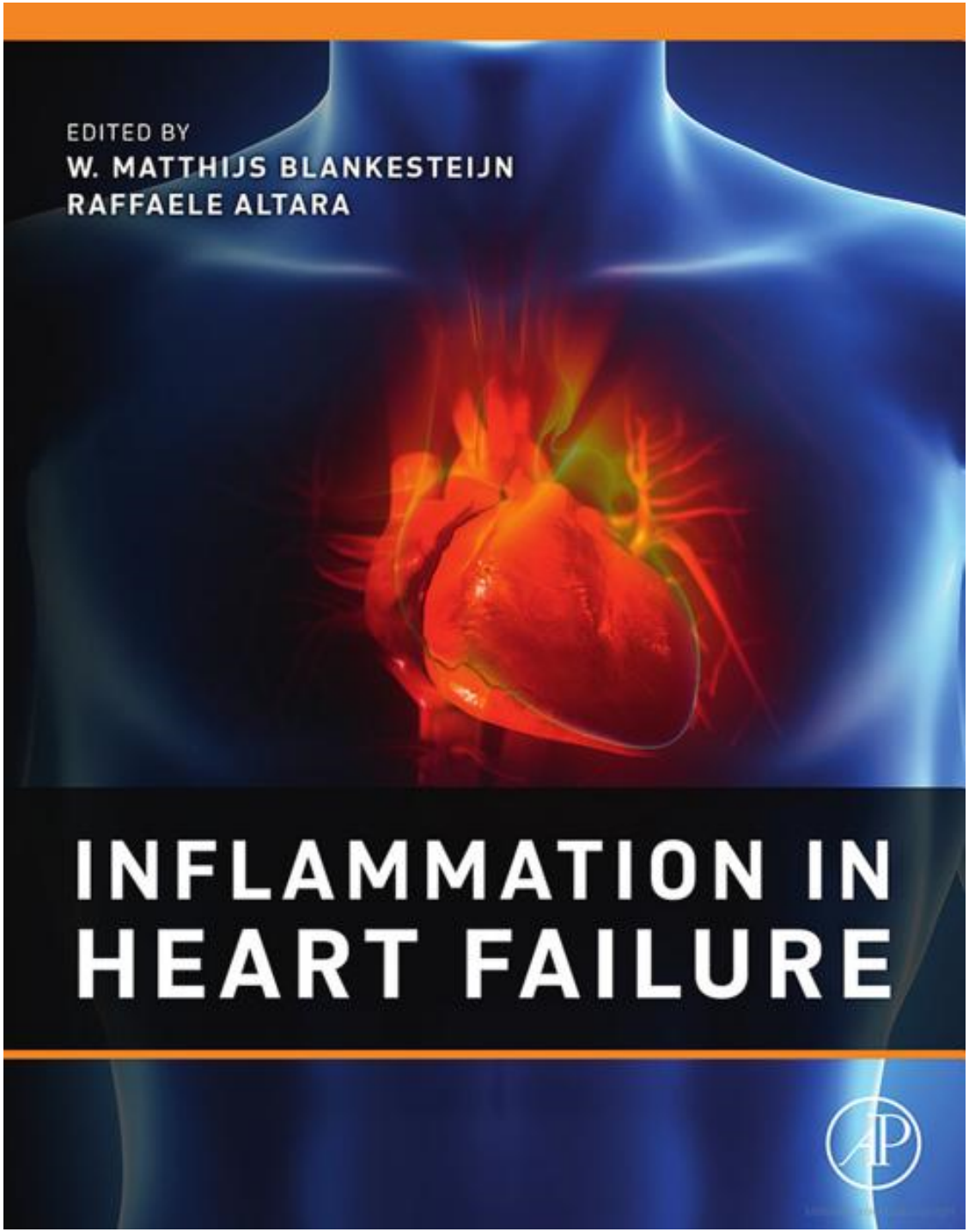
EDITED BY
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INFLAMMATION IN
HEART FAILURE

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525 B Street, Suite 1800, San Diego, CA 92101-4495, USA
225 Wyman Street, Waltham, MA 02451, USA
The Boulevard, Langford Lane, Kidlington, Oxford OX5 1GB, UK

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British Library Cataloguing in Publication Data
A catalogue record for this book is available from the British Library

TABLE 7.1 Association Between Circulating CRP Levels and Post-infarction Remodeling or Development of Heart Failure in Patients with Acute Myocardial Infarction—Cont'd

