Pakistani J. Pharm. Sci. 41-45, 1988.

CHANGES IN E.C.G. AND ELECTROLYTES DURING CORONARY LIGATION IN ANAESTHETIZED CAT*

SHAHID RASHID

Department of Pharmacology, Faculty of Pharmacy,
University of Karachi, Karachi-32, Pakistan.

ABSTRACT

The ligation of the coronary artery produces cardiac, and haemodynamic disturbances. Electrolytes disturbances were also noted during the assault (Harris et. al., 1954).

In present experiments local cardiac ischaemia was produced by coronary ligation and changes in E.C.C. and electrolytes were noted at various stages. Premature ventricular systoles and T wave inversion with an increase in K^+ and decrease in Na^+ was observed.

Introduction

The theory advanced by Harris et. al. in 1954 that K^+ is liberated from the ischemic and dead cardiac muscle cells diffuses in the blood and than during circulation reaches to the peripheral ischemic zone where it acts as a stimulating agent and causes the development of dysrhythmia.

It is suggested that catecholamine initiated dysrhythmias may be due to the in. creased permeability of the cell membrance to K^+ (Regan et. al 1967). Moreover it is repotted that following the occlusion of the left coronary artery plasma K^+ was found to be increased after six and twelve hours.

In present studies local cardiac ischemia was produced by coronary ligation and K^+ and Na^+ were measured in plasma. During this process of ligation E.C.G. were also monitored at different stages such as control; after thoracotomy, after temporary and permanent ligation etc. and at various time intervals after ligation. Saturation of 0r in arterial blood was also measured.

Procedure

Five cats of either sex weighing 1.6 – 3.0 kg. were anaesthetized with pentobarbitone Na (30 mg/kg intrathoracically) and prepared for recording as described by Alps, et al (1972). E.C.G. lead II (standard) was used throughout these studies which were recorded on Grass polygraph Model 7. Rectal temperature was measured by means of glass thermometer. 2m1. of bloods ampler were collected in heperanised vials for the determination of Na^+ and K^+ on an autoanalyser (Technicon, Method No. N-20b). O_2 saturation in the blood was measured by an oximeter (waters).

---

* This work was carried out at Wyeth Laboratories, Taplow — Berks.

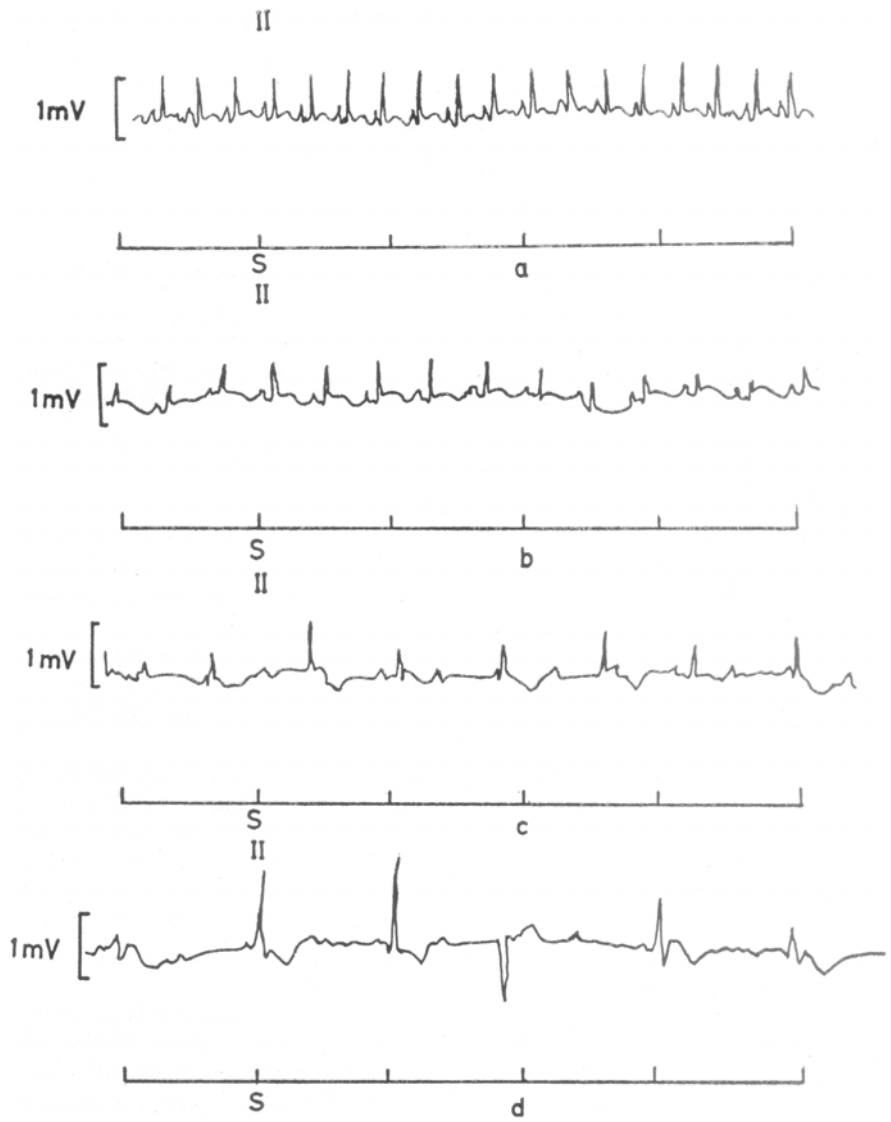

Fig. 1. Changes in E.C.G. during the procedure of coronary ligation are shown in lead II at 2.5 mm/Sec. which are as follows:
a) Control E.C.G. b) After opening the chest, note the reduction of voltage of QRS. c) Fifteen minutes after thoracotomy and first stage ligation. Inversion of T wave and A – V dissociation is prominent. d) 15 minutes after permanent ligation PV S and inversion of T wave.

