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GENERATING COMPUTER SIMULATIONS OF MOVEMENT USING MUSCLE SYNERGY INPUTS

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SUMMARY

Motor control research has long attempted to understand how the central nervous system coordinates the actions of a highly redundant muscle set during movements. As a potential answer, researchers have proposed the existence of a modular control architecture whereby multiple muscles can be simultaneously co-activated by a single, preset control pattern known as a module or synergy (See [1] for a detailed review). Individual contributions from finite synergies can then be scaled in magnitude and linearly summed up (termed synergy re-combination) to yield the resultant control signal (termed muscle excitation) which then causes muscle contraction [2]. By merely changing the temporal scaling patterns for recombination, a small set of synergies can thus generate excitation patterns for actuating a family of movements (e.g., resisting postural perturbations in various directions [3]).

Thus muscle synergies may represent fundamental neural control strategies responsible for actuating a family of movements. While methods such as non-negative matrix factorization (NNMF) can identify a set of subject-specific muscle synergies from EMG collected for a variety of movements, it is extremely difficult to conversely identify the entire family of movements which a given synergy set can actuate. Computer simulations using musculoskeletal models may however verify whether a specific movement belongs to that family of realizable movements. In this abstract we describe a novel simulation approach developed for that purpose and demonstrate how we can verify whether muscle synergies for healthy human walking are capable of reproducing experimentally observed gait mechanics in a model. Such a simulation method could have applications in the rehabilitation of movement disorders. The problem was first formulated as an optimal control problem (OCP) which was then solved by direct collocation [4,5], a numerical method known for its computational speed and flexibility of OCP formulation.

METHODS

The kinematics, kinetics and EMG signals of a healthy subject walking at 0.7 and 1.7 ms^{-1} were recorded in a motion capture laboratory. EMG signals were only recorded for 8 muscle groups on the right side of the lower extremity, namely the tibialis anterior (TA), medial gastrocnemius (MG), soleus (SO), vastii (VI), rectus femoris (RF), biceps femoris long head (BFLh), gluteus medius (GMEd) and gluteus maximus (GMax). NNMF based decomposition [6] of EMG signals from 7 gait cycles each at the two walking speeds yielded four muscle synergies (Figure 1). During simulation, only excitations for these 8 muscle groups were dictated by synergy recombination while other muscle excitations (termed 'free muscles/excitations') could assume arbitrary patterns.

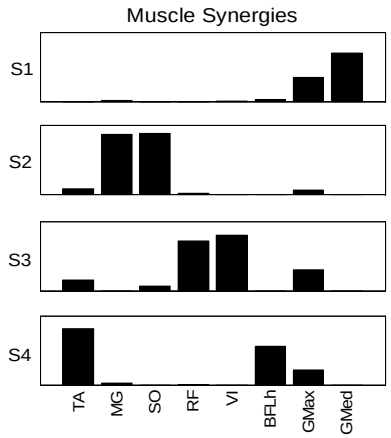

Figure 1: Contributions of the 4 muscle synergies (row-wise) towards excitations in 8 muscles. During recombination, the contributions from a synergy are scaled by a synergy-specific, time-varying activation coefficient and scaled contributions from each synergy are summed up to yield the respective muscle excitations.

The 'gait 2354' model from OpenSIM [7] first underwent scaling, inverse kinematics and inverse dynamics (ID) using the experimental data. An OCP then sought values of the time-varying synergy activations and free excitations which as control inputs to a forward integration of the muscle model, would reproduce experimental ID joint moments in the musculoskeletal model (similar in principle to the forward-inverse model in [8]). Direct collocation was used to convert the OCP into a numerical optimization problem whose cost function simultaneously minimized errors between simulated and ID moments along with activations in the free muscles. The optimization was solved using SNOPT [9].

Due to approximations involved in direct collocation, dynamic consistency of the OCP solution was first verified by actually integrating the muscle model with initial states and control signals from direct collocation. If errors between the resulting joint moments and those from ID were low, the synergies could be deemed capable of reproducing gait mechanics in the model. Additionally we compared synergy driven muscle excitations with the respective muscle's EMG (which synergies were derived from).

RESULTS AND DISCUSSION

Direct collocation was successful in solving the OCP and computing feasible control inputs for the model. Simulated joint moments hereby refer to the joint moments produced in the model during the forward integration test. Mean errors and correlation coefficients calculated between simulated and ID joint moments (Figure 2) over all degrees of freedom were 4.88 Nm and 0.98 respectively. The low errors and high correlations verified that the experimentally derived muscle synergies were indeed capable of reproducing healthy gait mechanics in the musculoskeletal model.

