Below are the detailed answers to the reviewers comments. 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) We apologize if our description of the tracing methodology was not sufficient. Our tracing was performed in two rounds. The first round was a skeleton based tracing that was performed using the open sourced software package TrakEM2. This process requires the placement of a point (or node) typically at the center of the neurite and to continue this process for the entire cell. This results in a 'ball-and-stick' type model of the neuron. The problem with this arrangement when viewed in 3D is that it is hard to spot false positives. This is because reducing the neurites thickness to a single point removes any sense of scale. This contextual information is very useful to 'catch' for example branches whose thickness do not match the thickness of the brach being traced indicating that it does not belong.

To overcome some of the limitations of skeleton based tracing, we performed a second round of tracing where the cells were traced volumetrically. This method displays in real-time the branches being traced. This process did catch a few errors that slipped by the previous round of tracing (as mentioned in the methods). In order to have confidence in the traces, we required multiple tracers (>2) reconstructing the same cell, and final assessment by an expert tracers. This process is what we refer to as 'multiple coverage'. We have now revised the text in the methods to make this process more clear (lines 569).

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) We thank the reviewer for bringing this point to our attention. Indeed there are some challenges associated with reconstruction of neurons from sections that are cut at 45nm. The most common problem occurs when the neurite being reconstructed is parallel to the sectioning plane. This makes it hard to follow these neurites. At such locations, typically it helps to reconstruct all abutting neurites and converge on the parent neurite by the process of elimination. Another strategy is to keep track of the intracellular organelles to guide in the reconstruction. We have highlighted some these challenges in the methods section (line 580). As was mentioned in the methods, of the 22 neurons, 2 neurons had problems in the axon reconstruction due to this anisotropy. These locations were subsequently recovered by having multiple tracers reconstruct at these locations using the above mentioned methods.

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) As the reviewer had mentioned it is possible to differentiate synaptic membranes as either symmetric or asymmetric, depending on the side where the densities were more prevalent. Along with this observation, to aide this classification the shape of the vesicles are also considered. Typically asymmetric synapses are associated with spherical vesicles and are thought to be from excitatory cells. Whereas symmetric synapses have 'flat' shaped vesicles and are associated with inhibitory cells. Similarly symmetric synapses have thin PSDs as compared to asymmetric synapses. In our dataset, it is very hard to observe any 'flat', non-spherical vesicles. There are however large and small vesicles as has been noted in the main text. Regarding the postsynaptic density if there exits differences we should expect to see a large variation in the thickness of the PSD and a bi-modal like distribution of the thicknesses. Instead we find a normal distribution of PSD thicknesses and no real difference in the shape of the vesicles. We have reported these finding in the main text (line 114) and supporting analysis in supplementary figure 2E.