SUMMARY AND CONCLUSION

In this study the effects of the calcium channel blocker "verapamil" and PGF2α on myocardial preservation during experimental acute myocardial infarction were studied.

The research was performed on adult Sprague - Dawely rats weighing 250 - 300 gm of either sex under urethane anaesthesia . The rats were classified into two main groups. The first main group included rats which underwent coronary artery ligation for 6 hours. This group was further subdivided into a control group which did not receive any premedications, a calcium channel blocker treated group receiving verapamil, a calcium gluconate treated group receiving calcium gluconate, a PGF2α treated group receiving PGF2α and an indomethacin treated group receiving indomethacin.

In the treated rats all drugs were given immediately after coronary ligation. The calcium channel blocker verapamil was given intravenously in a dose of 0.01 mg/100 gm rat body weight. The calcium gluconate was given intraperitoneally in a dose of 0.12 mg/100 gm rat body weight. The PGF2α was given intraperitoneally in a dose of 0.015 mg/100 gm rat body weight. The

indomethacin was given intravenously in a dose of 0.06 mg/100 gm rat body weight.

The parameters used were the T wave voltage, T wave area, S CPK and infarction size. The ECG parameters were recorded before coronary ligation and at 30, 60, 120 minutes and 6 hours after coronary ligation. The S CPK and infarction size were estimated at 6 hours after coronary ligation.

In this group of permanent coronary ligation, no beneficial effect of the calcium channel blocker verapamil was manifested while the deleterious effect of calcium gluconatewas manifested on ECG parameters at 30, 60, 120 minutes and 6 hours after coronary ligation.

The beneficial effect of PGF2α in this group was only shown in decreasing the T wave area at 6 hours after coronary ligation.

On the other hand, the injurious effect of indomethacin was well manifested in deteriorated ECG parameters at 30, 60, 120 minutes and 6 hours after coronary ligation. Also there were significant increases in both the S CPK and infarction size after 6 hours of coronary ligation.

The second main group was the reperfusion group in which the effects of reperfusion started at different times, with and without premediactions were studied. The rats were classified into: rats subjected to 30 minutes of coronary ligation, rats subjected to 60 minutes of coronary ligation, rats subjected to coronary ligation for 90 minutes, rats subjected to coronary ligation for 2 hours and rats subjected to coronary ligation for 3 hours. Each followed by reperfusion for 6 hours. The effects of calcium channel blocker "verapamil", calcium gluconate, PGF$_{2\alpha}$, calcium gluconate and indomethacin on each class of these rats were studied.

The same doses as in group one were used in this second group. Verapamil was given intravenously, 15 minutes before onset of reperfusion as also was indomethacin. While calciun gluconate and PGF$_{2\alpha}$ were given intraperitoneally, 30 minutes before onset of reperfusion.

The parameters used were T wave voltage, T wave area, S CPK and infarction size. The ECG parameters were recorded before coronary ligation, immediately before reperfusion, 30 minutes and 6 hours after reperfusion. The S CPK and infarction size were estimated at 6 hours after reperfusion.

The beneficial effects of the calcium channel blocker "verapamil" was manifested in rats subjected to 30, 60, and 90 minutes of coronary ligation before onset of reperfusion in the form of improved ECG parameters and decreased S CPK and infarction size after 6 hours of reperfusion. When the coronary ligation was prolonged to 2 hours and/or 3 hours, this beneficial effect was lost.

Also, the beneficial effect of PGF$_{2\alpha}$ was manifested in rats subjected to 30, 60, 90 minutes of coronary ligation before onset of reperfusion. This beneficial effect was shown in improvement of ECG parameters and decreased S CPK and infarction size after 6 hours of reperfusion. When the time of ischemia before onset of reperfusion was prolonged to 2 hours and/or 3 hours the beneficial effects on S CPK and infarction size were lost while the beneficial effect on ECG parameters persisted even when the coronary artery occlusion was prolonged to 3 hours.

Indomethacin on the other hand which is a potent antiprostaglandin, had injurious effects on ECG parameters, S CPK and infarction size on all studied subgroups

The failure of reperfusion per se to reduce infarction size even when reperfusion started early after 30 minutes of coronary occlusion together with the beneficial effects of calcium channel blocker "verapamil" and PGF2α at time of reperfusion is a good prove for excistence of reperfusion injury.

It can be thus concluded that our work proved the presence of reperfusion. Calcium channel blocker "verapamil" and PGF2α have beneficial effects on this reperfusion injury provided that coronary ischemia is less than 2 hours.