This study was designed to investigate if electric vagal nerve stimulation plays a role on inflammation and postoperation cognitive during aged rats.

The authors found that serum inflammatory cytokines of TNF-\(\alpha\) and IL-6 increased by the operation/anesthesia, but these cytokines could be decreased by the vagal nerve stimulation. The level of TNF-\(\alpha\) protein and mRNA expression of NF-\(\kappa\)B in hippocampus were also eliminated by the vagal nerve stimulation compared to S group. The Morris Water Maze results showed the escape latency of postoperation in the S group was significantly longer than the C group (\(P<0.05\)), and the times of crossing platform in the S group was lower than that of the C group (\(P<0.05\)).

The authors used in vivo studies to evaluate cytokines expression both in serum and hippocampus, cognitive function. Various techniques such as western blot and PCR were conducted to study cytokines expression levels. These methods significantly strengthened this paper. This is a well-written manuscript that examines the effects of electric vagal nerve stimulation on inflammation and postoperation cognitive during splenectomy. However, there are some concerns on the methods and data interpretation, need at least to be well discussed.

The methods on the electric vagal nerve stimulation are sparse. These really need to be substantially extended for the data presented to be appropriately interpreted by the reader.
Please provide justification on the why 1-7 days were selected as the observed time point? Since cognitive function impairment is a long-term process. Why the authors think 1-7 days after splenectomy could cause cognitive function damage?

The authors stated that "The NF-κB expression of the S group was increased significantly compared to the C and SV groups. There was similar expression between the C and SV groups. That showed VNS might reduce the pro-inflammation gene transcription." NF-κB, as a key transcriptional factor, plays a critical role in modulating inflammatory responses via regulating the expression of pro-inflammatory mediators. When inactive, NF-κB resides in the cytoplasm and forms a multiprotein complex with an inhibitory subunit, inhibitor of NF-κB (IκB). When activated by external stimuli, IκB phosphorylation, and degradation, the liberated NF-κB then enters the nucleus and activates transcription of multiple inflammatory response genes by interacting with κB elements in the promoter region. Thus, increased NF-κB activation is considered as an important pathogenic factor in many inflammatory disorders. In this study, the authors only tested total NF-κB expression, and this results can't gain the conclusion that "VNS might reduce the pro-inflammation gene transcription".

The pictures are not clearly and beautiful enough for publication.

**Are the methods appropriate and well described?**
If not, please specify what is required in your comments to the authors.

No

**Does the work include the necessary controls?**
If not, please specify which controls are required in your comments to the authors.

Yes

**Are the conclusions drawn adequately supported by the data shown?**
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No

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