State feedback control model can account for differences in abnormal pitch perturbation responses in Alzheimer's disease and cerebellar ataxia

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Introduction

It has been observed that patients with Alzheimer's disease (AD) and cerebellar ataxia (CA) demonstrate an abnormal speech motor control response to pitch perturbation experiments [1,2]. Here, we attempt to explain these differences using a state feedback control (SFC) model of pitch [3]. The differences between model parameters that correspond with experimental differences between patients and controls can generate hypotheses about how the speech motor control system may function differently in these patient populations.

State Feedback Control Model

- Our model of speech motor control is based on principles of state-feedback control (SFC) [1].
- In this model, motor commands are generated based on an internal estimate of vocal tract state.
- The estimated state and the generated commands are used to predict vocal tract state and auditory and somatosensory feedback for the next time step.
- The predictions are then compared with the actual feedback to calculate error signals.
- These errors are scaled by a Kalman gain and used to update the predicted state of the vocal tract, which is used to generate the next set of commands.

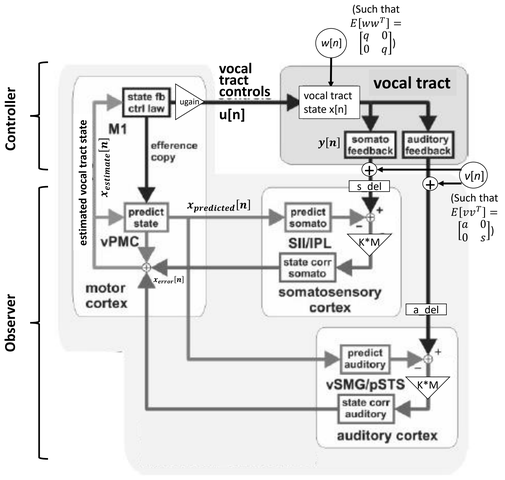


Figure adapted from [3]

Model Parameters

- **Vocal Tract State Feedback Model:**
  - System equations:

$$x[n] = Ax[n-1] + Bu[n-1] + w[n]$$
$$y[n] = Cx[n] + Du[n] + v[n]$$

  Where x = vocal tract state, y = sensory feedback (auditory and somatosensory feedback), u = control input, w = process noise, and v = measurement noise

  - Noise:

$$Q = E[ww^T] = \begin{bmatrix} q & 0 \\ 0 & q \end{bmatrix}$$
$$R = E[vv^T] = \begin{bmatrix} a & 0 \\ 0 & s \end{bmatrix}$$

  Where Q is state noise covariance, R is sensory noise covariance, a is auditory noise covariance, and s is somatosensory noise covariance.

- **Observer:**

$$x_{predicted}[n] = Ax_{estimate}[n-1] + Bu[n-1]$$
$$y_{predicted}[n] = Cx_{predicted}[n]$$
$$y_{error}[n-del] = y[n-del] - y_{predicted}[n-del]$$
$$x_{error}[n] = K * M * y_{error}[n-del]$$
$$x_{estimate}[n] = x_{predicted}[n] + x_{error}[n]$$

- **Controller (State Feedback Control Law):**

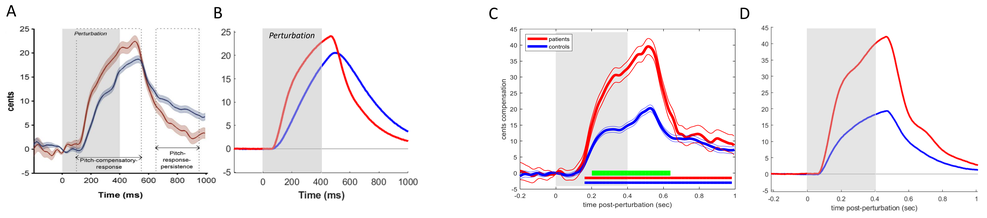
$$u[n] = ugain * (x_{target}[n] - x_{estimate}[n])$$

  Where K is a scaling factor on steady-state Kalman gain M, u is controller output, and ugain is controller gain. Sensory feedback y is a matrix of auditory and somatosensory feedback. Sensory feedback is delayed by del = [a_del,s_del]. Auditory feedback is delayed by a_del and somatosensory feedback is delayed by s_del.

- **Parameters explored are:**
  - Auditory **(a)** and somatosensory **(s)** noise covariance
  - State noise covariance **(q)**
  - Kalman gain scaling factor **(K)**
  - Controller gain **(ugain)**
  - Auditory **(a_del)** and somatosensory **(s_del)** feedback delays.
- Together, these parameters affect the characteristic shape of the pitch perturbation response

Results

- Adjusting Kalman gain scaling factor (K) on sensory feedback in simulation (Figure B) can reproduce many of the differences observed experimentally [2] (Figure A) between the pitch perturbation responses of controls (blue) and AD patients (red).
- Scaling somatosensory noise covariance (s) in simulation (Figure D) can reproduce many differences observed experimentally [3] (Figure C) between the pitch perturbation responses of controls (blue) and CA patients (red).



Discussion

- **Qualitative Analysis:** Quantitative conclusions are not possible due to the heterogeneity of vocal tract dimensions, damping, and other properties across individuals. However, a qualitative analysis of modelling results can provide insights into which computational and neural mechanisms may differ between patient and control populations, and lead to testable hypotheses for behavioral imaging studies.
- **Response Persistence:** A goal of future simulations is to more accurately model response persistence, i.e. the return to baseline pitch after perturbation offset and how it varies between controls and patients. Experimentally, the effect of a 400 ms pitch perturbation persists such that even 600 ms after perturbation offset, the subjects' pitch remains 4-10 cents above pre-perturbation baseline.

Conclusion

- **AD Patients vs. Controls**
  - Observed differences can be modelled by a uniform scaling of Kalman gains on both auditory and somatosensory feedback
  - **Hypothesis:** Higher overall Kalman gains are found in AD patients than in controls, but AD patients and controls have similar ratios of gains on auditory and somatosensory feedback.
- **CA Patients vs. Controls**
  - Observed differences can be modeled by selectively scaling somatosensory noise and Kalman gain on somatosensory prediction errors.
  - **Hypothesis:** CA patients have greater Kalman gain for auditory feedback prediction error than somatosensory feedback prediction error.
  - This could either be due to greater somatosensory noise, or to lower auditory noise in CA patients compared to controls.

References

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