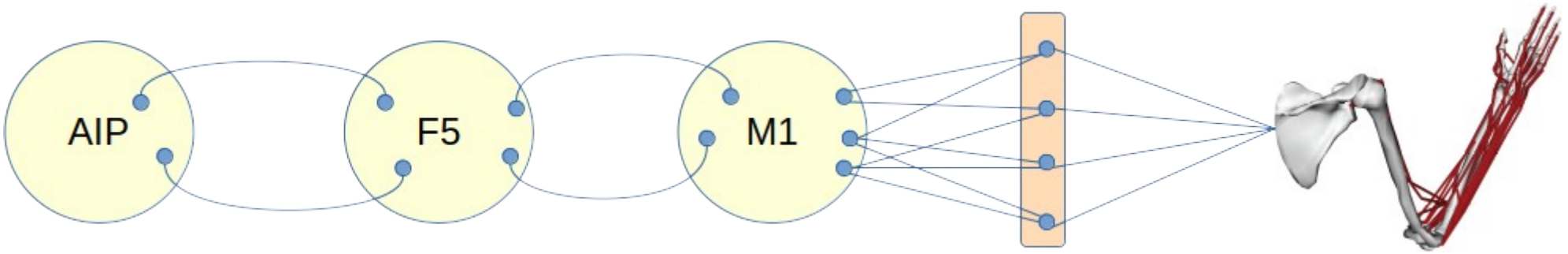
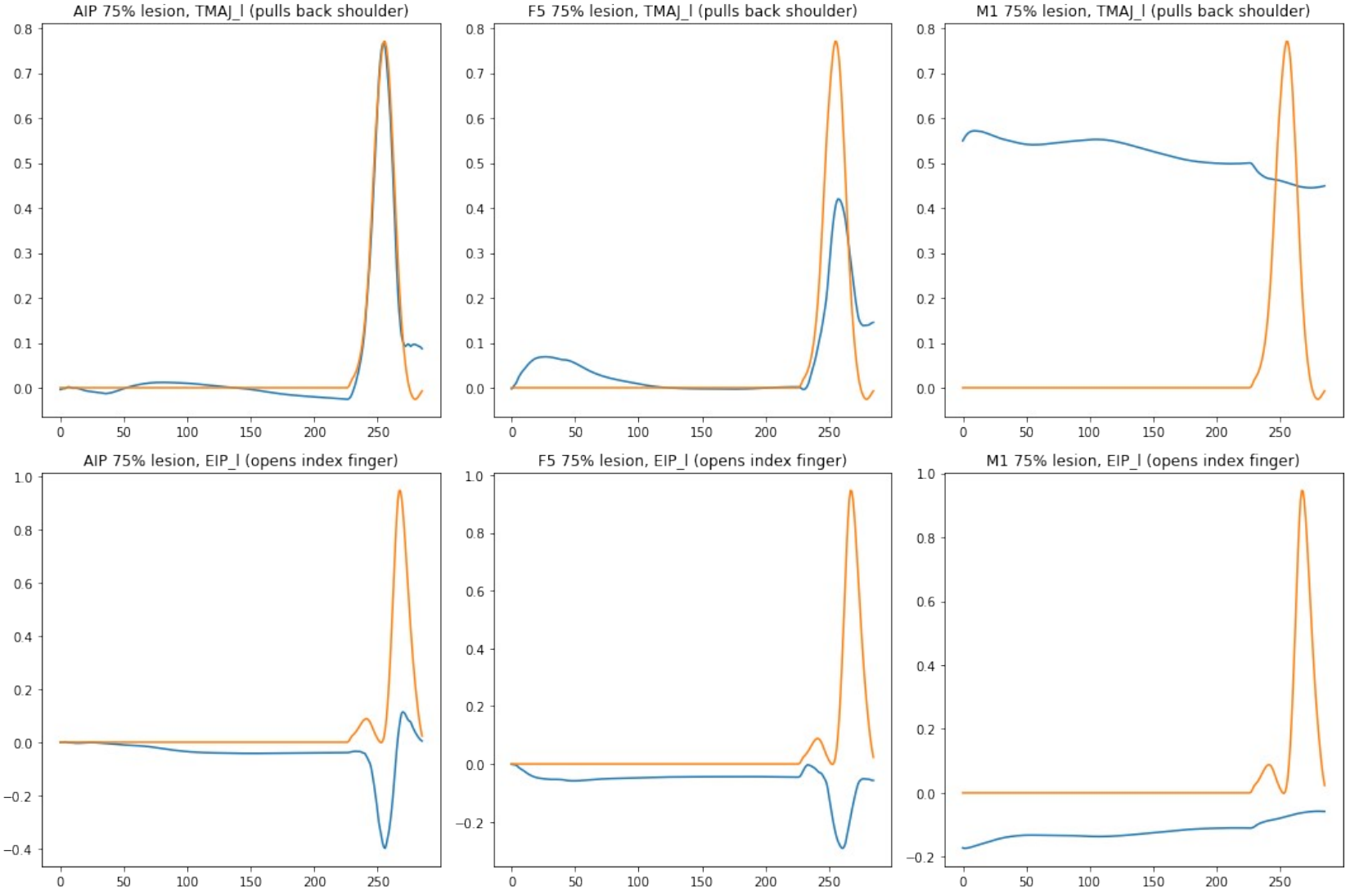
Review – the Michaels “modular RNN” model



