Author's response to reviews

Title: Tinnitus in elderly patients and prognosis of mild-to-moderate congestive heart failure: a cross-sectional study with a long-term extension of the clinical follow-up

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Author's response to reviews: see over
Answers to reviewer 2

Reviewer: Berthold Langguth  
Reviewer's report:

Dear Sir, we thank you again for your extremely important contribution in the review of our manuscript. Going to the specific issues you raise:

1. The authors interpret the observed correlation between tinnitus and severity of congestive heart failure in the sense of a causal relationship. However this conclusion is not supported by the data.

   A: We agree with your comment that our point of view was too much “cardiolologically” oriented. Accordingly we have attenuated the concept, concentrating our conclusions on an association in spite of a casual relationship.

2. The interpretation of a tinnitus as a consequence of disturbed microcirculation of the inner ear stands in contrast to the current pathophysiological knowledge about tinnitus. The authors ignore in their introduction all recent knowledge about the mechanisms of tinnitus generation.

   A: Some other hypotheses have been now added to the discussion. We do not intend to ignore the other causes of tinnitus that are very well (and probably even better!) supported by the literature. However since a final and unique explanation is not actually available we support that the possible involvement of an insufficient perfusion at the microcirculation level might be a plausible trigger for tinnitus onset or exacerbation in patients with impaired peripheral perfusion as those affected by CHF. A statement about this particular issue has been added to the discussion.

3. The authors do not provide an explanation how the proposed mechanism (microcircular dysfunction of the inner ear) may generate tinnitus without having an influence on auditory thresholds. These issues have to be addressed both in the introduction and the discussion section, before publication of the manuscript can be recommended.

   In our population of patients the simpler explanation might be the extreme variability in the entity of the hemodynamic derangement that affect the patients with heart failure and that could promote the onset of tinnitus on an “ischemic” and reversible basis without any major evidence of a concomitant and persisting hearing impairment. Actually, tinnitus is a spontaneous symptoms that is immediately perceived without any external stimulation whereas the perception of the hearing impairment needs an external acoustic trigger. This could have increased the sensitivity of our patients toward tinnitus beyond the complaint of any hearing loss. We could also speculate the possibility that two separate mechanisms of action might contribute to the same symptom, namely one functional (hemodynamic derangement) and the other more structural (hearing disturbance). A couple of sentences about this issue has been added to the discussion.