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Heart failure update

A never-ending story

Heart failure in one patient does not equal heart failure in another. This notion is obvious because heart failure is a syndrome with many different underlying causes and is not a disease. However, some pathogenetic principles and mechanisms operate in all forms. This issue of *Herz* examines these and their eventual treatment in the context of the different underlying causes by experts with international reputation in their fields.

Brenner and Ertl examine the current state of remodelling and adverse remodelling after myocardial infarction. These are the adaptive and maladaptive responses to cardiac overload or ischemic injury both at the site of infarction and the non-infarcted area. Wicks and Elliot chose to examine inherited metabolic disorders (IEM) with cardiac manifestations such as cardiomyopathy, arrhythmia and valvular dysfunction. Fatty acid oxidation defects, glycogen, lysosomal and perioxisome storage diseases, mitochondrial cardiomyopathies, organic acidaemias, aminoacidopathies and congenital disorders of glycosylation have all been reported to cause cardiomyopathy. Kloos, Katus and Meder expect that with the next generation of genomic sequencing technologies (NGS) comprehensive genetic dissection of cardiomyopathy patients will reveal clinically relevant information, novel causes and modifiers of this complex disorder.

Ruppert et al. examine differential gene expression profiles which have become valid tools in the study of inflammatory heart disease. Molecular signatures are defined as individual sets of genes, mRNA transcripts, proteins, genetic variations or other variables, which can be markers for a particular phenotype of heart failure and cardiomyopathies. Izumi and Nishii exemplify the important role of interleukin (IL)-10 as a serological biomarker of inflammation in myocarditis. The most dramatic cardiac inflammation is attributed to giant cell myocarditis, for which Cooper and ElAmm suggest that the regulation of inflammatory pathways differs from lymphocytic myocarditis. Kühl and Schultheiss demonstrated a disease-specific differential expression of micro RNAs (miRNAs) in patients with an erythrocyte infection of small vessels by Parvo B19V when compared to other aetiological agents. Maisch and Pankuweit provide an overview on aetiologically based treatment algorhythms in myocarditis and peri- or epimyocarditis. Autoreactive forms are currently treated with immunosuppression and viral forms with i.v. immunoglobulins or interferon beta together with conventional heart failure therapy. Thiene and Basso nicely show that arrhythmogenic right ventricular cardiomyopathy with its histological characteristics of fibrofatty replacement with or with inflammation is not restricted to the right ventricle. Fibrous tissue is the histological hallmark of restrictive cardiomyopathy. Zwas and Keren show that this disease entity should be clinically separated from diastolic failure in"heart failure with normal ejection fraction" on the one hand and from pericardial constriction on the other.

The experts of these topics and many more are part of an international symposium on heart failure and cardiomyopathies in September 2012 in Marburg, which is an activity of the ESC Working Group on myocardial and pericardial diseases, the International Society of Heart Failure and Cardiomyopathies, the German Cardiac Society and the Cardiac Promotion Society Marburg. We are grateful to the sponsors, who make this international event a highlight for inflammation in the never-ending story of heart failure and cardiomyopathies in the hometown of Emil von Behring, who was the first Nobel Prize Winner in Medicine.

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