Experimental studies concerning mechanisms of post-cardioplegic rhythm troubles

— ABSTRACT —

Keywords: cardioplegia crystalloid, Langendorff, Sabax, microelectrodes, papillary muscle, action potential, mechanogram

Introduction:

Post-cardioplegic arrhythmias are one of the most frequent complications of the heart surgery procedures. The most common post-cardioplegic arrhythmia is atrial fibrillation, with incidences between 20 and 50%. Those incidences are varying with the detection methods used in studies. Our experiments tried do determine the role of different cardioplegic solutions and different cardioplegia protocols in arrhythmia induction.

Material and method:

The present work is based on two different studies:

1. The assessment of arrhythmogenic potential of different cardioplegics using classical glass microelectrodes.

This study was carried on 75 Whistar male rats weighting 273g. ± 16 g., divided in 3 groups. The first group (control) included 28 Whistar male rats. The hearts were excised and the papillary muscles were exposed to a simple cold cardioplegia using Tyrode solution. The second group included 25 rats exposed to "classical" cold cardioplegia protocol using St. Thomas Hospital cardioplegic solution. The third group was composed of 22 rats, exposed to a cold crystalloid cardioplegia protocol using Sabax A+B solution.

In all 3 groups, after 2 hours of cardioplegic arrest, the muscles were reperfused with Tyrode solution. Ten minutes after full cardioplegic recovery, we measured action potentials and mechanograms. We also monitored rhythm and conduction troubles that occurred after cardioplegic arrest.

The action potentials were recorded using standard capillary glass microelectrodes filled with KCl 3M.

The action potentials recording software was developed by us and allows the recording, stocking and analysis of all needed parameters during electrophysiological experiments. The software also allows automatic data transfer into Microsoft Excel for graphical display.

Results:

In the first group (control) we measured a force loss of 8.33 % (p=0.036) after full cardioplegic recovery. The APD_{90} increased with 37.38 msec, which represents 44.17%. In 8 of the papillary muscles we recorded sustained arrhythmias following cardioplegic arrest. The second group had a force of 0.500 ± 0.11 mN before cardioplegia and 0.48 ± 0.07 mN at 10 minutes after recovery. APD_{90} increased with 16.51 msec, representing 19.5%. This group presented 3 cases of post-cardioplegic non-sustained arrhythmias, most of them having an „extrasistolic" pattern. In the third group, the paired t-test showed that the force increased with 0.13 mN after cardioplegic arrest (p=0.002). APD_{90} increased with 39.76 msec, representing 45.5%. In this group we had no arrhythmic activity.

2. Study of the arrhythmogenic risk following cardioplegic arrest in Langendorff isolated rat hearts.

This study included 40 Whistar male rats weighting 280g. ± 20 g., divided in 3 groups.

The first group included 13 rats exposed to cold cardioplegia using Krebs-Henseleit [K-H] solution. The second group was composed of 12 rats exposed to a "classical" cold crystalloid cardioplegia using Sabax A+B solution. The third group included 15 rats, in this group the hearts were exposed to a medium temperature cardioplegic protocol, used in actual "organ care" systems. Electrocardiograms and systolic left ventricular pressures (SLVP) were recorded before and 10 minutes after full cardioplegic recovery. The induction time, recovery temperature and arrhythmic events were monitored.

The ECG amplifier for Langendorff experiments was designed and built by our team using high performance integrated circuits produced by Texas Instruments and Analog Devices companies.

Results:

The induction time in the first group was 82.1 ± 6.7 seconds, 58.5 ± 40 sec. in the second group and 64.2 ± 4.5 second in the third group. The first group presented a 21.7% force loss (p<0.0001). The other 2 groups had no significant loss of force after cardioplegic arrest. Five records of the first group presented ventricular fibrillation (VF), 2 VF were recorded in the cold

Sabax cardioplegia protocol and none in the last group. Sustained ventricular tachycardia (VT) was found in 4 hearts of the control group, 2 in the second and 2 in the third group. Complete atrio-ventricular block (AVB) occurred equally in both groups using Sabax cardioplegia (2 AVB III /group).

Conclusions:

1. At our knowledge, there are no other studies in literature comparing two different cardioplegic protocols using Sabax solution.

2. The ECG recording system designed and constructed by us for Langendorff experiments is a very reliable one, with high performances, offering good quality signals despite large amount of electrical parasites occurring in Langendorff experiments.

3. The Sabax solution reduces the arrhythmic events, when compared with simple cold cardioplegia.

4. The use of oxygenated cardioplegic protocols at medium temperatures offers better myocardial protection versus “classical” cold crystalloid cardioplegia.

5. The arrhythmic incidents frequency was zero in papillary muscles experiments using Sabax solution. Our isolated hearts experiments showed that the arrhythmic incidents frequency was lower in Sabax groups versus “classical” cardioplegia protocols

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