The main concerns of the reviewer were from the lack of clarity in the methods that concern the reconstruction of neurons. Secondly, the reviewer also wondered about the ability to classify the synapses in this dataset based on techniques that are used for mammalian tissue. Some of the questions posed by the reviewers could not be assessed since this involved large amount of reconstructions. In short we have addressed the reviewers concerns and have made efforts to clarify the text to make the reconstruction methods lucid. We have also carried out additional analyses to attempt to classify the synapses based on conventions used for mammalian tissue.

1. *Given the numerous postsynaptic input sites that were identified on the different morphological types, I was anticipating the authors would then proceed to reconstruct the presynaptic neurites. They did not. This was disappointing because I think a lot could be learned from such an effort. Such as: identification of additional putative integrator neurons that were not revealed by calcium imaging, identification of descending axons that presumable carry velocity information, an analysis of what fraction of inputs originate from different input types, etc.. Again, I understand the limitations of the volume size, but some additional effort to gain more insight about the actual circuit connectivity within the integrator circuit would significantly strengthen the manuscript.*

   (a) As we had indicated previously in our plan of action, the reviewer requested for anatomical reconstruction of all cell that were both Pre and Postsynaptic to the 22 integrator cells that were reconstructed in this study. This is approximately 3000 cells that would need to be reconstructed. This represents a 100X expansion of the current study. This is therefore outside the scope of this publication. Moreover integrator neurons cannot be identified by anatomy alone, and all physiologically identified integrator neurons in this study have been reconstructed.

2. *Following on this point, the identification of just 6 synapses between the 22 cells is a very low number. It begs for the not analyzed presynaptic cells to be traced and analyzed to the extent possible. Is there a theoretical prediction of the degree of connectivity needed to support positive feedback in the integrator circuit?*

   (a) Like the above question, this question could only be answered by the detailed reconstruction of all cells that are synaptically connected to the 22 identified cells. As was discussed in the plan of action, this is also out of the scope of this publication.

3. *The authors found no substantial correlation between time constants and the variables they examined. Did they correlate time constants with synapse numbers/densities for each cell? Again, some idea of the fraction of synapses driven by descending inputs versus intra-hindbrain inputs would be interesting.*

   (a) The reviewer has requested for the correlation of time constants to synapse numbers and densities. We have calculated and reported these correlation in the supplementary figures (Sup. Fig. 1B) along with the other variables that were previously reported. Both these analyses did not reveal a statistically significant difference in the . This is likely due to the small sample size of this study.

   (b) The second part of this question requests to compute the fraction of inputs onto the integrator neurons form other neurons within the hindbrain to outside the hindbrain. We agree with the reviewer that the results would be very interesting. However, to do this we would have to reconstruct all the neurons that are presynaptic to the integrator neurons. As we have mentioned previously, this is out of the scope of this publication.

4. *The description of the tracing methodology is not sufficient to assess reliability. At a minimum the authors should expand on what the second round of tracing actually entailed and what exactly 'multiple coverage' means.*

   (a) In the first round of tracing, we used the software package TrakEM2 to create skeleton representations of neurons. Most tracings were made by the first author (A.V.), and difficult locations were decided by consensus of E.A. and A.V.

The second round created 3D reconstructions of neurons, using semiautomated methods and help from expert tracers who were not authors. Each cell was reconstructed by one tracer, reviewed and corrected by another tracer, and reviewed and corrected by the first author (A.V.). This is referred to as 'multiple coverage', the same cell is covered by more than one tracer. This process did catch two significant errors that slipped by the first round of tracing (one underreconstructed axon and one overreconstructed dendrite). Other errors were minor in terms of the neurite length involved. We have now revised the text in the methods to make this process more clear (lines 569).

(b) Subjectively, we were more confident after the second round of tracing because the real-time feedback from 3D reconstructions enables shape-based recognition of suspicious locations, which can then be checked for errors. For example, one can easily spot a sudden change in neurite thickness, which suggests a merge error. This is easy with 3D reconstructions but not with skeletons.

5. *Did the authors find 45 nm section thickness to be sufficient to unambiguously trace all neurites? If so, they should explicitly state this because it would serve as a useful data point for the field. If not, the authors should quantify the prevalence of tracing ambiguities.*

   (a) Indeed the reconstruction can be difficult if the neurite is parallel to the sectioning plane, and especially thin. Such locations are usually disambiguated by dense reconstruction of all adjacent neurites, which reveals the correct answer by a process of elimination. (This technique was used to correct the two significant errors uncovered by the second round of tracing.) Tracing intracellular organelles can also aid disambiguation of neurite reconstruction. We have highlighted such techniques in the revised methods section (line 580). After such techniques are applied, the end result is that every cell has at most one or two truly ambiguous locations. The ambiguity might be caused by a thin neurite parallel to the sectioning plane, or deteriorated ultrastructure (due to electroporation, poor fixation, or possibly developmental pruning of axons). In truly ambiguous locations, we err on the side of undertracing.

6. *Is there evidence that excitatory versus inhibitory synapses are ultrastructurally distinct in fish as is the claim in mammalian tissues (i.e. symmetric vs. asymmetric synapses)? Given the inference of transmitter type based on soma position, the authors could presumably at least look for differences that could help corroborate transmitter types.*

   (a) For an asymmetric synapse, the postsynaptic density (PSD) should be darker and extend farther into the postsynaptic cell. By eye, we were unable to distinguish between asymmetric and symmetric synapses. For a more objective method, we quantified image intensity near synaptic clefts. There was no evidence of a bimodal distribution in the darkness/thickness of the synaptic density. Furthermore the synaptic density did not extend asymmetrically into the postsynaptic cell. In fact, we saw evidence of a presynaptic density (Supplementary Fig. 2E), suggesting that the mammalian notion of an asymmetric synapse is not applicable here.

   (b) For a symmetric synapse, the vesicles should be more flat. We found a few flat vesicles, but no synapses at which many vesicles were flat. We did see synapses at which there were many large dense core vesicles, as reported in the main text.

   (c) We have reported these finding in the main text (line 114) and supporting analysis in supplementary figure 2E.