The preparation was left for 30 minutes for estabilization and then control readings were taken. The chest was opened through midstemum using diathermy. Respiration was artificially maintained by a Palmer respiratory pump (30-35 cc/min.).

Experiment was further left for 15 minutes after thoracotomy but before ligation and parameters were again recorded. The pericardial cradle was formed and left descending coronary artery was separated and prepared for ligation. Two stage ligation technique (temporary and than permanent) was used (Harris 1948) and parameters again recorded. No drugs were used throughout these studies.

Results and Discussion

Changes in E.C.G.: In all experiments E.C.G. waves were monitored at four stages control, after thoracotomy, after 1st stage of ligation and 15 minutes after permanent ligation. The features evident in all the experiments were the lowering of voltage (after thoracotomy) and inversion of T wave. Apart from these changes, in one experiment premature ventricular systole and in another A-V dissociation was recorded. These cats survived upto one to three hours after ligation of the coronary artery. Typical changes of E.C.G. are shown in Figured.

In one typical experiment a sudden inversion of T wave was seen just after 1st stage ligation. No reversal in E.C.G. configuration was seen even this ligation was removed. After permanent ligation alongwith inversion of T wave ectopics have been recorded. After 15 minutes of the ligation frequent ectopics were noticed and at 30 minutes ventricular fibrillation episodes were evident. Cat died one hour after permanent ligation due to ventricular fibrillation.

Electrolytes changes: Mean values with standard errors of Na^+ and K^+ (in mol/L) in five experiments have been shown in Table (1a) at 4 points. A gradual though statistical non significant decrease in Na^+ while as an increase in K^+ level has been observed. In one typical case significant changes in electrolytes have been recorded which are also shown in the Table (1b).

O_2 % in arterial blood was suddenly decreased after opening the chest but level was maintained after subsequent stages (Table la).

A sudden obstruction of any major artery produces ischemic pain. This is particularly true in the case of coronary artery and in clinical blockade of this vessel produces angina and in severe case heart attack and even death.

The cause of death under these circumstances may be the precipitation of dysrhythmias due to electrolyte disbalance and accumulation of Co. Other probable cause is the lack of oxygen in ventricular muscle leading to necrosis. These both causes go hand in hand with others such as catecholamine release after occlusion of coronary artery.

In the present findings the K^+ level increased in blood after thoractomy and ligation while a decrease was observed in Na^+ which shows a retention of Na^+ in the cells. These changes go together with the inversion of T wave and then ectopics which is in support of the theory that K+ liberated from ischemic and dead cardiac cells, diffuses

into the blood and reaches the heart muscle where it acts as a stimulating agent and causes the development of ectopics (Harris et. al 1954).

Table 1(a). Showing the electrolytes measured in Mood before and after the coronary ligation. O_2 concentration of blood in percentage is also shown. Each value represents the mean of five experiments.

Electrolytes & O_2	Control	Just after thoracotomy	15 minutes after throracotomy	15 minutes after ligation
Na^+ (m. mol/L)	148 ± 5.00	141 ± 4.00	142 ± 3.00	143 ± 4.00
	N.S.	N.S.	N.S.	N.S.
K^+ (m. mol/L)	2.96 ± 0.24	2.96 ± 0.24	3.02 ± 0.17	3.30 ± 0.44
O_2 (%)	94 ± 1.14	90 ± 1.97	92 ± 1.93	92 ± 2.0

Table 1(b). Showing the electrolyte values in one of the typical experiment

Na^+ (m. mol/L)	154	154	158	153
K^+ (m. mol/L)	2.35	2.70	2.50	3.80

The same theory was also supported by the experiments in dogs by Sergievsky & Invashkevich (1963), when an increase in blood K+ was observed in dogs whose heart were subjected to coronary ligation.

It is well established that electroytes play an important role in producing dysrhythmias (Surawicz 1972). Hyperkalemia alter cardiac rhythm, conduction velocity, increase the peak amplitude of the T wave (Rashid 1976). In present studies E.C.G. changes specially inversion of T wave could he due to injury current and excess release of K^+ from the infarcted areas of the ventricles.

References

Alps, B.J.; Hill, M., Johnson, E .S. and Wilson. A .B. (1972). Br. J. Pharmac. 44, p.52.
Harris, AS. (1948). Am. Heart. J. 35, p.895.
Harris, AS., Bislini, A., Russel, R.A., Bringham, J.C. and Friestone, J E. (1954). Science 119, p.200.

Rashid, S. (1976). Experimental studies on antidyshythmic drugs with special reference to analgesics. Ph.D. thesis Univ. of Bradford.
Regan, T.J., Harmann, M.A., Burke, W.M. and Aldewurtel, H.A. (1967). J. Clin. Invest. 46, p.1657.
Sergievsky, V.S. and Invashkevich, E.I. (1963). Pat. Biziol ek sp. Er. 7, p.37.
Surawicz, B. (1972). In cardiac arrhythmias. A symposium Ed. Han. J. Charles C. Thomas, Illionis, p.211-2.