

Author's response

We gratefully receive the commendations of Kardesoglu et al. [1] in the Letter to the Editor entitled 'Accurate assessment of autonomic imbalance in heart failure'. The authors highlight the value of a simple and reliable test for the prognosis and treatment planning of heart failure (HF) patients.

Several studies have dealt with the evaluation of autonomic nervous system (ANS) in HF in an attempt to assess and measure sympathetic nervous system (SNS) activity. The cardiac sympathetic nerves are preferentially stimulated in severe HF with plasma norepinephrine (Nor) release to increase 50-fold in untreated patients [2]. In 1997, Esler et al. [3] published a study in which measurement of plasma Nor as a research tool was replaced by sympathetic nerve recording (clinical microneurography). Radiotracer methods measured regional sympathetic activity in the heart, providing information on regional sympathetic function that was previously lacking.

In the same year, Kurata et al. [4] used [123I] metaiodobenzylguanidine (MIBG) imaging to assess cardiac sympathetic nerve abnormalities. They found that increased cardiac SNS activity may be associated with increased myocardial MIBG clearance and decreased heart rate variability (HRV), including low-frequency power.

Another study used quantitative iodine-123 metaiodobenzylguanidine (MIBG) myocardial imaging to examine cardiac sympathetic nerve activity in 33 children with chronic HF. This method is clinically useful as a predictor of therapeutic outcome and mortality in children with chronic HF [5].

However, in everyday practice, it is much easier to assess autonomic modulation with HRV [6]. Measurement of HRV is a non-invasive way of obtaining reliable and reproducible information on autonomic modulation of heart rate, although there is a difficulty in using HRV as a quantitative estimate of autonomic dysfunction in HF. Consequently, Adamson et al. [7] used an implantable device to measure long-term continuously. In another article, the authors reviewed neural control of heart rate, briefly described HRV and summarized research data which conclusively demonstrated that HF is associated with altered heart HRV [8].

In our study, pupillometric measurements were successfully used to assess the ANS activity in patients with HF. In order to replicate our previous results, we used a larger sample size and our findings were confirmed (Keivanidou Anastasia, 'Evaluation of autonomous nervous system by pupillometry in healthy subjects and heart failure patients' PhD thesis, 2009, Aristotle University of Thessaloniki, Greece). This study is currently under review. However, the relation between these changes in different stages of HF should be confirmed. Furthermore, the application of this test for its use on patients with HF before and after treatment could give us valuable data concerning potential therapeutic options. Finally, the relation between our findings and autonomic imbalance should be confirmed with a more objective test. To conclude, pupillometry does not set the diagnosis, but it can however be used as an additional marker of the ANS activity in HF patients.

We would like to thank Kardesoglu et al. [1] for encouraging further studies to confirm the potential use of pupillometry in the prognosis and treatment planning of HF.

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