Title : HIGH DOSE BARBITURATE EFFECTS ON CIRCULATORY HEMODYNAMICS AND MYOCARDIAL INFARCT SIZE IN DOGS WITH ACUTE CORONARY OCCLUSION

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Introduction. High dose barbiturate therapy has been suggested as an effective way of reducing neurologic damage following cardiac arrest. In many of these situations intrinsic ischemic cardiac disease is the cause of the circulatory arrest. In order to evaluate the influence of high-dose barbiturate therapy upon cardiovascular performance and the possible extension of a myocardial infarction, we performed the following study.

Methods. Following surgical preparation to permit instrumentation allowing measurements of intracardiac pressures, systemic arterial and venous pressures, and determination of cardiac output in two groups of dogs (n=6), the left anterior descending branch of left coronary artery was ligated. Direct EKG mapping of ST segment changes was then performed at 12 standardized sites. The chest was then closed and both groups of dogs sedated with the same doses of morphine and diazepam using a time base schedule. They were given pancuronium and mechanically ventilated with an air/oxygen mixture with 5 cm of positive end expiratory pressure for the 24 hour period of the study. The experimental group received 120 mg/kg of thiopental intravenously which was given in divided doses by infusion pump during the one hour immediately following coronary vascular occlusion. This dose of thiopental is sufficient to cause an isoelectric EEG as measured by biparietal electrodes. Unpaired T-tests were performed to determine significance ($p < .05$).

Results. Following occlusion of the coronary vasculature, the ST segment elevation for the control and barbiturate groups was respectively 2.02 ± 0.86 mm and 1.58 ± 0.80 mm (±SE). These ST changes were not significantly different ($p > 0.05$). Heart rate and central venous pressure did not differ significantly in the two groups at any time following the infarction. Upon administration of thiopental, cardiac output fell (↓35%) while systemic vascular resistance remained unchanged resulting in a significant lowering of the blood pressure for approximately four hours following barbiturate administration. This drop in cardiac output was paralleled by a rise in left ventricular end diastolic pressure (↑60%). The size of the myocardial infarction, as determined by planimetry of the left ventricle, was 22.7% in the group receiving barbiturates and 20% in the control group, and the infarction size was not statistically different between the groups ($p > 0.05$).

Discussion. Our results indicate that large doses of thiopental have little influence on the ultimate size of acute myocardial infarction, over a 24 hour period, when given to animals following acute coronary occlusion. This occurred despite a reduction in blood pressure which could potentially reduce collateral blood flow in the myocardium. This potential deleterious effect may have been avoided because barbiturates reduce myocardial oxygen demand. Evidence for possible precarious deterioration of myocardial performance can be found by the elevated end diastolic pressure in the thiopental group. This, however, was a transient finding, and our overall result indicates that high doses of barbiturates may not significantly increase the risk of enlarging the area of myocardial infarction under the intensive care conditions simulated in our laboratory.