Study	Number of Patients	Clinical Setting	Timing of Sampling	Method and Timing of Remodeling Assessment	Main Finding
Takahashi *et al.* [26]	31	STEMI	On admission, and 2 weeks and 6 months after AMI	Coronary angiography; on admission, and then 2 weeks and 6 months after infarction	Increased peak serum CRP level was associated with a greater increase in LV volume after anterior AMI
Uehara, *et al.* [27]	139	STEMI	Immediately after 1, 2, 3, and 7 days, and 1 month after the onset of AMI	Echocardiography, 1 month after infarction	CRP is a useful factor for predicting LV remodeling
Xiaozhou *et al.* [28]	106	First infarction	3 days after AMI	Echocardiography on the third day and third month after infarction	NT-proBNP was more effective than hs-CRP as a predictor of dilative remodeling
Berton *et al.* [29]	220	Myocardial infarction	On the first, third, and seventh day after admission	Echocardiogram between the third and the seventh day after admission and 1 year after recruitment	Peak CRP is a strong independent predictor of global and heart failure-related mortality following infarction
Bursi *et al.* [30]	329	STEMI and non-STEMI	On admission	Evaluation of medical records (1.0 ± 0.6 years after the event)	CRP is an independent predictor of heart failure and death
Hartford *et al.* [31]	1618	Acute coronary syndromes	Day 1 ($n = 757$) or day 4 ($n = 533$) after admission	Follow up 75 months	CRP is associated with long-term mortality and heart failure, but not reinfarction

myocardial infarction, both NT-proBNP and hs-CRP measured 3 days after the acute event correlated with increases in left ventricular end-diastolic volume (LVEDV) during the remodeling phase; however, the correlation coefficient was lower for hs-CRP [28]. A growing body of evidence suggests that elevated hs-CRP not only is associated with adverse remodeling, but also predicts the development of heart failure following acute infarction. In patients with acute myocardial infarction, high peak levels of CRP were independently associated with the development and progression of heart failure [29]. In a multimarker approach, baseline troponin, BNP, and CRP measurements provided independent unique prognostic information predicting the development of heart failure [34]. In patients surviving myocardial infarction, there was a strong positive graded association between CRP levels and the risk of developing heart failure; this relation was independent of the size of the infarct and of the occurrence of recurrent ischemic events [30]. In patients with ACS, CRP (and other more specific inflammatory mediators, including interleukin-6 (IL-6), sPLA(2)-IIA and intercellular adhesion molecule (ICAM)-1) assessed on the first day after the acute event, were associated with long-term mortality and development of heart failure, but not with reinfarction [31]. The usefulness of CRP as a biomarker providing relevant pathophysiologic information in patients with myocardial infarction is limited by its nonspecific role in the inflammatory process; use of CRP in this setting may be more informative when accompanied by measurement of other more specific inflammatory mediators.

7.3.1.2 Myeloperoxidase

Myeloperoxidase (MPO), an enzyme with potent oxidant effects that is abundantly produced and released by myeloid cells [35], is expressed in vulnerable plaques and is considered a marker for unstable coronary lesions. Increased serum MPO levels have adverse prognostic implications in healthy individuals, predicting risk of coronary heart disease [36]. In patients with established coronary disease, MPO levels provide important prognostic information. Baseline plasma levels of MPO were associated with the incidence of recurrent ischemic events in patients

Cardiol 2010;106:1410–6.
[24] Mather AN, Fairbairn TA, Artis NJ, Greenwood JP, Plein S. Relationship of cardiac biomarkers and reversible and irreversible myocardial injury following acute myocardial infarction as determined by cardiovascular magnetic resonance. Int J Cardiol 2013;166:458–64.
[25] Orn S, Manhenke C, Ueland T, Damas JK, Mollnes TE, Edvardsen T, et al. C-reactive protein, infarct size, microvascular obstruction, and left-ventricular remodelling following acute myocardial infarction. Eur Heart J 2009;30:1180–6.
[26] Takahashi T, Anzai T, Yoshikawa T, Maekawa Y, Asakura Y, Satoh T, et al. Serum C-reactive protein elevation in left ventricular remodeling after acute myocardial infarction–role of neurohormones and cytokines. Int J Cardiol 2003;88:257–65.
[27] Uehara K, Nomura M, Ozaki Y, Fujinaga H, Ikefuji H, Kimura M, et al. High-sensitivity C-reactive protein and left ventricular remodeling in patients with acute myocardial infarction. Heart Vessels 2003;18:67–74.
[28] Xiaozhou H, Jie Z, Li Z, Liyan C. Predictive value of the serum level of N-terminal pro-brain natriuretic peptide and high-sensitivity C-reactive protein in left ventricular remodeling after acute myocardial infarction. J Clin Lab Anal 2006;20:19–22.
[29] Berton G, Cordiano R, Palmieri R, Pianca S, Pagliara V, Palatini P. C-reactive protein in acute myocardial infarction: association with heart failure. Am Heart J 2003;145:1094–101.
[30] Bursi F, Weston SA, Killian JM, Gabriel SE, Jacobsen SJ, Roger VL. C-reactive protein and heart failure after myocardial infarction in the community. Am J Med 2007;120:616–22.

2. INFLAMMATORY BIOMARKERS