Chapter 8
Thermography: Basic Principles of Data Acquisition

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ABSTRACT
This chapter is devoted to Thermography, and more specifically, to the basic principles and mechanisms of data acquisition. A detailed description of the potential mechanisms of increased heat generation by vulnerable plaques is provided, along with a list of Thermography devices. Additionally, a special subsection of the chapter deals with the limitation of intracoronary thermography, an extremely crucial issue for both the clinical and research utility of Thermography.

INTRODUCTION
During the last decades, the scientific interest in interventional cardiology has been focused on the early detection of vulnerable lesions, which are ultimately responsible for a majority of acute coronary and cerebrovascular events (Hamdan, Assali, Fuchs, Battler, & Kornowski, 2007), (Corti, Hutter, Badimon, & Fuster, 2004). Novel diagnostic techniques have offered promise as methods of detecting vulnerable plaques (Honda & Fitzgerald, 2008), (Tan & Lip, 2008).

It is by now well established that acute coronary syndromes are usually caused by plaques initially associated with <50% diameter luminal narrowing (Corti, Hutter, Badimon, & Fuster, 2004). Moreover, it seems that inflammatory processes play a key role in the initiation, progression, and complications of atherosclerotic disease (Libby, Ridker, & Maseri, 2002), (Ross, 1999). Since atherosclerosis is an inflammatory process, vulnerable plaques characterized by an increased inflammatory infiltrate, generate heat. Based on this concept, intracoronary thermography has been introduced as a catheter-based technique for the functional imaging of atherosclerotic
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plaques, with the ability to identify potential vulnerable and culprit plaques in patients with coronary artery disease. After the first clinical application of intracoronary thermography, several thermography catheters have been designed. Intracoronary thermography is able to detect thermal heterogeneity, which has been shown to be present more often in unstable coronary plaques, and positively correlated to vulnerable plaque morphology characteristics and serum markers of systemic inflammation (Toutouzas, et al., 2007), (Toutouzas, et al., 2007), (Worthley, Farouque, Worthley, Baldi, Chew, & Meredith, 2006), (ten Have, Gijsen, Wentzel, Slager, Serruys, & van der Steen, 2006), (Rzeszutko, et al., 2006), (Madjid, Willerson, & Casscells, 2006), (Toutouzas, et al., 2005), (Toutouzas, Drakopoulou, Stefanadi, Siasos, & Stefanadis, 485-489), (Leborgne, et al., 2005), (Dudek, et al., 2005), (Stefanadis, et al., 2003), (Schmermund, Rodermann, & Erbel, 2003). Heat produced, has also been shown to have a good predictive value for clinical events after percutaneous coronary intervention. However due to several technical shortcomings of current technology, the method is yet to be validated in large prospective trials and thus, the benefit of this method in current clinical practice still needs to be determined. However it still remains a tool that may be used in the future to direct local and/or systemic therapy in patients with coronary artery disease.

POTENTIAL MECHANISMS OF INCREASED HEAT GENERATION BY VULNERABLE PLAQUES

Vulnerable plaques are characterized by several pathologic features including: (1) a thin fibrous cap (<65 lm); (2) a large lipid pool; and (3) activated macrophages near the fibrous cap. a large lipid core, a thin fibrous cap, high neo-vessel formation, and infiltration by inflammatory cells. Several of these features (ie, presence and acti-

vation of inflammatory cells, neo-vessel formation, and fibrous cap thinning) can potentially lead to increased production and dissipation to heat the plaque surface (Naghavi, et al., 2003). This concept is based on the hypothesis that if atherosclerotic lesions are inflamed by virtue of inflammatory cell infiltration, they will give off more heat than normal areas of the arterial system (ten Have, Gijsen, Wentzel, Slager, & van der Steen, Temperature distribution in atherosclerotic coronary arteries: influence of plaque geometry and flow (a numerical study), 2004), (Shah, 2003).

The possible reasons for this increased heat production are: 1) high metabolic rate of macrophages, 2) ineffective thermogenesis (indicated by increased expression of mitochondrial uncoupling protein 2 and 3), 3) increased neoangiogenesis and 4) infections. Macrophages, T-cells, and mast cells are very active cells with a high metabolic rate and high rate of energy consumption (Ten Have, Gijsen, Wentzel, Slager, Serruys, & van der Steen, 2005). Arterial foam cells that have been found in autopsy studies as constituents of the vulnerable plaque consume three times more oxygen than isolated smooth muscle cells. In specific, a study using numeric simulations has suggested that heat-producing macrophages at the shoulder region of vulnerable plaques contribute most to the higher temperature measurements. Moreover, in another study it was demonstrated that the final temperature of the plaque is determined by a balance of heat produced by the macrophages and the cooling effect of blood flow (ten Have, Gijsen, Wentzel, Slager, Serruys, & van der Steen, A numerical study on the influence of vulnerable plaque composition on intravascular thermography measurements, 2006), (ten Have, et al., 2007). The ineffective thermogenesis may be attributed to the lack of oxygen and ischemia on vulnerable plaques that leads to ineffective metabolism of nutrients and greater loss of energy in the form of heat instead of ATP production. More interestingly, macrophages in atherosclerotic plaques show increased expression of mitochon-