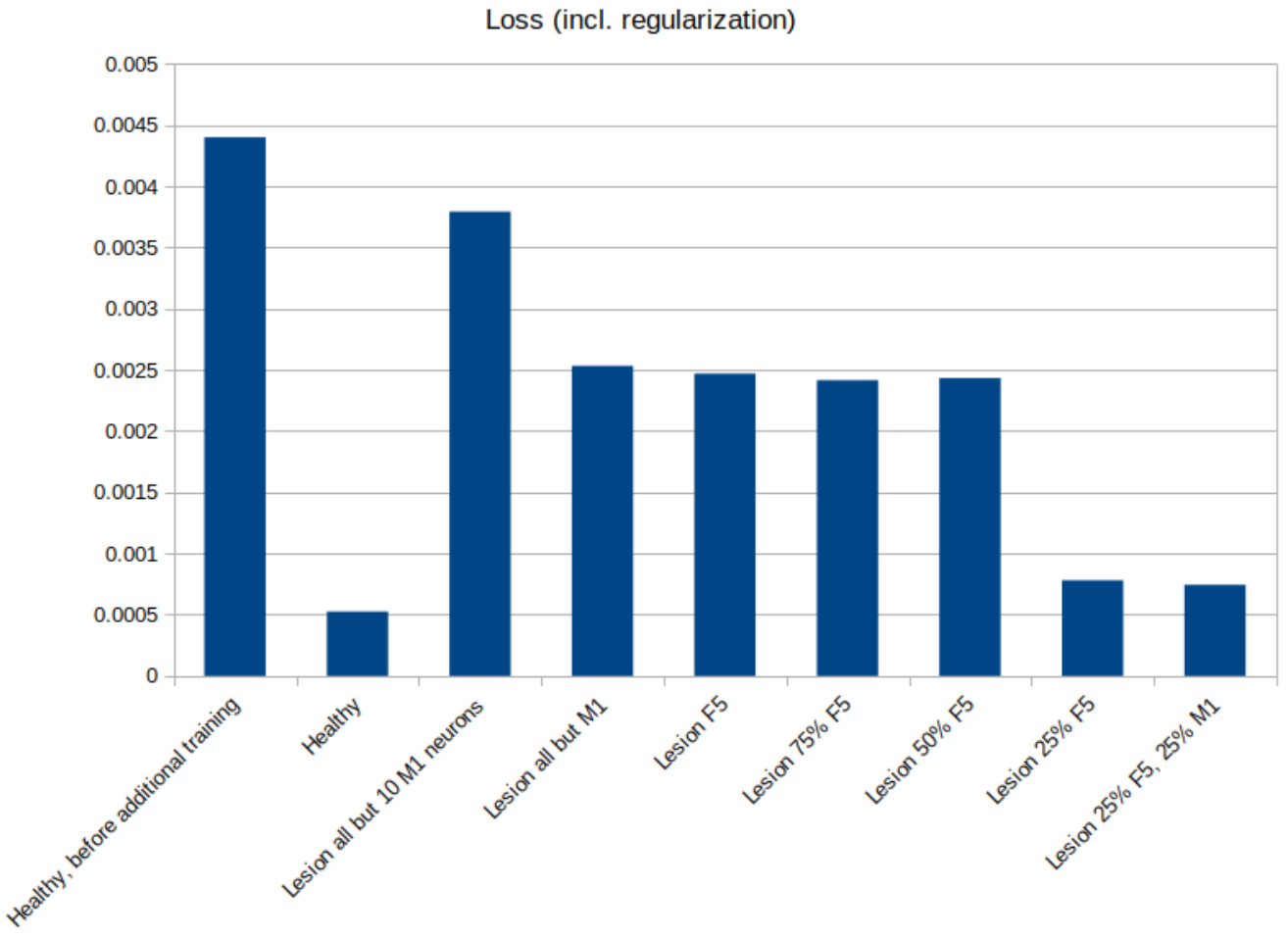
Effect of a lesion



Simulating recovery before adding coproc?

- Goal: simulate recovery after lesion
- Co-processor will improve from there

- Challenge: how do we train it?
  - Learned solutions will be very different than original, unless care is taken
  - Is this due to HFO vs. Adam? Stopping condition?
  - Do we care?



