INTRODUCTION

Catheter ablation was initially described in 1982 by Gallagher et al. (1). Before 1990, it was performed primarily with high-energy direct-current (DC) shocks using a multipolar electrode catheter positioned in the heart and attached to a standard defibrillator. Under general anesthesia, 100–360 J of direct current energy was delivered between the distal electrode and a patch placed on the patient’s chest. This produced an explosive flash, heat, and increased pressure. Myocardial injury resulted from heat, barotrauma, and direct electrical injury.

More recently, DC energy has been largely replaced with radiofrequency energy as the preferred energy source during catheter ablation procedures. As a result of this change in energy source, the use of catheter ablation increased more than 30-fold between 1989 to 1992, from an estimated 450 to 15,000 procedures per year (2).

RADIOFREQUENCY ENERGY ABLATION

Radiofrequency catheter ablation is performed by delivery of a continuous, unmodulated, sinusoidal, high frequency (500,000 cycles per s) alternating electrical current
between the tip of an electrode catheter and a ground plate positioned on the back or chest. Because the ground plate has a much larger surface area than the tip, current density is focused at the smaller electrode. Current flows from the active electrode into the underlying tissue in alternating directions at high frequency. As a result of ionic agitation in the tissue, resistive heating ensues. Thus, the tissue underlying the ablation electrode, rather than the electrode itself, is the source of heat generation. This contrasts with a thermal probe or soldering iron, in which a resistive element positioned within the probe is the source of heat generation.

The tissue undergoing resistive heating transfers heat to surrounding tissues by conductive heat transfer. Since direct resistive heating falls precipitously with increasing distance from the ablation electrode, it is responsible for heating only a very narrow rim of tissue extending approx 1 mm beyond the ablation electrode \(^3\). The majority of lesion volume is determined by the relative contributions of conductive heat exchange into surrounding tissue and convective heat loss toward the relatively cooler moving blood.

The principal mechanism of tissue destruction during radiofrequency catheter ablation procedures is thermal injury. Elevation of tissue temperature leads to denaturation of proteins and evaporation of fluids, resulting in subsequent tissue destruction and coagulation of tissue and blood \(^4,5\). Temperature-dependent depolarization of myocardial tissue and loss of excitability occurs at temperatures greater than 43°C. Between tissue temperatures of 43°C and 50°C, there is reversible loss of excitability. Once the tissue reaches a temperature greater than 50°C, irreversible tissue injury occurs \(^6\). Electrode-tissue interface temperatures in excess of 100°C cause tissue desiccation and plasma protein denaturation, which result in the formation of coagulum. The development of a coagulum results in a rapid increase in impedance, which leads to a dramatic decrease in current density, thereby limiting further lesion growth. As a result, the lesions created during radiofrequency catheter ablation procedures have well-demarcated borders and are small: 3–6 mm in width, depth, and length (see Fig. 1).