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IDENTIFICATION OF ISCHEMIA IN THE PRESENCE OF PERSISTENT ST CHANGES ON THE ECG

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INTRODUCTION: Ischemia in an electrocardiogram (ECG) can be identified by comparing the ST segment value with a threshold[1]. Alternatively, change in ischemia can be determined by comparing the difference in feature values between two ECGs with a threshold[2]. The aim of this study was to determine if this difference in methodology can account for much of the large difference in the incidence of ischemia reported.

METHODS: MAC-12^R (Marquette Electronics, Milwaukee, WI) digital electrocardiograph was used to record leads I, II, III, AVR, AVL, AVF, V_4 and V_5 on 115 patients undergoing coronary artery bypass surgery. One ECG was recorded before the start of anesthetic induction and another recorded prior to skin incision.

The first method considers ischemia to be present in an ECG if there is a 1 mm downsloping ST depression or 1.5 mm upsloping ST depression or 1.5 mm ST elevation in a non-Q wave lead or 1 mm ST depression in the presence of early repolarization compared to the other ECG if 2 ECGs are being compared (TOGETHER) or compared to the QRS onset baseline if only one ECG is considered (SEPARATE).

The second method considers ischemia to be present if in a non-Q wave lead, there is a 0.5 mm downsloping ST depression or 0.75 mm upsloping ST depression or 0.75 mm ST elevation or 0.5 mm ST depression in the presence of early repolarization.

RESULTS: The number of patients with ischemia prior to induction only, after induction only and both before and after induction is shown in Table I.

TABLE I

METHOD	2ECGs	PRE	POST	BOTH
I	TOGETHER	3	0	–
	SEPARATE	6	2	7
II	TOGETHER	6	0	–
	SEPARATE	8	2	19

It is evident that when the two ECGs are considered separately, a much higher incidence of ischemia is observed. An analysis of multiple ECGs revealed that the patients detected as having ischemia by SEPARATE method but not TOGETHER method had persistent ST changes and not an acute ischemic episode.

DISCUSSION: Thus the large differences in the incidence of ischemia observed in different studies may be explained primarily on the basis of the difference in methodology of ECG analysis.

The SEPARATE method yields a spuriously high incidence of ischemia in the presence of ST changes caused by reasons other than ischemia and small perturbation in ST values due to noise.

REFERENCES: 1.Slogoff S, et al. Anesthesiology 62:107, 1985. 2.Knight AA et al. Anesthesiology 68:681, 1988

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Title: THE USE OF MYOCARDIAL TISSUE IMPEDANCE TO DETERMINE ESMOLOL'S EFFECTIVENESS TO PROTECT AGAINST ISCHEMIA

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Introduction: Changes in tissue impedance have been shown to correlate with the onset and progression of ischemia in various tissues. Certain drugs may change the onset of ischemic tissue damage because of their effect on myocardial metabolism. Utilizing two standard ventricular pacing electrodes, an impedance monitor, and microprocessor-controlled frequency ranging, the rate at which myocardial tissue becomes ischemic can be observed. The purpose of this investigation is to define possible beneficial effects of esmolol on canine hearts with acutely occluded coronary arteries (LAD) as compared to control canines given placebo.

Method: After institutional approval, 3 separate groups of dogs were anesthetized and fully hemodynamically monitored. Normovolemia was maintained. After median sternotomy, the LAD artery was isolated in a distal position so that a discrete area of the left ventricle would be rendered ischemic upon LAD occlusion. Impedance was measured at 2.5 minute intervals by using AC current at a frequency of 1600 Hz through two standard ventricular pacing leads placed in the ischemic region distal to the location of the LAD ligature. Impedance was then measured for 15 minutes (baseline). Randomly, the dogs were given a bolus of either 500 ug/kg esmolol [Low Dose], 2000 ug/kg esmolol [High Dose], or 5 ml of Ringer's solution [Control]; followed by a maintenance infusion of either 200 ug/kg/min of esmolol, 1000 ug/kg/min of esmolol, or 1 ml/min placebo, respectively. After 10 minutes of infusion, the LAD of each dog was occluded with a ligature (clamped) until there was a 20% increase from baseline impedance. The ligature was then released (unclamped) allowing reperfusion. After the impedance stabilized or after 20 minutes, the study infusion given to the dogs was stopped. Impedance was then measured for an additional 20 minutes.

Results: The characteristic impedance response to LAD occlusion for the low dose esmolol dogs (Fig.1) was similar to the control group. However, the paradoxic response to high dose esmolol was not expected (Fig. 2). The average time required for a 20% increase in impedance for the control dogs was 46 $\pm$ 11 min, for the Low Dose dogs 51 $\pm$ 10 min, and for the High Dose dogs 27 $\pm$ 14 min. This 20% rise was significantly ($P<0.05$) shorter for the High Dose dogs compared to the control and Low Dose dogs. Heart rate did not change more than 5% from baseline for the control or Low Dose esmolol dogs; however, the High Dose esmolol dogs heart rate dropped 12 $\pm$ 4% bpm. There were no other hemodynamic differences between groups.

Conclusion: We found in previous studies that impedance is a sensitive indicator of ischemia in the geographic area affected by LAD occlusion.[1] We would have expected that the well documented beta-blocking effects should have protected this area and the rise in impedance should have been at least as long as the placebo group. No protection against ischemia could be found with the lower esmolol dose; a dramatic and paradoxical decrease in time to 20% impedance rise was found in the High Dose group. Neither the standard clinical dose of esmolol [Low Dose] nor the excessively high dose of esmolol [High Dose] seem to provide myocardial protection from acute ischemia as measured by myocardial impedance. We postulate that high dose beta blockade increases myocardial fiber length and, therefore, myocardial O_2 consumption. It is this increase in myocardial O_2 consumption which results in a steeper rise in the measured impedance.

Reference:
1. Anesthesiology 1990; 73:A482

Fig. 1 LOW DOSE ESMOLOL

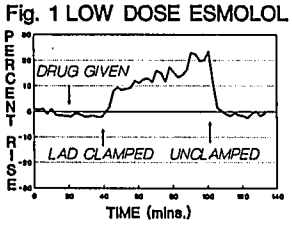

Fig. 2 HIGH DOSE ESMOLOL