pericardial effusion: 0 = no effusion, 1 = mild effusion, 2 = massive effusion).

Subsequently, hearts were fixed in formalin (4%) and finally embedded in paraffin. In 2 μm thick, Hematoxylin-Eosin (H.E.) stained slices the severity of myocardial inflammation was determined by the following histological criteria according to our previous research [11]: density of inflammatory infiltration (graded 0-3), strength of inflammatory destruction with rarefaction and pushing-apart of heart muscle fibres (graded 0-3), occurrence of necrosis (graded 0-3), occurrence of pericarditis (graded 0-2). Results were allied into a microscopic-score-system, with a maximum score of 11 points, to express severity of myocarditis.

For comparison of LGE-areas with histologically proven areas of inflammation, infiltrated areas were bordered manually in three representative slices from base to apex in a way similar to ROI drawing in CMR images. Area of inflammation was expressed as percentage of the whole slice. Measurement was performed using a dedicated software (analySIS® 5.0, Soft Imaging System, Münster, Germany). Identical slices distribution of inflammation was encoded using the same segment model as for LGE-distribution.

**Statistical analysis**

All statistical analyses were performed with dedicated software (SPSS® 12.0 for Windows, SPSS Inc., Chicago, Illinois, USA).

Percentages of LGE and inflammatory areas relative to healthy myocardial tissue are expressed as means ± standard deviation (SD). Due to non-symmetric distribution of serological parameters (troponin, haptoglobin, proBNP) and ordinal scale of macroscopic and histopathological scores we had to use non-parametric statistics on these data. Thus, all correlations were calculated by bivariate correlation analysis using Spearman’s correlation coefficient; significant differences of serological parameters between experimental and control group were evaluated by the Mann-Whitney test. Averages; averages for macroscopic and histopathological scores are expressed as median.

In each CMR-sequence with associated slices, four-field-tables, expressing sensitivity, specificity, positive and negative predictive values for LGE, were used to display accuracy of CMR. Histopathological analysis was defined as gold standard.

Results were considered to be significant at p < 0.05.

**Results**

All animals of the control group were histopathologically healthy and none revealed LGE by CMR-examination, in contrast all animals of the experimental group (n = 10) had histologically proven myocarditis.

**Comparing topographic distributions of LGE and inflammation**

Histologically proven inflammation was predominantly located in the anterior and lateral wall of right and left ventricle (see Figure 1); LGE was found mainly in the anterior and lateral wall of the left ventricle, in no case inside the right ventricular wall (see Figures 2 and 3).