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The emerging role of inflammation in atrial fibrillation and the potential of anti-inflammatory interventions: reply

The letter by Dr Korantzopoulos and coworkers draw attention to some new studies which were not published when the review ‘Inflammation in the genesis and perpetuation of atrial fibrillation’1 was written. Dr Korantzopoulos mentions four studies on markers of inflammation and AF, studies which are interesting and adds to the body of evidence in favour of an association between AF and inflammation, although it should be emphasized that the observed associations cannot be interpreted as indicating a role for C-reactive protein in the pathogenesis of AF. It is possible that elevated C-reactive protein levels are simply indicative of a generalized inflammatory state antedating AF. Moreover, the majority of studies are limited by the use of C-reactive protein as the only marker of inflammation. C-reactive protein is a non-specific acute-phase protein primarily produced in the liver in response to most forms of tissue damage, infection, inflammation, and malignant neoplasia. Studies in patients with acute myocardial infarction have indicated that cytokines, i.e. interleukin-6 (IL-6) is produced locally in the heart, whereas C-reactive protein is produced mainly in the liver and taken up locally by phagocytosing white blood cells.2 Thus, measurement of cytokines (IL-6, TNF-α, etc.) may be more specific than C-reactive protein and may hold important information in AF patients. This is underlined in the study by Psychari et al.,3 who measured C-reactive protein and IL-6. Both markers of inflammation were significantly elevated in AF patients and both were independent predictors of left atrial size. In addition, IL-6 levels were positively related to AF duration which may indicate a role for inflammation in the process of atrial remodelling. Interestingly, IL-6 levels were not independently related to the presence of AF, which is in contradic-

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References


Six minute walk test

Dr Refsgaard and Dr Hager have commented on our article regarding 'Six minute walk test' (6MWT).

We concluded that '6MWT' is a simple and inexpensive test, which is not robust enough to evaluate treatment effects in clinical trials. However, it may have a role in clinical practice as a routine part of evaluation (as many patients avoid symptoms by reducing their activity).

A recently published trial on 1077 elderly heart failure patients shows that change in symptoms corresponds with change in 6MWT walking distance. The corridor walk test is limited by some very practical considerations such as the length of a quiet corridor where the patient's performance will not be affected by the staff. We agree that encouragement should be administered, not only for improving performance on 6MWT, but also to comfort the patient who might find it awkward to walk in silence for 6 min. Standardization of encouragement may be of value. The value of repeated baseline walk tests is disputed. In the statement issued by the American Thoracic Society (ATS) regarding execution of the 6MWT change in walk length from first to second walk varied from 0 to 17% in a variety of diseases including chronic heart failure, and the authors concluded repeated baseline tests to be unnecessary in most settings. Indeed, 6MWT has proven to yield equally stable results regardless of repeated walk tests.

Ultimately, the 6MWT can be of use in the clinical setting and in evaluating treatment in selected heart failure patients, but in order to ascertain comparable and reliable results there is a need for further standardization. We agree with Dr Hager that the statement issued by ATS can provide the framework for this.

Regarding peak oxygen uptake as the 'golden standard' of heart failure assessment we must respectfully disagree with the author. Peak oxygen uptake has in our opinion, with the exception of patients eligible for heart transplant, not outperformed 6MWT, neither as a prognostic indicator nor in the evaluation of treatment and is more expensive to use and more cumbersome for the patient.

Conflict of interest: none declared.

References

1. Refsgaard J. 'This is a walking test, not a talking test': the six minute walking test in congestive heart failure. Eur Heart J 2005;26:749–750.

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