Physiol. Res. 42: 77–79, 1993

Mapping of Activation, Recovery and Activation-Recovery Intervals in the Human Heart

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Summary

Peroperative epicardial mapping of activation, recovery and activation-recovery intervals in the human heart has been performed in a group of 12 patients. These patients had the coronary disease but electrocardiograms with normal characteristics. For this mapping, 240 unipolar electrograms were simultaneously recorded with the system SATAPEC built in our laboratory. The results confirm the classical data obtained on the dog heart. In particular, it was well established that the duration of activation corresponding to activation-recovery intervals is shorter at the base than at the apex of the posterior surface of the heart. With SATAPEC it is very easy to obtain epicardial mapping of electrical activity in a few minutes during open heart surgery.

Key Words

Mapping – Activation – Recovery – Human heart

Introduction

The aim of this work was to map epicardial electrical activity in the human heart during cardiac surgery for several reasons : a) little is known about activation (particularly about recovery and activation-recovery intervals) in human normal or pathological hearts *in situ*, b) previous results were obtained from less than one hundred electrograms which were generally not recorded simultaneously, c) some discrepancies exist between the results reported by Durrer *et al.* (1970) in the isolated human heart, Wyndham *et al.* (1979) in the human heart or by Abildskov (1975) in the dog heart.

Material and Methods

It is obviously impossible to have patients without cardiac diseases. In our study we followed a homogeneous group of 12 patients with coronary disease but without previous myocardial infarction, ventricular hypertrophy or conduction defects. All patients had normal electrocardiograms without ST segment displacement and with a duration of the QRS complex less than or equal to 0.1 s.

240 unipolar electrograms were simultaneously recorded with an epicardial mesh connected to the automatic mapping system SATAPEC built in our laboratory (d'Alché *et al.* 1990). The reference potential was taken at Wilson's terminal.

Activation times, recovery times and activation-recovery intervals were computed on 240 electrograms in the following manner: a) local activation time corresponded to the time of the most negative derivative during the QRS complex, b) local recovery time corresponded to the time of the most positive derivative during the T wave, and c) local activation-recovery intervals corresponded to the difference between recovery and activation times.

After a necessary normalization of times due to the difference in cardiac frequency and after the display of tracings for control and corrections, if necessary, the maps were visualized and statistical analysis was performed by mapping of the means, standard deviations (S.D.) and standard errors of the means (S.E.M).

