Response to Reviewer 2 Comments

**Point 1:** Their interpretation of VHF are still not correct. I agree that the physiological meaning of VHF is controversial, but I do not agree with their statement that "consensus emerge that VHF is a robust sign of ANS dysfunction". The papers I have recommended to them (see, e.g. [Front Physiol 8:255, 2017], [J Cardiovasc Electrophysiol, Vol. 16, pp. 954-959, September 2005]) point clearly to the interpretation of VHF as non-autonomic influences. Those very-high frequency oscillations are usually referred to as erratic or fragmented patterns. This must be taken into account in their discussion to keep readers up to date and not to falling into sterile discussions.

**Response 1:** It is so kind of you to point out the correct physiological interpretation of VHF for us! The papers you recommended are cited by us as reference [6] and [17] now. After taking the fragmented patterns of hearts into consideration, we are able to explore the underlying mechanism of CHF more convincingly. Since ANS dysfunction is not the only reason for CHF, we estimate both ANS and non-autonomic function by analysing HRV. VHF, usually referred to as erratic or fragmented patterns, could reflect the frequent changes in heart acceleration. HF, LF and VHF could serve as robust markers of ANS function. The significant difference of TE (*→1) reflect a weakened coupling zone between ANS and non-autonomic influences. It can be inferred that the control of ANS over non-autonomic components is weakened after perturbation, resulting in an increase in the overall amount of short-term variability. This conclusion corresponds with the results in reference [6, 17]. Meanwhile, TE (3→2) and TE (3→4) represent a ANS dysfunction in CHF group, consistent with reference [26, 53]

**Point 2:** The explanation to point 3 given in their last response is nice and should be added to the paper.

**Response 2:** We added the explanation in Line 283-286 to make the concepts clear for readers. Thanks for your reminding!