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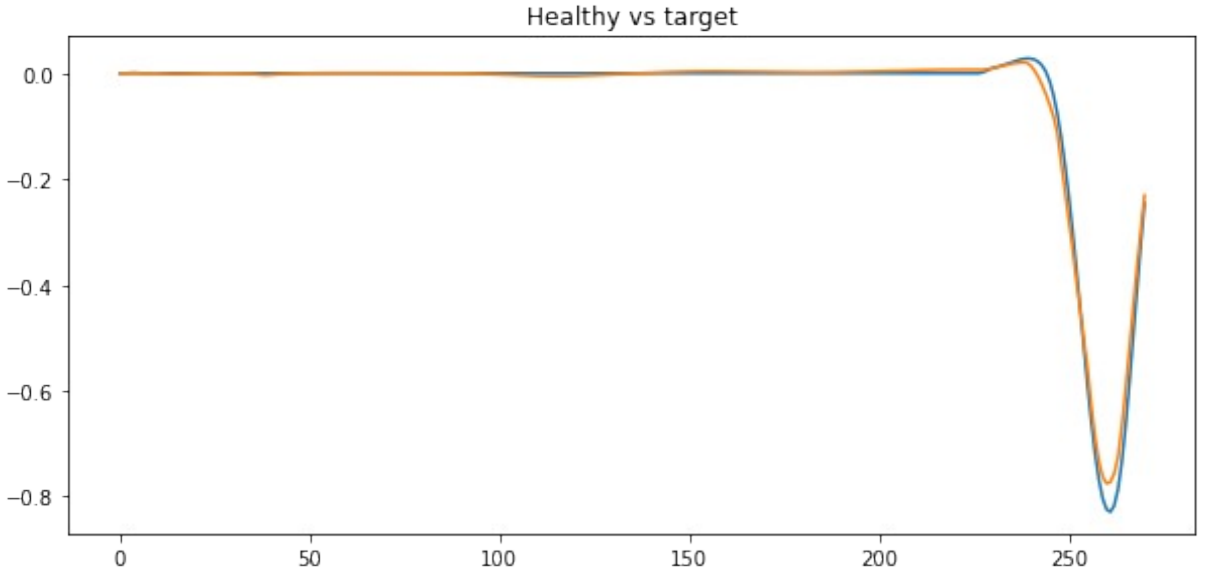
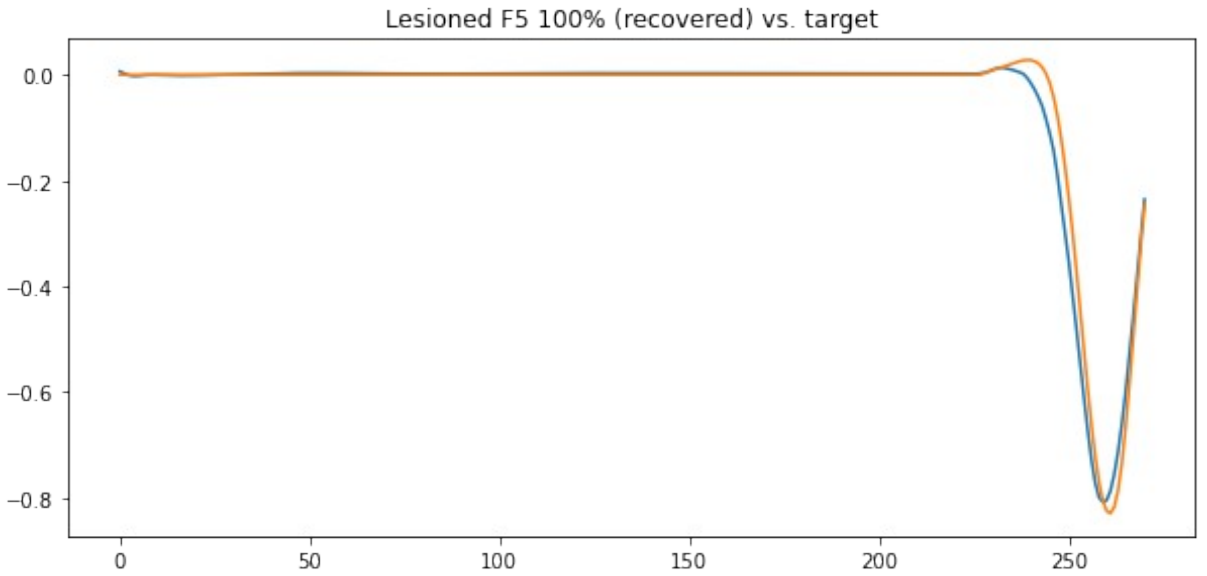
