THE PRODUCTION OF VENTRICULAR ARRHYTHMIAS FOLLOWING LIGATION OF THE RIGHT CORONARY ARTERY IN DOGS

Sir,

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Right ventricular infarction and associated ventricular arrhythmias, once considered rare, are now commonly recognised in patients with inferior wall infarction and clinical data is strongly suggestive of that cardiovascular changes occurring with human myocardial infarction vary importantly depending upon the site of myocardial infarction. Sympathetic hyper-activity is associated with the anterior wall infarcts while posterior wall infarcts lead to increased parasympathetic activity. Prevalence of second and third degree heart block is more frequently associated with the posterior wall infarction than after anterior wall infarction (1-5). The ventricular arrhythmias associated with inferior wall infarction responded very well to bretylium. However the left ventricular tachycardia associated with anterior myocardial inchaemia or infarction did not (6).

Harris’s (7) method of ligating the anterior descending branch of left coronary artery to produce left ventricular tachycardia is well established. However, to the best of our knowledge no method for the production of experimental right ventricular tachycardia is available in the literature. Therefore, the present study was undertaken to develop an experimental model for producing right ventricular tachycardia by ligating the right coronary artery, which can be quite useful for studying the differential effect, if any, of drugs on selective ventricular tachycardias, originating from either side of the heart.

Mongrel dogs of either sex (8-27 kg), were anaesthetised with pentobarbitone sodium 30 mg/kg intravenously. The trachea was intubated and positive pressure artificial respiration was instituted. ECG lead II was recorded. The animals were divided into 2 groups.

In one group of experiments (n=4) the coronary artery was ligated at a distance of 15 mm away from the right atrio-ventricular groove following the method of Harris (7), while in another group of 7 dogs the right coronary artery was ligated at the atrio-ventricular groove itself.

None of the animals from group I developed spontaneous ventricular arrhythmias during 72 hrs of observation period. However, ECG record showed raised ST segment and inverted T wave. These animals were challenged with small dose of adrenaline (3 µg/kg) intravenously to confirm the development of myocardial infarction. All these animals developed a paroxysm of ventricular tachycardia (PVT) for a brief period of 3-5 min. However, adrenaline did not evoke any ventricular arrhythmias in the control animals.

**TABLE 1**: Development of ventricular tachycardia following two stage ligation of right coronary ligation in the dog at the atrio-ventricular groove.

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All the 7 animals from the second group developed spontaneous ventricular arrhythmias with a mean ventricular tachycardia rate of 196 ± 7.23 beats/min. The time course of development of arrhythmia is presented in Table I.

The optimal time to test antiarrhythmia drugs is between 18-24 hrs. Ligating the right coronary artery 15 mm away from the atrio-ventricular groove produced myocardial infarction but no ventricular arrhythmias which was confirmed through the adrenaline challenge. However, ligation of the right coronary artery at the atrio-ventricular groove evoked spontaneous ventricular arrhythmias indicating that the infarction size was sufficient to evoke arrhythmias but inadequate when the ligation was 15 mm away from the atrio-ventricular groove.

The present study indicates that the new model can be useful in studying the differential effects, if any, of antiarrhythmic drugs in selective right or left sided experimental ventricular arrhythmias.

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REFERENCES


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