Lack of evidence for synergistic control of muscles across legs or anatomical groups during treadmill walking

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Summary

'Muscle synergies' have been proposed to simplify neural control of movement[1] and cortical damage is thought to change the structure of these neural commands [2, 3]. Here we test these hypotheses by analyzing muscle activity from unimpaired and post-stroke subjects during a walking task. We asked the following questions:

- Is dimensionality of EMG data evidence of synergies? (Spoiler: probably not)
- Can we find evidence of synergistic neural control during walking? (Spoiler: yes, by looking in inter-trial variability)
- What is the structure of the synergies? (Spoiler: anatomical muscle groups)
- Are they different between stroke patients and controls? (Spoiler: no)

Methods

- 16 chronic post-stroke subjects (6 female) and age and sex matched controls completed a split-belt treadmill task.
- EMG recorded from 15 muscles on each leg.
- Band-pass(20-500Hz), rectification and low-pass (10Hz) filtering to extract EMG amplitude.
- Synergy extraction: factorization of data ($X \approx H.W$) was performed using PCA unless indicated otherwise.
- Factorization performance assessment (EAF or tVAF):

$$d(H,W) = 1 - ||X - H.W||_F^2 / ||X||_F^2$$

Muscle name	Abbrev.	Muscle name	Abbrev.
Tibialis anterior	TA	Rectus Femoris	RF
Peroneus longus	PER	Vastus Lateralis	VL
Medial Gastrocnemius	MG	Vastus Medialis	VM
Lateral Gastrocnemius	LG	Sartorius	HIP
Soleus	SOL	Adductor Magnus	ADM
Biceps Femoris	BF	Gluteus Medius	GLU
Semitendinosus	SMT	Tensor Fascia Latae	TFL
Semimembranosus	SMB		

Table 1: List of muscles & abbreviations.

ID	Age	Fugl-Meyer (/34)	TM speed (m/s)	Affected	Sex	Control age	Control speed (m/s)
P01	44	33	1.13	R	F	46	0.94
P02	55	26	0.81	R	F	51	1.02
P03	65	29	0.60	R	F	65	1.08
P04	58	21	0.45	R	F	58	0.9
P05	55	31	0.94	L	M	57	1.04
P06	64	31	0.33	L	M	52	1.05
P07	78	22	0.23	R*	M	78	0.66
P08	54	23	0.87	L	F	52	1.16
P09	66	30	0.77	R	M	68	0.85
P10	60	26	0.90	R	F	62	0.98
P11	77	30	0.35	R	M	75	1.11
P12	59	32	0.70	R	M	57	0.99
P13	52	32	0.96	R	M	52	1.16
P14	66	29	0.77	L	M	64	1.25
P15	75	32	0.94	R	M	74	1.11
P16	49	33	0.72	R	M	49	1.08
Avg.	61 ± 9.9	28.7 ± 3.95	0.72 ± 0.26	12R/4L	10M/6F	60 ± 9.9	1.02 ± 0.14

Table 2: Subject summary

Low-dimensionality in muscle activity results from spectral features

Desynchronized EMG data is low-dimensional

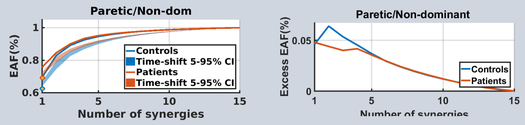

Figure 1: EAF vs. number of synergies in actual and randomly time-shifted data. Time-shifted data was generated by delaying each individual EMG signal by an arbitrary amount. This de-synchronizes activity across muscles while preserving spectral properties. Right: Excess EAF. Excess is actual EAF minus the median of the time-shifted distribution. Less than 6% of the signal energy can be accounted for by muscle synchronism (synergies).

Spectral features are different across populations

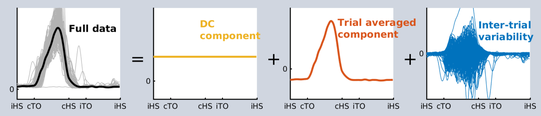

Figure 2: Decomposition of a pseudo-periodic signal: DC, trial-averaged, and inter-trial variability. The energy of these three adds to the total energy ($E = \sum_i x_i^2$).

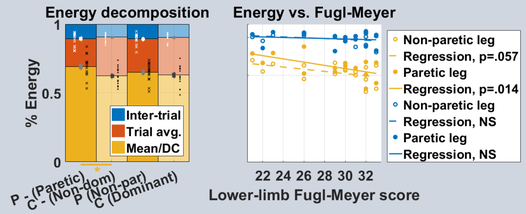

Figure 3: Average DC component across muscles is statistically different between patients and controls. Fugl-Meyer scores correlate positively with DC component of EMG signal energy (p=.014) for the paretic leg.