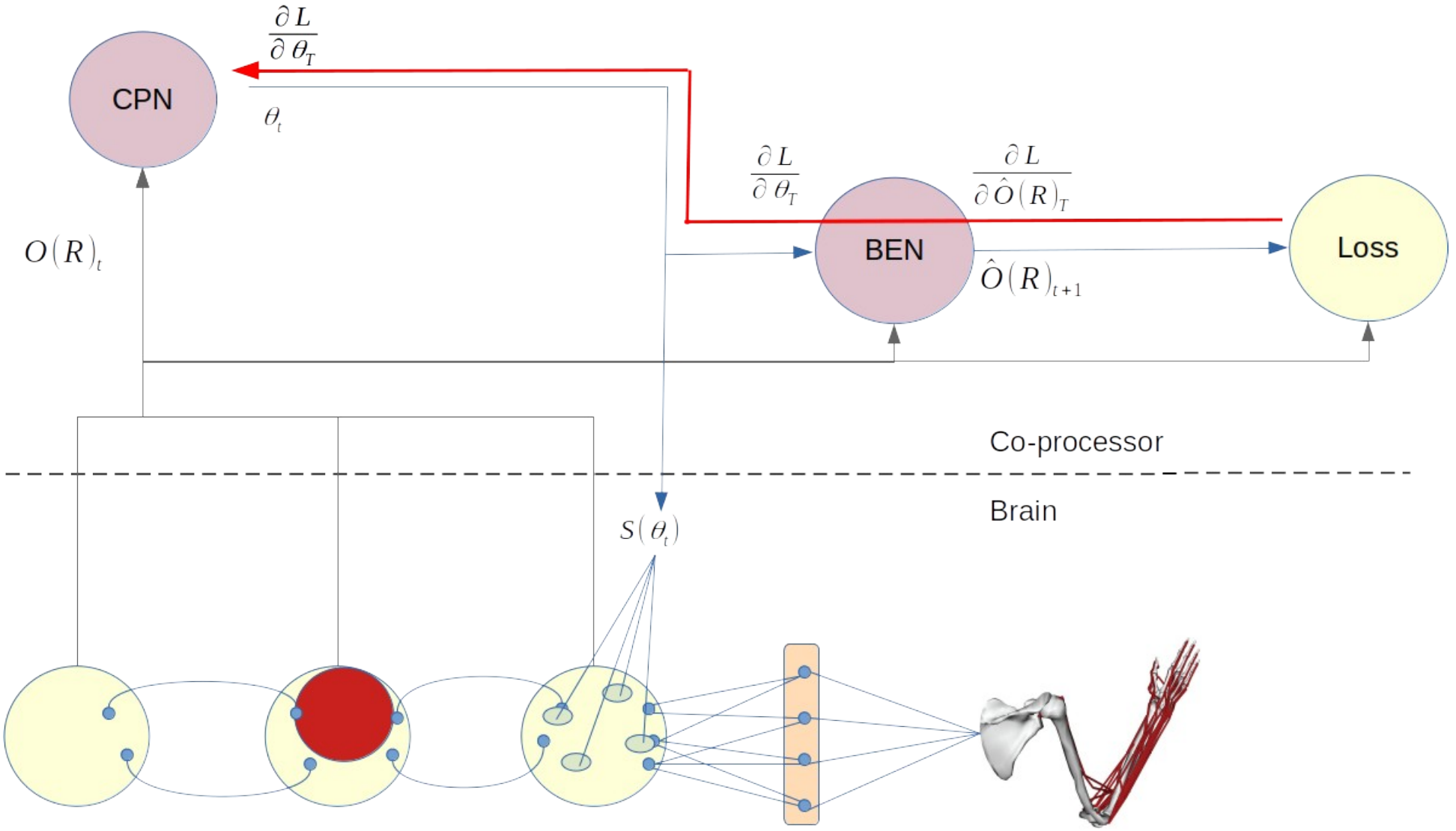
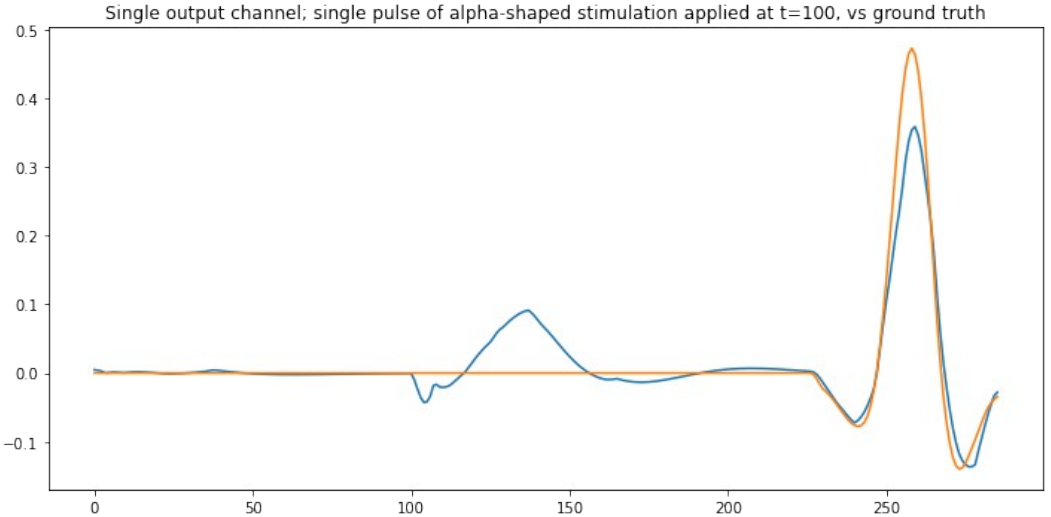


