Post-Stroke Movement with Motion Capture and Musculoskeletal Modeling

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Research of post-stroke locomotion via musculoskeletal (MSK) modeling has offered an unprecedented insight into pathological muscle function and its interplay with skeletal geometry and external stimuli. Advances in solving the dynamical system of post-stroke effort and the generic MSK models used have triggered noticeable improvements in simulating muscle activation dynamics of stroke populations.

stroke biomechanics musculoskeletal modeling pathophysiology motion analysis

1. Introduction

Post-stroke locomotion has been at the epicenter of clinical research [1][2][3] attempting to elucidate the neural and motor deficits underlying its deviation from the norm and to inform the rehabilitation process [4]. Hemiparesis—one of the most common signs of stroke—results mainly in muscle weakness, reduced peak muscle torque, a decrease in power generation and early fatigue [5], and an overall challenging postural stability [6]. Hence, stroke gait has been characterized as energetically expensive [7], with the lower limb joint power loss of the paretic leg mainly compensated by the increased mechanical output of the non-paretic side [8].

Methods to explore the pathophysiology of post-stroke movement involve clinical examinations and rigorous biomechanical analysis. The latter has mainly been addressed via the usage of traditional biomechanical models limited to the calculation of spatiotemporal parameters, joint angles, net joint moments, and, by extension, joint work [9][10][11][12]. Additional metrics include electromyography (EMG) [13][14] and muscle synergy analysis [15][16]. Their usability by non-experts made them applicable to many clinical settings, and they are widely used to assess patient function. However, they cannot sufficiently describe the interplay between external forces acting on the body and the internal muscle function to counteract it.

Musculoskeletal (MSK) modeling can build on the shortcomings of mainstream biomechanical analysis via incorporating mathematical formulations to solve the inherent muscle redundancy problem related to sharing the external loads amongst a joint's musculature [17][18]. The computational biomechanics community has invested decades into simulating healthy human movement, of both the upper and lower body, in the pursuit of mathematical formulations that can accurately describe its mechanical etiology. Hence, three-dimensional (3D), cadaver-based computer models of muscle geometry and physiology [19][20][21][22] have been extensively produced

and rigorously used to simulate human locomotion and have often been validated against synchronously measured muscle activity [23][24] or in vivo contact forces from joint replacement devices [25][26][27].

While these musculoskeletal models have provided reasonable results for healthy cohorts, pathological motion has proven more difficult to model. The central nervous system (CNS) intrinsically dictates muscle activation to result in healthy coordinated motion based on external and internal feedback. However, disease-driven impairment of this process substantially challenges the hypotheses of muscle coordination and CNS targets. Early efforts have accommodated a variety of methods to explain the observed motion [28], understand individual muscle roles [29], or predict a new motion by simulating a specific stroke-related symptom [30] to understand post-stroke motion. Furthermore, the personalization of muscle models according to individual measurements has also been incorporated in solving the muscle redundancy problem via the utilization of EMG signals to guide specific muscle activation levels [28]. User-friendly platforms, such as Opensim [31] or Anybody [32], have contributed to further dissemination of such modeling frameworks. Yet, their usage among medical experts has been highly discouraged, mainly due to (i) their complexity and their demand for highly technical skills, (ii) the poor dissemination of newly developed MSK models and modeling software, and (iii) the lack of knowledge of their possibilities to address clinical challenges.

2. Assessment of Orthotic Device and Exoskeleton

Documented usage of MSK modeling in the assessment of orthotic devices is scarce yet very informative regarding the effect of external devices on body function. Simulating the mechanical interplay between a device and the human body can be very challenging. In the study by Akbas et al. [33], different prescribed external knee torques driven by a wearable exoskeleton to maximize knee flexion during the pre-swing phase of post-stroke gait were simulated as coupled forces acting on the thigh and tibia, using 3D musculoskeletal models and a combination of inverse and forward dynamics analysis, namely, computed muscle control—CMC. In brief, CMC calculates the model accelerations that best match the experimental ones, estimates muscle forces for each instance that will achieve those accelerations using inverse dynamics, and performs a forward dynamics simulation that uses the forces to drive the MSK model to the next instant. Calculated muscle forces and fiber stretch velocities showed that such induced external torques may trigger rectus femoris hyperreflexia in stroke patients and consequent stiff-knee gait, coupled simultaneously with gluteus medius activation, mainly responsible for apparent hip circumduction.

Choi et al. [34] used a 3D musculoskeletal model to model the muscle function of a stroke patient when walking with ankle–foot orthosis in different prescribed shank-to-vertical angles. MSK modeling and standard inverse kinematics (i.e., calculation of joint angles based on recorded motion) facilitated the estimation of gastrocnemius musculotendon unit operating length, which enabled improved control of gait kinematics and the kinetics for maximum ankle plantar flexor moment and toe clearance during gait.

Both studies highlighted the usefulness of MSK modeling to present valuable information on muscle mechanics as influenced by external devices and guide their configuration to match the patient’s needs and improve biomechanical factors. In the past, MSK modeling has been used to test various “what if” scenarios and predict the

best assistive devices' parameters to optimize walking performance [35][36] or to avoid ankle injuries [37]; hence, future studies should build on this acquired knowledge to develop an a priori design optimized for assistive devices in silico and tailored to stroke patients' specific needs in order to avoid a potentially detrimental prescription.