Synthetic random data is low dimensional in absence of synergies

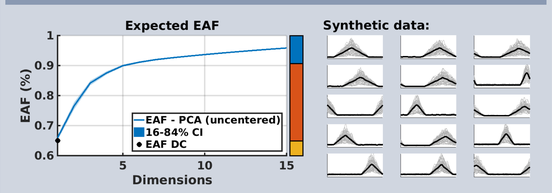

Figure 4: Right panel: sample of synthetic data after post-processing. Black trace represents the mean across cycles and grey traces represent individual cycles. Left panel: distribution of EAF vs. dimensionality. EAF was computed with both uncentered PCA and NNMF (results overlap). Even when each channel's data is generated independently of others (no synergies) a few dimensions account for most of the energy of the dataset.

Inter-trial variability shows neural control structure

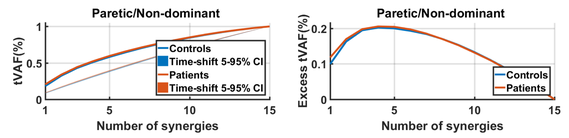

Figure 5: Left: tVAF vs. number of synergies in actual and randomly time-shifted data, averaged across subjects. Time-shifted distributions show little structure (data does not lie in low-dimensional space). Right: Excess tVAF vs. number of synergies. 4 synergies account for 20% more variance than expected by the signal's spectral characteristics.

Bilateral synergies are not observed in walking

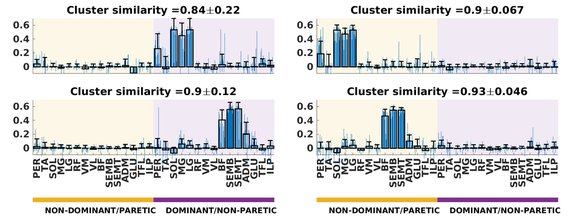

Figure 6: Clustered synergies extracted from trial-to-trial data. 8 synergies extracted per subject (N=32) on bilateral data, and separated into 10 clusters using cosine distance. Only the four most consistent clusters are shown. The cluster centroids (thick bars) show no cluster includes significant activity from muscles in both legs, and each of the eight most significant clusters have a symmetric contralateral cluster.

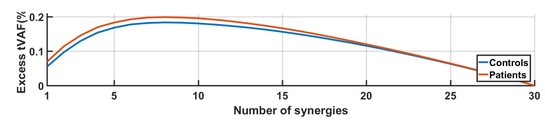

Figure 7: Excess tVAF vs. number of synergies in the bilateral dataset. 8 synergies account for 20% more variance than expected by chance, consistent with a lack of bilateral synergies (4 unilateral synergies per side also account for 20%).

Weak muscle covariation outside anatomical groups

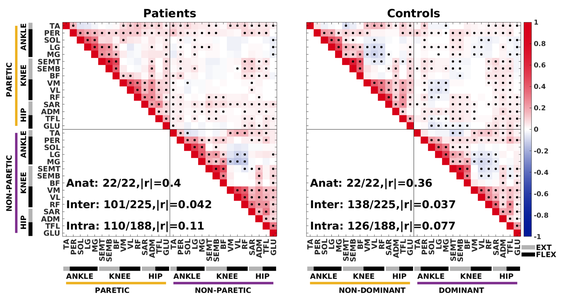

Figure 8: Comparison of covariance matrices on trial-to-trial variability during baseline walking. Colors reflect mean value across the population, black dots indicate values significantly different from 0. While many correlations are significant (dots), the median correlation values ($|r|$) are very low except for anatomical groups.

Full covariances change with task, but inter-trial doesn't

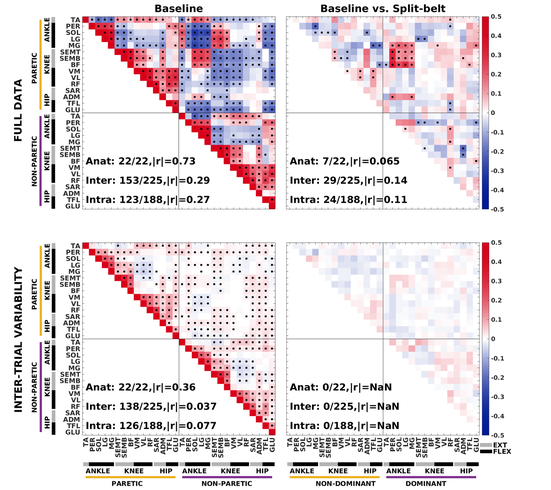

Figure 9: Top: full data covariance matrix in baseline (left) and difference to split-belt walking (right). Bottom: inter-trial variability covariances. Dots indicate significance. Comparisons were classified as same anatomical group (Anat), in same leg (Intra) or from different legs (Inter). Number of signif./total comparisons is displayed in text along with median r.

Conclusions

1. Low-dimensionality of co-recorded EMG signals is mostly caused by its spectral characteristics and NOT muscle coordination.
2. Reported differences in synergy number/structure in pathological populations [3, 2] may be explained through differences in spectral characteristics between groups.
3. Inter-trial variability is a better way to assesses # of muscle synergies & synergy structure.
4. Contrary to previous reports [4], synergies for walking extracted from trial-to-trial data are unilateral and mostly symmetrical in patients and controls.

References

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