Myocardial acute and chronic histological modifications induced by cryoablation

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We use cryoablation as the treatment of choice for AF (atrial fibrillation): the technique consists of a linear cryoablation at \(-60\)°C connecting the four pulmonary veins and the posterior mitral leaflet. Incomplete ablation of atrial myocytes may be responsible for AF recurrence should cryoablation be incomplete (i.e. non-transmural) because of associated atrial hypertrophy [1]. In fact should any electrical path be spared, because of incomplete myocyte necrosis, the anomalous circuit could perpetuate in spite of the surgical treatment. For this reason we decided to evaluate in humans the acute and chronic histological modifications and the severity and the extent of the myocyte damage taking place in the atrial wall after cryoablation [2].

In four consecutive patients, operated on for mitral surgery, an atrial biopsy (1 cm²) was taken from the right (two patients) or left (two patients) appendage once the heart was, respectively, canulated or already under cardioplegic arrest during cardiopulmonary-bypass (CPB) at moderate hypothermia. These locations were chosen because they are easily accessible and they would have

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been resected being part of the canulation site or of the auricle ligature. Finally their trabeculations offer less contact to the probe thus making it even more difficult to create an homogeneous lesion.

Each specimen was immediately divided into two portions, one (control) was fixed in 4% buffered formalin while the other underwent cryoablation at −60°C for 2 min before being fixed as above. In a fifth patient an autopsy was performed 1 month after the operation, the patient having died of septicemia. In this particular case specimens could be obtained directly from the area that had been cryoablated during the original surgical procedure. Attention was focused on two areas: the site of cryoablation and the area comprised by cryoablation.

Myocardial biopsies were carefully oriented on tissue paper and 3 μm thick sections were obtained and stained by haematoxylin and eosin (H&E) and Masson’s Trichrome.

All cryoablated specimens were consistently characterized by extensive myocellular damage involving the full thickness of the atrial wall. Myocardial cells displayed shrunk and degenerated cytoplasms; an increased distance between myocytes was documented throughout the wall, due to interstitial edema. Sarcoplasmic vacuolization, increased cell roundness with indistinct membranes and loss or irregular thickness of bands of contraction were major morphologic features of treated case. Nuclei were retained but showed extensive pycnotic changes. No signs of acute or chronic inflammation were documented. (Fig. 1).

In the fifth case, the atrial area which had undergone cryoablation was carefully examined. Viable myocytes were not detected and in the boundaries of the treated area we appreciated a gradual transition from fibrosis to normal atrial myocardium. All these features could be appreciated even at low magnification (Fig. 2).

In conclusion cryotherapy rapidly induced a number of major transparietal degenerative nucleo-cytoplasmic cell changes and the lack of viable myocytes. These morphologic features are the sign of an irreversible injury and can not be reconciled with cell survival and life.

References