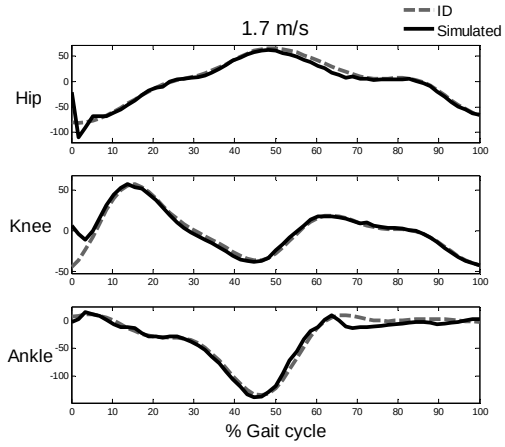

Figure 2: Comparing ID and simulated joint moments (from forward integration of the DC solution) for the flexion/extension degrees of freedom at 3 joints for 1 gait cycle at 1.7 ms^{-1}. All moments specified in Nm.

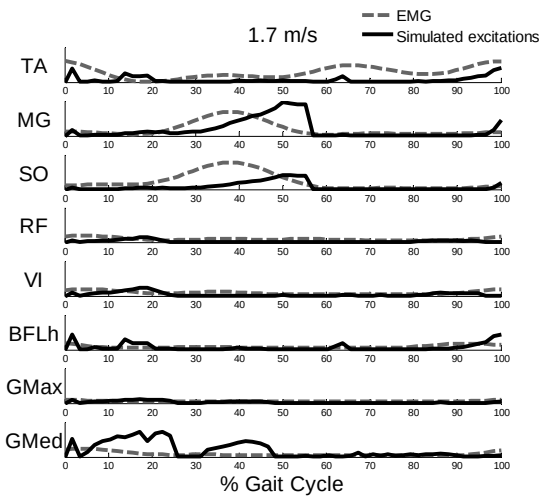

Figure 3: Comparing simulated excitations from direct collocation with experimental EMG for one gait cycle at 1.7 ms^{-1}.

The mean correlation coefficient between simulated excitations and EMG (Figure 3) over all synergy driven muscles was 0.29 ($\pm$ 0.13). Thus low correlations were observed despite synergies being capable of reproducing EMG signals if needed (from NNMF). Incorrect model parameters could be the reason behind this observation. For example, differences in EMG-excitation peaks could be caused by incorrect activation/deactivation constants [10]. Such inaccuracies probably necessitated simulated synergy activation profiles to differ from the NNMF values, so that experimental joint moments could be reproduced in the model. The presence of free muscles especially at the hip where surface EMG for most muscles is evasive could have also influenced the excitations of synergy driven muscles.

CONCLUSION

While earlier studies [11] have developed simulations using muscle synergies as inputs, the cost of computing the time varying activation coefficient profiles restricted them to simulating movements similar to ones which the synergies were derived from using NNMF. The methodology proposed in this study overcomes this cost by using direct collocation instead of simulated annealing that was used earlier. Thus for example, one could verify whether walking synergies can also actuate jumping, or whether a stroke subject's walking synergies can also actuate healthy gait by merely varying the activation profile. This could potentially be useful in planning rehabilitation from movement disorders. Synergies computed from a stroke subject's EMG could be used as control inputs to simulate healthy gait mechanics whereby identifying compensatory muscle coordination strategies that would be utilizing the stroke subject's inherent neural control strategies.

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REFERENCES

1. Tresch, M.C., and Jarc, A., *Current opinion in neurobiology*. **19**: 601–607, 2009.
2. d'Avella, A., and Pai, D.K., *Journal of motor behavior*. **42**: 361–369, 2010.
3. Ting, L.H., and Macpherson, J.M., *Journal of neurophysiology*. **93**: 609–613, 2005.
4. Kaplan, M.L., and Heegaard, J.H., *Journal of biomechanics*. **34**: 1077–1083, 2001.
5. Ackermann, M., and van den Bogert, A.J., *Conference proceedings:Annual international conference of the IEEE EMBS*. **3**: 5444–5447, 2010.
6. Clark, D.J., et al, *Journal of neurophysiology*. **103**: 844–857, 2010.
7. Delp, S.L., et al, *IEEE transactions on biomedical engineering*. **11**: 1940–1950, 2007.
8. Buchanan, T.S., et al, *Medicine & science in sports & exercise*. **11**: 1911–1916, 2005.
9. Gill, P.E., et al, *SIAM Journal on optimization*. **12**: 979–1006, 2002.
10. Gopalakrishnan, A., *PhD Thesis*, University of Delaware, 2012.
11. Neptune, R.R., et al, *Journal of biomechanics*. **42**: 1282–1287, 2009.