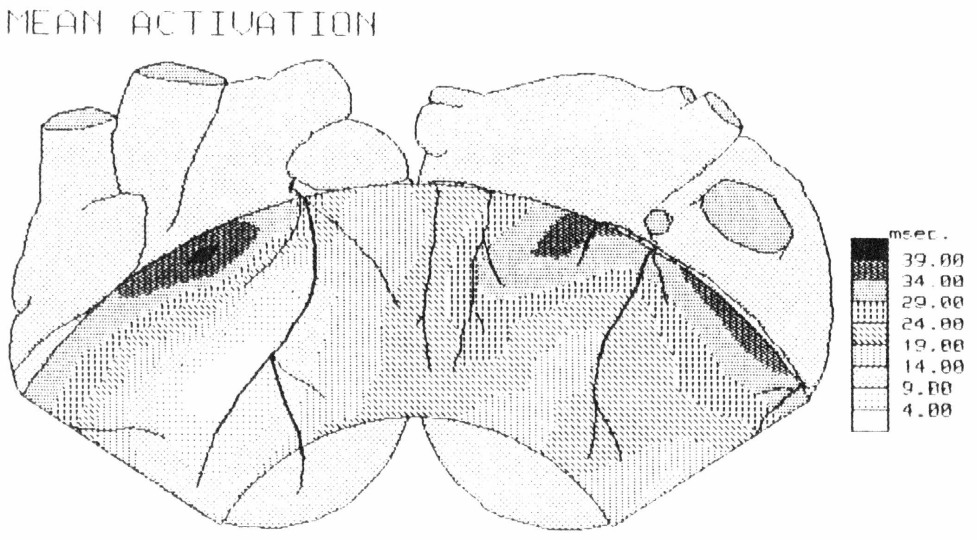

Fig. 1
Map of mean epicardial activation

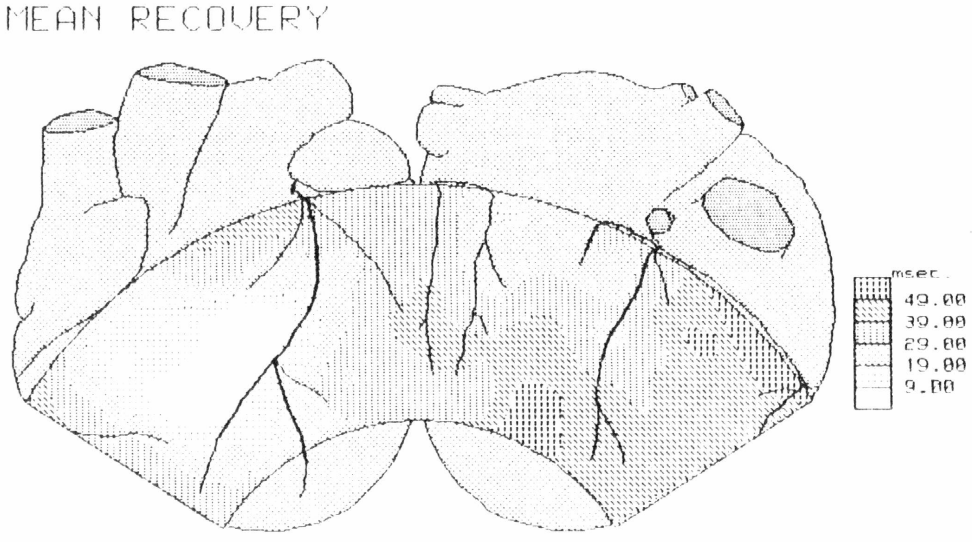

Fig. 2
Map of mean epicardial recovery

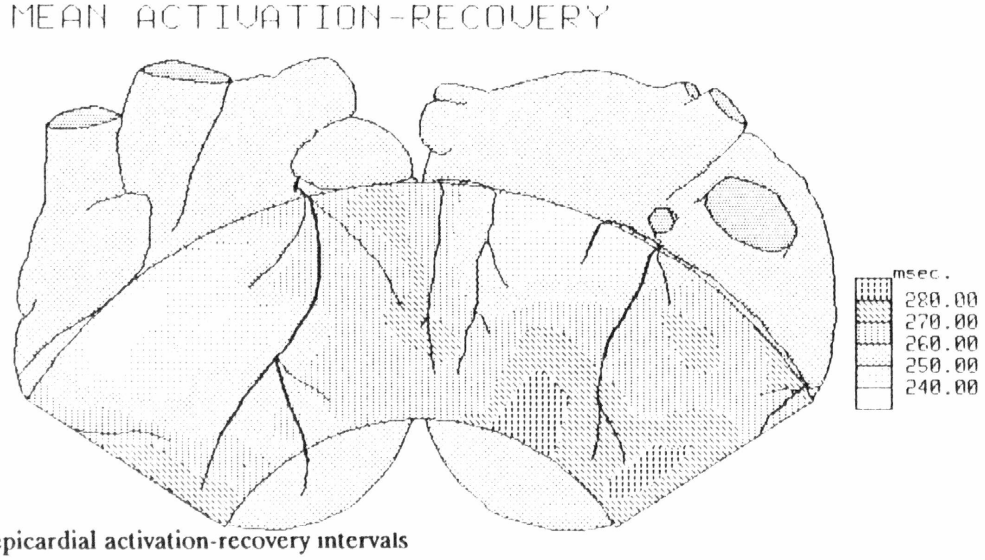

Fig. 3
Map of mean epicardial activation-recovery intervals

Results

Mean epicardial activation (Fig. 1)

Total duration of activation was less than 40 ms. Activation starts near the apex of the right ventricle on the anterior surface with a small dispersion of values (about 5 ms) in this area. The activation ends at the base of the left ventricle, but an abnormal delay was observed at the base of the right ventricle due to an ischaemic area observed in 9 patients, with S.D. equal to 20 ms. Except of the anterior base area, where S.E.M. does not exceed 5 ms.

Mean epicardial recovery (Fig. 2)

Total duration of recovery is about 50 ms. On the anterior cardiac surface, the shape of the recovery map is similar to that of activation. On the posterior cardiac surface, the first area of recovery is located at the base of the left ventricle and the last area of recovery at the left ventricular apex. However, this result observed in the mean map is mainly due to two patients. The other individual maps have a pattern quite similar to the activation one. These important variations of recovery give high values of S.D. (about 30 ms) and S.E.M. (less than 20 ms).

Mean epicardial activation-recovery intervals (Fig. 3)

This map presents the durations of activation at the epicardial surface which occur in the range between 230 and 280 ms (i.e. a difference of 50 ms). The shortest durations were located at the anterior surface and at the base of the posterior surface of the left ventricle. The longest durations were observed at the apex of the posterior surface. The values of S.D. are high (10 to 30 ms for a total duration of 50 ms), indicating a large dispersion of individual maps. The S.E.M. values, shorter than 10 ms at the posterior surface and 20 ms at the anterior surface, were regularly distributed.

Discussion

The values of activation and recovery times are in good agreement with those generally observed under identical conditions.

The propagation of activation from apex to base of the epicardial surface shows good homogeneous results contrary to those obtained by Wyndham *et al.* (1979) on the human heart *in situ* in a group of patients with a similar pathology. For instance, 42 sites of epicardial breakthrough in 11 patients were noted by Wyndham *et al.* (1979), but these authors explored only 54 to 70 epicardial ventricular sites in each heart with a hand-held probe. The delay we have observed anteriorly in the conus region of 9 patients is probably due to the presence of an ischaemic area.

Till now, the sequence of ventricular recovery of excitability stems almost exclusively from the measurements on the dog heart.

The durations of activation are shorter at the base than at the apex. The latter finding corresponds to the results reported by Abildskov (1975) on the dog heart. However, a possible influence of coronary disease cannot be excluded.

The great ease of epicardial mapping during open heart surgery with our system SATAPEC makes it feasible to use as a tool for a best evaluation of the cardiac ischaemic zones, the areas of conduction delay or the location of periinfarctus zones causing ventricular arrhythmias.

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