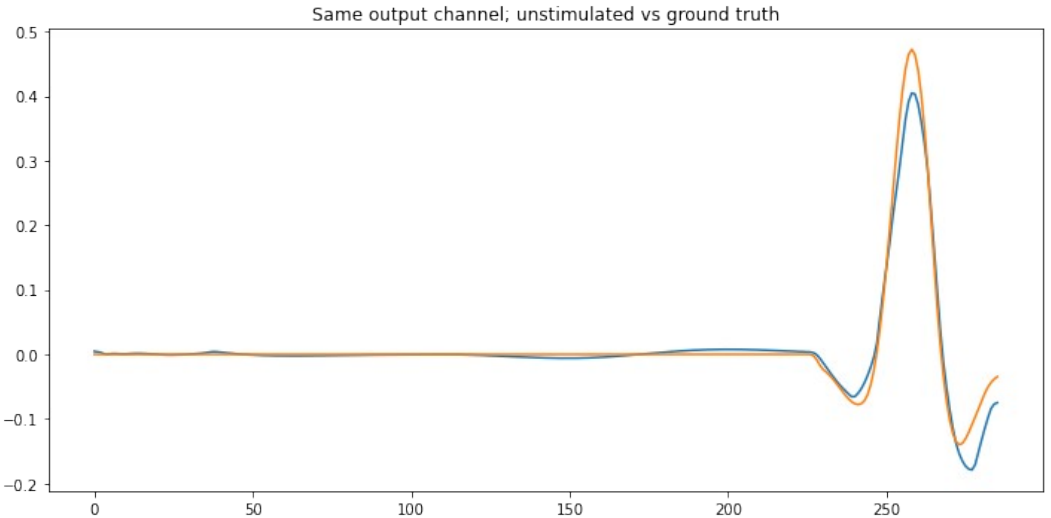


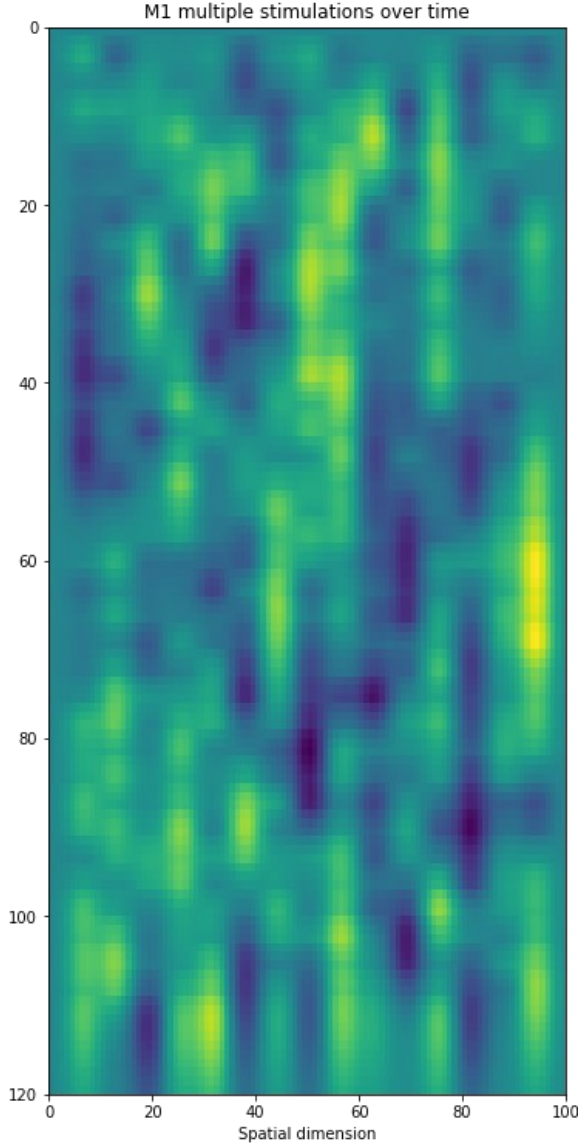
Driving towards target brain activity



Stimulation function



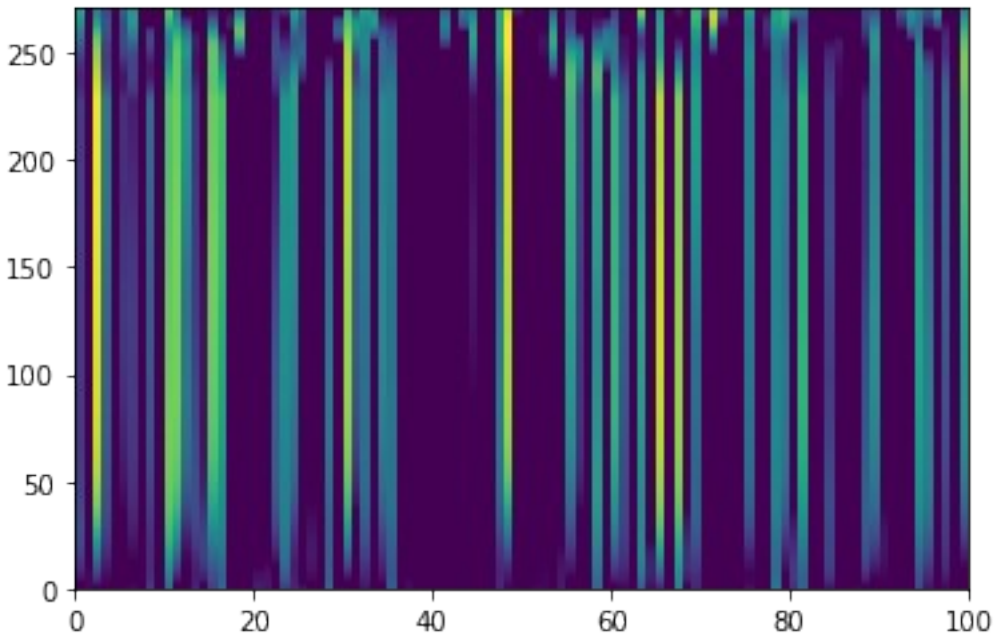




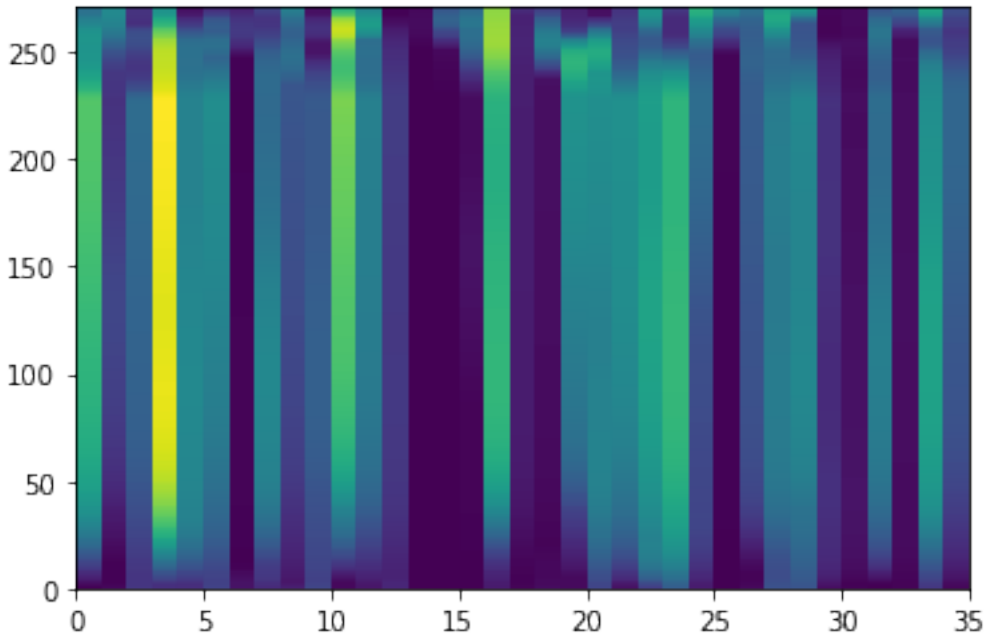
Observation function

- Represents our inability to observe every neuron directly
- Co-proc must operate on incomplete information
- Examples: observe a subset of neurons, average “nearby” neurons

M1 activity



M1 activity, spatial (Gaussian) convolution



Brain emulator network (BEN)