3. Assessment of Intervention

Evaluating the effect of treatment is very crucial to designing and implementing rehabilitation plans for stroke patients; however, there is a very limited usage of MSK modeling to that end. The study by Lampire et al. [38] has found that BOTOX injection on spastic rectus femoris can increase peak knee flexion during swing phase as well as gait velocity, with MSK modeling used to show that the maximum normalized length and lengthening velocity of the specific muscle increases post intervention and improves stiff-knee gait.

Fast functional electrical stimulation (FastFES) treatment—a gait retraining intervention combining fast treadmill gait and electrical stimulation—on post-stroke ankle PF function has been the focus of both the following studies. Knarr et al. [39] used a mix of inverse and forward dynamics analysis (CMC and induced acceleration analysis (IAA)) to demonstrate that MSK modeling can detect the improvements witnessed in clinical practice by calculating increased ankle PF activation and consequent contribution to center-of-mass (COM) acceleration during the double support phase of gait, leading to an overall gait velocity increase. The next study by Sauder et al. [40] simulated specific muscle stimulation settings to predict the functional outcome post-intervention, thus finding optimal muscle stimulation settings for non-responders to standard FastFES treatment. This particular workflow included a rigorous and highly complex personalization of the musculoskeletal model, optimizing joint positions, muscle–tendon parameters, ground–foot contact model settings, and muscle synergy factors based on muscle EMG, kinematics, and kinetics data to most accurately reproduce the experimental marker position and ground reaction forces during gait. The muscle redundancy problem was solved through a direct collocation method.

4. Assessment of Movement Deficits

The vast majority of studies researched post-stroke gait and the accompanying muscle deficits, usually incorporating some level of personalization in the MSK model used. The work of Hall et al. [41] used CMC and IAA methods to identify limited forward propulsion in stroke patients, while Allen et al. [42] found that the asymmetry in forward propulsion is coupled with PF-impaired coordination. Both Hall et al. [43] and Henderson et al. [44] found that non-paretic enhanced muscle output functions as compensation for paretic limb weakness. A forward dynamic approach to explore muscle impairment was implemented by Knarr et al. [45] to predict how normal gait would be changed if specific limited muscle capacity was introduced in the model. An alternative method to determine which motor deficits drive stroke gait—initially generated motions of normal gait that can later be perturbed based on simulated muscle length and muscle lengthening velocity feedback gains (reflexes)—was implemented by Jansen et al. [46], while Ong et al. performed a similar analysis using a model of muscle force reflex [47]. This workflow allowed the authors to reproduce basic patterns of stroke gait, such as equinus or foot-drop gait, via modulating feedback gains on these reflexes, hence simulating spasticity. A recent study by Santos et al. [48] employed

predictive simulations and personalized muscle–tendon parameters to study the effect of isolated muscle impairments on the motion of one stroke patient and how FES can attenuate them. The knee extensors' weakness was related to knee hyperextension, and stiff-knee gait could be corrected by coupled knee flexor strengthening and knee extensor weakening. They later predicted and validated that FES on spastic rectus femoris can correct knee hyperextension and drop-foot but not stiff-knee gait. Finally, similar to work by Sauder et al. [40], EMG, kinematics, and kinetics data were used by Meyer et al. [49] to optimize joint center locations and muscle parameters that best follow recorded motion in an effort to best simulate patient-specific neuromuscular deficits or calculate the metabolic cost of stroke gait [50]. This method consisted of different complex simulation steps, a large number of which comprised a rigorous utilization of muscle synergy analysis to build dynamically consistent MSK models.

Upper extremity muscle deficits were modeled in three studies. Ultrasound images from stroke patients were used to update a generic arm musculoskeletal model of elbow extensors [51], indicating better compliance with measured external torques and the need for individualization of MSK modeling workflows. Such necessity was also outlined by Ashgari et al. [52] who used optimal control to distribute muscle weights to minimize experimental and predicted hand trajectory, thus exploring patient-specific neural control in hand movements. Last, MSK modeling was used to explore and quantify upper limb spasticity during passive planar elbow movements through inverse dynamics methods [53]. More specifically, Ang et al. predicted the angles in different angular velocities of elbow extension where spasticity sets in, that is, where abnormal muscle forces are elicited during motion. Thus, the angle at zero velocity was calculated and suggested as an objective biomarker of spasticity level.

To sum up, MSK modeling can serve as a means to objectively quantify muscle deficits and understand patient-specific motion strategy, which may enable clinicians to infer safer assumptions about the underlying neurological impairments. Recently, the Stroke Recovery and Rehabilitation Roundtable has strongly recommended that conventional biomechanical analysis should be incorporated in standard stroke motion evaluation [54], adding that kinematic and kinetic movement quantification is much needed. MSK modeling can build upon the availability of such data. The calculation of post-stroke muscle forces can help evaluate specific motion deficits and design treatments that focus on individual muscle force output.

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