ligation of the circumflex artery in sympathectomized dogs VF appeared in 22% of cases, compared with 52% in intact dogs. Another group of workers [10] showed that TVF in dogs was lowered by 33% in coronary occlusion, by 42% during stimulation of the stellate ganglion, and by 63% during a combination of both. After removal of the stellate ganglion TVF was lowered by only 11% after ligation of the coronary artery. The results of the last-mentioned authors are identical in many respects with those of the present experiments.

The problem of which spinal nerve fibers it was whose blocking by PA was responsible for the changes in TVF, thus arises. Analysis of the results is made difficult by the fact that neither the level nor the number of spinal ganglia which send their efferent fibers to the heart has yet been established [2]. We know that PA blocks ventral efferent preganglionic (sympathetic) and also afferent, dorsal sensory fibers [11]. The increase in TVF during PA can evidently be linked with blockade of postganglionic sympathetic fibers running to the heart, which could take place theoretically in the following ways: through a decrease in the concentration of sympathetic mediators (catecholamines) in the heart and through dilation or abolition of spasm (by the infarct) of the coronary arteries and collaterals in the zone surrounding the infarct. Catecholamines, whose level is increased in myocardial infarction, are known to promote the onset of arrhythmias. Catecholamines and, in particular, adrenalin depress TVF. Under the influence of catecholamines, whose concentration in the myocardium is higher in arrhythmias than in normorhythmia, the potassium ion concentration in the heart muscle falls whereas the sodium ion concentration rises. In the modern view, VF is largely attributable to migration of potassium ions from the myocardial cells into the extracellular medium and the shift of sodium ions from the extracellular medium inside the cells.

It can also be tentatively suggested that in the zone of sympathetic denervation the parasympathetic coronary-dilating effect is strengthened, with the development of dilatation of the coronary arteries [4] and collaterals, which could lead to an improvement of the blood flow both in the intact myocardium and, in myocardial infarction, in the zones surrounding the infarct and may increase the electrical stability of the myocardium.

The experimental data described above, confirmed by clinical observations, thus suggest that PA in acute myocardial infarction can be used not only as a highly effective method of analgesia, but also for its antifibrillatory action.

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