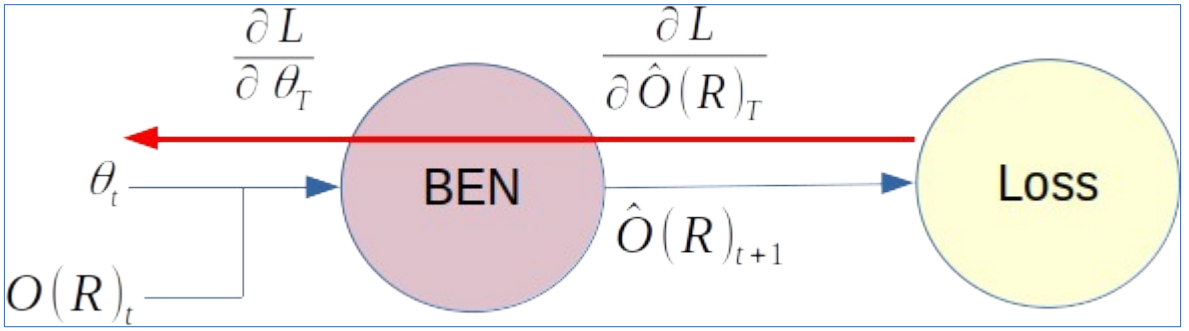
- A stimulation and brain dynamics model
  - Similar in structure to Yang/Shanechi et al.
- Trained independently, on random stimulations

- Loss is backpropagated through BEN to train co-proc:
  - Prediction loss – ignored; so BEN needs to generalize well and be low error at this point.

  - Stimulation loss – trains co-proc

$$\frac{\partial L}{\partial \theta_T}$$

$$\hat{O}(R)_{t+1} = f(O(R)_t, \theta_t)$$



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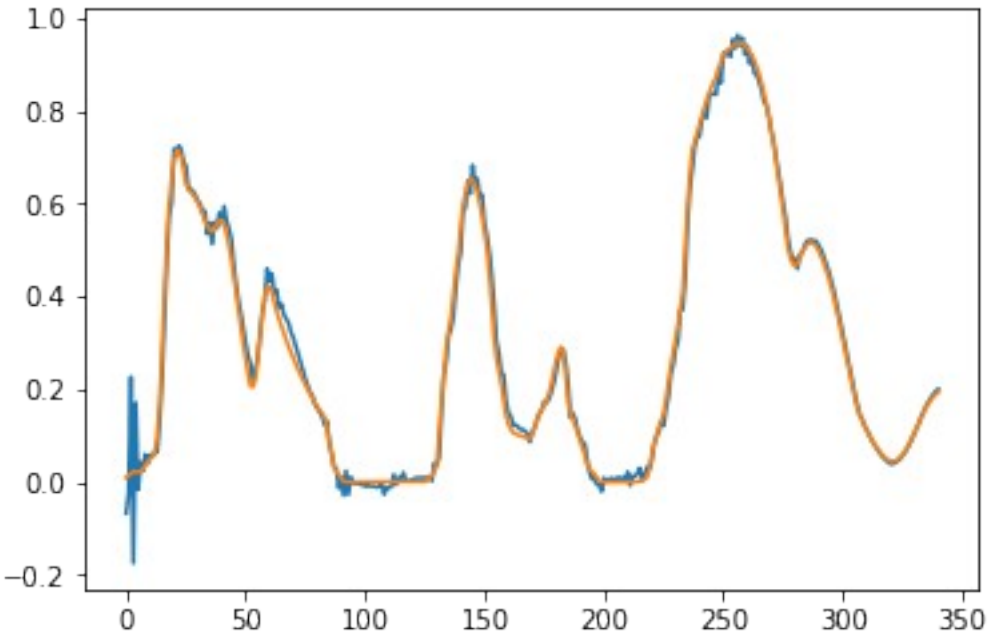

Outstanding challenges / next steps

- CPN learning is extremely slow
  - Gradients are ~4-8 orders of magnitude smaller than model params
  - Hypothesis: BEN’s neurons are saturated; more regularization needed

- Revisit recovery, and retraining the healthy network