A pulse-step model of speech motor control

EVIDENCE FOR AN EXTRINSIC PACEMAKER

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What's new here?

We are proposing that discrete pulse step signals descend from the motor cortex (MC) to set muscle activation levels. Discrete MC control models are rare but not unknown e.g. the bang-bang control model by Lieb et al [1].

Isn't this the equilibrium point λ model?

It shares Feldman's concept [2] that motor commands adjust motoneuron (MN) activation threshold levels and spindle or mechanoreceptor afferents activate MNs according to the threshold.

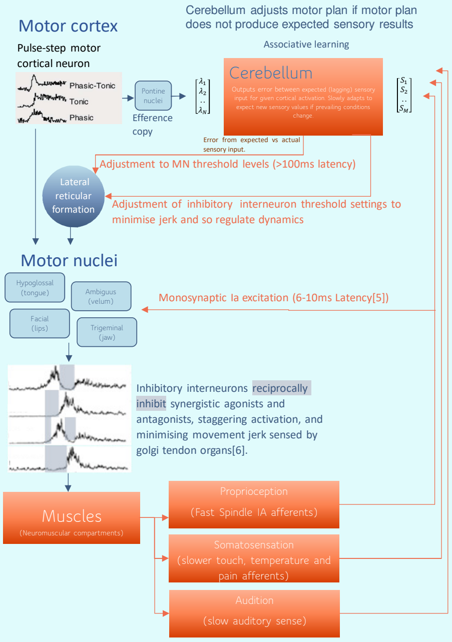
Key differences

1. Simple tonic **final position control** but **with** an initial phasic pulse that determines **velocity**.
2. The apparent continuous nature of λ is due to **inhibitory interneurons** within and between motor nuclei which **delay MN activation** by briefly, reciprocally, suppressing MN threshold levels.
3. Cerebellum compares (lagging) sensory input with memory of efference copy of MC neuron output. **Difference between expected and actual sensory input adjusts MN activation** levels after >100ms.

Hypothesis

It is known that individual hypoglossal neurons respond to oscillatory input with increased spiking and fire with greater temporal precision when the input is delivered at the "preferred" frequency of the cell [3]. **We hypothesise the existence of a rhythmic cortical or cerebellar pacemaker that phase-locks each pulse and step.**

The pulse step model



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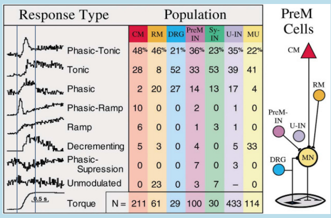
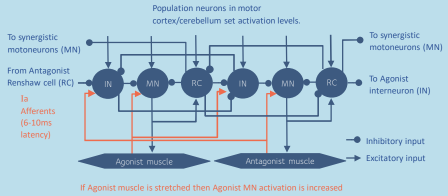


Response Type	CM	RM	DRG	PreM IN	Sy-IN	U-IN	MU
Phasic-Tonic	48%	46%	21%	36%	23%	35%	22%
Tonic	28	8	52	33	53	39	41
Phasic	2	20	27	14	13	17	4
Phasic-Ramp	10	0	0	2	0	1	0
Ramp	6	0	0	1	3	1	0
Decrementing	5	3	0	4	0	5	33
Phasic-Supression	0	0	0	7	0	3	0
Unmodulated	0	23	0	3	7	–	0
Torque N =	211	61	29	100	30	433	114

Summary of response patterns for different populations of neurons during generation of flexion and extension torques at the wrist.

CM	corticomotoneuronal cells
RM	rubromotoneuronal cells
DRG	premotor afferents in dorsal root ganglia
PreM-IN	spinal premotor interneurons
Sy-IN	spinal interneurons with synchrony effects
U-IN	spinal unidentified interneurons
MU	motoneurons

Motor nucleus (simplified Ia circuit [4])



If Agonist muscle is stretched then Agonist MN activation is increased and Antagonist MN activation is suppressed.

