Heart failure in elderly patients with chronic obstructive pulmonary disease: reply

We thank the authors for their comments and agree that obstructive sleep apnoea (OSA) could be a risk factor for heart failure in our chronic obstructive pulmonary disease population, just like other known risk factors for heart failure, such as hypertension, diabetes, and atrial fibrillation, which were present in a substantial proportion of our chronic obstructive pulmonary disease patients.

As we mentioned in our discussion paragraph, the outcome panel assessed whether patients had heart failure or chronic obstructive pulmonary disease according to the ESC and GOLD criteria, respectively. These criteria are not influenced by the presence of OSA. We therefore disagree with the authors that (possible) concomitant presence of OSA in some of our chronic obstructive pulmonary disease patients could have influenced our prevalence estimates of heart failure. Presence or absence of OSA did not influence the outcome panel’s diagnosis of chronic obstructive pulmonary disease or heart failure. The introduction of the term ‘pure’ chronic obstructive pulmonary disease is therefore incorrect and even somewhat misleading.

References

8. Arno W. Hoes. Julius Center for Health Sciences and Primary Care University Medical Center Utrecht Utrecht The Netherlands Tel: +31 30 2538193 Fax: +31 30 2539028 E-mail address: f.h.rutten@umcutrecht.uu.nl
In an editorial to that article, Refsgaard reported. Especially, practice runs prior to exact protocols of the 6MWT were not possible. They also found that in many studies the subtle factors influencing the results. The "golden standard" of heart failure assessment (at least for survival)5,6 and indication for transplantation6,7 remains the maximal cardiopulmonary exercise test (CPET) with measurement of peak VO2, even though it does not reflect daily activity. Also there is a lack of data on patients being treated with β-blockers and/or ICD's, that have proven to prolong survival but do not increase or even decrease VO2max, and a CPET is not as easily performed as a 6MWT.

References
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Alfred Hager
Department of Pediatric Cardiology and Congenital Heart Disease
Deutsches Herzzentrum München


Miguel A. Arias
Servicio de Cardiología
Complejo Hospitalario de Jaén
Pza del Zodiaco No. 8, 5B
23009 Jaén
Spain
Tel: +34 637 463857
Fax: +34 953 270692
E-mail address: maapalomares@secardiologia.es

Alberto Alonso-Fernández
Servicio de Neumología
Hospital Universitario La Paz
Madrid
Spain

Francisco García-Rio
Servicio de Neumología
Hospital Universitario La Paz
Madrid
Spain

Carlos Pagola
Servicio de Cardiología
Complejo Hospitalario de Jaén
Jaén
Spain

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Comment on six-minute walk test as an outcome measure for the assessment of treatment in intervention trials of chronic heart failure

Olsson et al.1 outlined in their article that the 6-minute walk test (6MWT) has not yet been proven to be a robust test for identification of the effectiveness of pharmacological treatment because of many even subtle factors influencing the results. They also found that in many studies the exact protocols of the 6MWT were not reported. Especially, practice runs prior to baseline measurements and standardization of patient motivation are urgently needed. In an editorial to that article, Refsgaard2 even suggested that no encouragement at all should be given to the patients during the test. However, as mentioned in the conclusions, ‘the test may be of greater value with more advanced heart failure, where it may function as a maximal exercise test’.

This can hardly be obtained without encouragement. The American Thoracic Society published guidelines3 on the 6MWT, where many details such as the length of the corridor, or even the exact wording of the encouragement and its timing were fixed. If all centers abide by those guidelines, or at least report where they did not, results will be much more comparable.

The ‘golden standard’ of heart failure assessment (at least for survival)4,5 and indication for transplantation6 remains the maximal cardiopulmonary exercise test (CPET) with measurement of peak VO2, even though it does not reflect daily activity. Also there is a lack of data on patients being treated with β-blockers and/or ICD’s, that have proven to prolong survival but do not increase or even decrease VO2max, and a CPET is not as easily performed as a 6MWT.

Thoroughly analysing directions of the still valid Guidelines on diagnosis and management of acute pulmonary embolism (PE) published by European Heart Journal in 2000 throughout the management of our patients with PE caused by heparin-induced thrombocytopenia Type II (HIT II),1 we have noticed that its section ‘Epidemiology and predisposing factors’ completely fails to give any attention to HIT II as the possible risk factor for venous thrombo-embolism, whereas this issue received little attention under the section ‘Treatment of PE’. Apart from the introduction of immediate active non-heparin anticoagulants upon heparin therapy discontinuation, the latter section also advises the use of r-hirudin derivatives in patients with HIT associated with a new thrombotic episode or aggravated PE. However, the same applies to the danaparoid sodium administration which is suggested in low doses of no more than 2 × 750 IU subcutaneously, today considered as preventive doses, while the intravenous route remains vague as to both the specific group of patients to receive it and its dosage.1

We have adopted a more recent attitude that a therapeutic danaparoid regimen initiated with a loading dose of 2250 units followed by the stated maintenance dose of 400 units/h and later 300 units/h over the first 7h further continued with 200 units/h is considerably safer than low preventive doses in HIT with thromboses.2–4 The given full therapeutic doses are more than 4.5 times higher during the first therapeutic day and about three times higher than the preventive ones a day after the drug initiation.3

Our attitude that the preventive doses, nowadays held to be underdosed even in cases of HIT without thrombosis, so-called isolated HIT, may lead to the prolongation of natural course of disease is further...