Table 1 The effects of dobutamine (10 μg kg^{-1} min^{-1}) following acute coronary artery occlusion in anaesthetized greyhounds

	Pre-dobutamine	*Post-dobutamine*
Mean systemic blood pressure (mmHg)	133 ± 3	134 ± 3
Heart rate (beats/min)	188 ± 13	210 ± 10*
LV dP/dt max (mmHg/s)	2278 ± 225	3313 ± 297*
LVEDP (mmHg)	7.7 ± 1.3	5.5 ± 1.2*
Cardiac output (l/min)	2.2 ± 0.4	2.6 ± 0.3*
Transventricular driving pressure (mmHg)	3.5 ± 2.8	8.2 ± 3.2*
Myocardial blood flow (normal region (ml/min)	75 ± 12	96 ± 14*
Coronary sinus oxygen content (ml/100 ml)	9.7 ± 0.5	12.4 ± 0.3*
Myocardial blood flow (ischaemic region) (ml 100 g^{-1} min^{-1})	27 ± 6	34 ± 6*
Coronary vein oxygen content (ml/100 ml)	8.5 ± 0.1	13.1 ± 1.3*

* $P < 0.05$ Paired t test.

dobutamine-induced increases in myocardial contractility and work did not extend the ischaemic area since epicardial S-T segment elevation was slightly reduced or remained unchanged. In contrast, isoprenaline in a dose (0.1 μg kg^{-1} min^{-1}) that increased dP/dt max. to the same degree, consistently *increased* S-T segment elevation, an effect related to the more pronounced tachycardia. Dobutamine and isoprenaline also differed in their effects on local myocardial temperatures, dobutamine causing significantly smaller increases especially in the subendocardial layers of the infarcting zone.

These effects of dobutamine on blood flow, temperature and S-T elevation may result from an increase in transventricular pressure (Marshall & Parratt, 1974) and thus favour subendocardial blood flow.

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Comparative effects of chloralose and pentobarbital on atrioventricular conduction in the dog

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Two electrodes were implanted in mongrel dogs of either sex: one at the upper end of the atrioventricular node, the other 2 cm away in the atrial myocardium. The electrode leads emerged in the region of the back of the neck. It was then possible to study in the conscious dog atrioventricular conduction by stimulating the atria at increasing frequencies in successive stages. In the unanaesthetized animal at rest, there was a difference in the sensitivity to the atrial stimulation depending on the stage of the respiratory cycle: minimal (135 ± 15 beats/min) during expiratory bradycardia, maximal (240 ± 14 beats/min) during inspiration.

Chloralose (0.8 g/kg i.v.) did not substantially modify sinus rate or atrioventricular conduction ($n = 7$). It did not alter these two stages but decreased the positive chronotropic effects and to a lesser extent, the dromotropic effects of atropine (0.1 mg/kg i.v.).

Pentobarbital (25 mg/kg i.v.) suppressed

respiratory arrhythmia and its phasic influence on conduction. It decreased both the positive chronotropic and dromotropic influence of atropine.

In conclusion, pentobarbital is contraindicated as an anaesthetic for the study of atrioventricular conduction in the dog. Although chloralose seems preferable, it also attenuates if to a lesser extent, the facilitating dromotropic influence of atropine.

Heterogeneous reactivity of atrial and ventricular cardiac contractile tissue

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In order to investigate induced changes in the effective refractory period of the atrial and ventricular cardiac muscle by means of cardiotropic drugs, the interventricular septum was removed in 24 mongrel dogs under chloralose anaesthesia and during total cardio-pulmonary by-pass to obtain independent atrial and ventricular contractions. The comparison of the variations of the effective refractory period in the right atrium and in one of the ventricles was checked by the extra-stimulus method, the contractile fibres being directly under the dependence of an electric pacer (frequency: 150/min) to prevent rhythm-induced changes of the refractory period.

Acetylcholine (0.5, 1 and 2 mg kg^{-1} min^{-1}) considerably shortened the effective refractory period of atrial cardiac fibres ($P < 0.05$), but did not provoke any change in the ventricular contractile tissue.

Ouabain (30, 40 and 50 μg/kg) and quinidine (5, 10 and 20 mg/kg) induced larger variations in the refractory period of atrial contractile tissue than in the ventricular tissue.

Nevertheless, the heterogeneous reactivity of the contractile tissue, present both in the atrium and in the ventricle, is not a general law. As a matter of fact, the isoproterenol (0.5, 1 and 2 μg/kg/min)-induced shortening of the effective refractory period for both atrial and ventricular tissue is not significantly different.

A method for experimental study of arrhythmias following myocardial infarction in closed-chest dog. Its use as a test for antiarrhythmic drugs

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A small metallic coil was wedged distally either in the anterior interventricular artery or in the circumflex artery through coronary catheterization under X-ray control. While the animal recovered from anaesthesia a thrombosis developed progressively. The ECG was monitored by telemetry on a 24 h basis. All changes of the cardiac rhythm and/or of the QRS complexes were analysed on a specially built electronic device.

The method has been applied to 50 dogs on which a total of 55 infarctions were elicited. All animals but one showed ventricular arrhythmias. Two of them died from ventricular fibrillation. More than half of the dogs had episodes of ventricular tachycardia. In all cases trains of ventricular extrasystoles were observed. The delay in onset of the arrhythmia was variable, ranging from 70 min to 30 hours. The duration of the periods of arrhythmia ranged from one to six days.

Ajmaline, amiodarone, phenytoin, disopyramide, quinidine, lidocaine, procainamide, propranolol and verapamil were administered during various types of ventricular arrhythmias.