Method

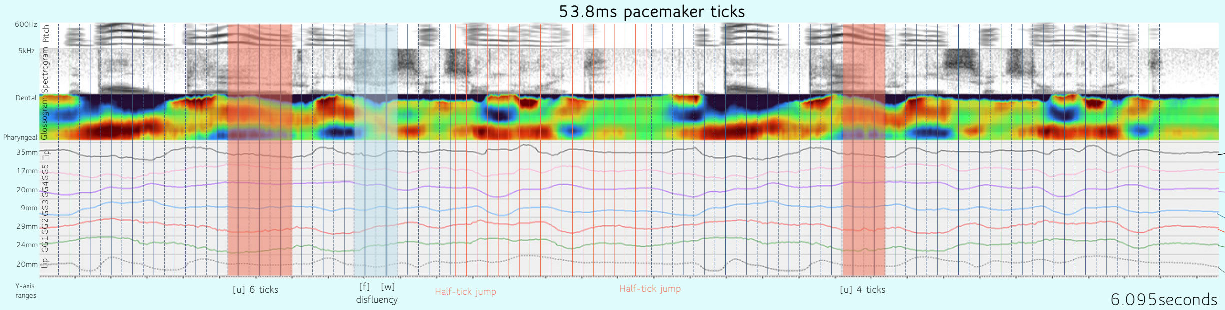
A co-registered EMA and ultrasound single speaker dataset was used to estimate keypoints along the entire tongue surface. Contraction and extension of 5 points along the tongue body plus a point on the tip were measured relative to the short tendon. Lip closure distance between EMA coils was also measured. A vertical grid was then manually fitted so that it best aligned with peaks and troughs of the measures. A single utterance with two repetitions of a sentence is analysed in this presentation. The first repetition includes a disfluency.

Dataset

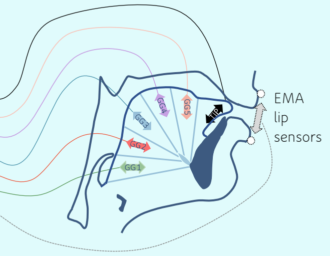
220 phonetically balanced sentences recorded by a male Scottish English speaker 50-60yrs. Synchronous instrumentation included 22kHz audio, Carstens AG501 EMA, 4 tongue sensors, upper/lower lip, jaw sensors. MicrUS 81Hz midsagittal Ultrasound. 60Hz head stabilised lip video .

The bulb blew when he (fw)switched on the light. The bulb blew when he switched on the light.

53.8ms pacemaker ticks

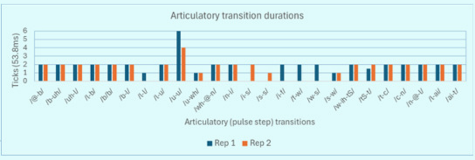


DeepLabCut tongue keypoint estimates trained on speaker specific EMA data. Accuracy s.d. ±1.6mm [7]



Observations

1. Rhythmic ticks of period 53.8ms align with the same acoustic landmarks in both repetitions and with initiation of many if not all articulatory transitions.
2. Most phone transitions take two ticks (107.6ms)
3. After the disfluency, in the /tS/ segment, there appears to be a half-tick jump. Then another half-tick jump in the pause between repetitions (indicated by red grid ||||||).
4. /u/ in "blew" reduced in duration by 2 ticks in second rep.



If there is a rhythmic pacemaker, how might local prominence work?

Three ways

1. Increase/decrease pulse to **vary the velocity** at which the end effector reaches its target.
2. **Sustain the tonic target activation level** for an extra N pacemaker ticks, increasing prominence
3. Learn a pulse step sequence that goes from phone A to phone C but passes through sensory locus of phone B e.g. **a syllable plan** (see Sosnik [9]), reducing prominence.

Discussion

We have used carefully designed novel measures of genioglossus contractions rather than traditional EMA sensor tangential velocity measures, taking the analysis a step closer to the underlying motor commands.

Evidence of an underlying pacemaker presented here is subjective and still exploratory. We have not yet found a means for automated detection of a pacemaker.

Similar tick periods are observed in other recordings by this speaker in the dataset. Mean=54.0ms s.d.= ±0.3ms N=11

Cortical oscillatory entrainment of single hypoglossal motor units has been observed, by means of a correlation with electroencephalography (EGG), to peak at around 20Hz [8] which matches the rate discovered here.

It should be noted that the pulse step model is **compatible with**, rather than a competitor to, **the DIVA speech production model** [10].

References

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