Chapter 9
Research and Clinical Utility of Thermography

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ABSTRACT

This chapter completes the description of the Thermography within this publication. While the previous chapter of this section dealt with principles of data acquisition, this chapter provides a detailed description of the research and clinical utility of thermography. Separate sections are devoted to the ex vivo thermography studies, to the role of thermography in experimental models and finally to the contribution of thermography in clinical studies.

INTRODUCTION

Atherosclerosis is the underlying cause of acute coronary syndromes, the most important entity of cardiovascular diseases (Rosamond, et al., 2008). Despite advances in current imaging modalities the ability to identify patients that are at high risk for an acute coronary event is still limited mostly because these events may occur as the first manifestation of coronary atherosclerosis in previously apparently asymptomatic individuals with non-flow-limiting vulnerable plaques (Honda & Fitzgerald, 2008). Thus, advances in the identification of vulnerable plaques can be an important step in preventing myocardial infarction and sudden cardiac death.

Several histological characteristics of plaques that are prone to rupture and cause an acute coronary event have been identified. These vulnerable plaques possess: (1) a thin fibrous cap (<65 lm); (2) a large lipid pool; and (3) activated macrophages near the fibrous cap (Naghavi, et al., 2003). The
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Figure 1. Marked temperature heterogeneity was observed (in degrees Celsius) over the surface of endarterectomy samples from carotid plaques

EX VIVO THERMOGRAPHY STUDIES

The concept that plaque temperature is a marker of local inflammation was originally proposed by Casscells et al. in 1996 (Figure 1) (Casscells, et al., 1996). This was the first study demonstrating that heat is generated from inflamed atheromatous plaques in humans. In this study, carotid artery samples obtained by endarterectomy were probed with a thermistor (24-gauge needle tip; accuracy 0.1 °C; time contrast 0.15s). Plaques showed several regions in which the surface temperatures varied reproducibly by 0.2–0.3°C. Points with substantially different temperatures could not be distinguished from one another by the naked eye and were sometimes very close to one another (< 1mm apart). Temperature correlated positively with cell density (r = 0.68; p = 0.0001) and inversely with the distance of the cell clusters (mostly macrophages) from the luminal surface (r = –0.38; p= 0.000 (Figure 2) (Casscells, et al., 1996). The thermal heterogeneity observed could also be confirmed using an infrared camera in vivo. In order to assess the possible contribution of infection to generation of heat, the genus-specific monoclonal antibody CF-2 against Chlamydia pneumonia was used (Madjid, Naghavi, Malik, Litovsky, Willerson, & Casscells, 2002). However,