INTRODUCTION
The ability to grasp and manipulate objects is essential for activities of daily living. Successful object manipulation requires control both of hand posture and of the force applied to the object, often simultaneously. Impairment of this motor control following stroke leads to hand disability. While isometric force or torque generation and velocity of hand movement have been studied independently in stroke survivors, the impact of the stroke on the dynamic interaction of these quantities, or force production during movement, remains relatively unexplored. Thus, the goal of this study was to compare the torque production in paretic and non-paretic hands during isokinetic tasks following stroke. We hypothesized that the paretic hand would produce lower torque when moving at high velocities. Additionally, we expected power deficits to be accentuated at higher velocities and to be greater in finger extension compared to finger flexion.

METHODS
The maximum flexion and extension torque produced by the MCP joint was measured in the paretic and non-paretic hand of ten moderately impaired (Stage 4 or 5) chronic stroke survivors (as measured on the Stage of Hand Section of the Chedoke-McMaster Stroke Assessment [1]). The subject’s wrist was fixed in a neutral posture with a fiberglass cast, which was clamped to a tabletop such that the palm was perpendicular to the table. The fingers were coupled to a servomotor (PMI Motion Technologies, Radford, VA) through a U-shaped shaft. Rotation of the shaft produced identical rotation of the MCP joints of the four fingers. Each trial consisted of a maximum isokinetic flexion (from 0° to 60° flexion) or isokinetic extension (from 60° to 0° flexion) movement. The servomotor controlled movement during each trial at one of four constant rotational velocities: 0°/s (isometric), 10°/s, 60°/s or 120°/s. The experimenter verbally encouraged subjects to exert maximum torque in opposing the imposed movement of the shaft throughout all trials. A total of 24 trials (3 trials x 2 directions x 3 velocities) were completed, with sufficient rest between trials to minimize fatigue. Position and torque were recorded from the optical encoder (PMI) and torque transducer (Transducer Tech, Temecula, CA) on the servomotor. EMG signals were recorded from the flexor digitorum superficialis (FDS) and extensor digitorum communis (EDC) muscles (Delsys Inc., Boston, MA).

Position, velocity and torque data was low-pass filtered at 20 Hz using a 30th order FIR filter. Mean torque recorded from the transducer during each trial was compared between the four movement velocities. Power, a measure of the force production at a given velocity, was calculated according to $P(t) = \tau(t) \cdot \omega(t)$ where $\tau$ represents torque and $\omega$ represents angular velocity at a given time, $t$. Power deficits were computed at each movement velocity as the percent decrease in power produced by the paretic hand compared to the non-paretic hand. EMG data was bandpass filtered (10-320 Hz), rectified and low pass filtered (10 Hz) to obtain an envelope. Muscle activation at each movement velocity was evaluated as a percentage of the maximum activity during isometric contractions. A repeated measures ANOVA was performed to explore the effects of velocity and direction on EMG, torque and power production ($\alpha = 0.05$, SPSS, Chicago, IL).

RESULTS AND DISCUSSION
As expected, significantly lower flexion and extension torques were produced with the paretic hand ($p<0.05$, Fig.1). During isometric contractions, the mean torque produced by the paretic hand was less during extension (1.69 Nm) compared to flexion (2.05 Nm). However, torque deficits, or the
amount of torque produced in the paretic hand compared to that produced by the non-paretic hand, were considerably less during extension (26% compared to a 41% deficit during flexion). For isokinetic contractions, though, relative deficits were greater for extension torque than flexion torque production (Fig. 1).

In fact, many subjects were unable to maintain extension torques throughout the duration of isokinetic movement trials, thereby resulting in a mean flexion torque during extension trials. Power deficits in general increased with greater rotational speed (Fig. 2). Extension deficits increased from 26% at the isometric contraction to the maximum value (100%) at 10°/s, and stayed at this level for higher speeds. Flexion deficits jumped from 41% at isometric to 57% at 10°/s and continued to rise to 76% at 120°/s.

Thus, strength testing of stroke survivors under isometric conditions may underestimate the level of impairment, as even at very low movement speeds torque deficits increase substantially. The mechanisms of this power impairment have not been fully elucidated. Co-contraction of agonist and antagonist muscles could contribute, but we actually observed a smaller ratio of antagonist to agonist activity in paretic muscles than in non-paretic muscles during both flexion and extension trials (Fig. 3).

An ultrasound study recently conducted in our laboratory suggests that muscle atrophy, while present, accounts for only a small percentage of these deficits. A shift toward type I (slow twitch) muscle fibers from type II (fast twitch) fibers would decrease the ability of stroke survivors to generate force, particularly at high movement velocities. Yet, some studies in this area suggest that there is actually a shift from type I to type II fibers [2]. Type II fibers, however, may be more susceptible to atrophy than type I fibers [3]. Additionally, an inability to voluntarily activate all existing muscle fiber or a decrease in the number of active motor units in hand muscle would also affect force production. Our data suggest further investigation into the underlying neuromuscular mechanisms behind isokinetic force production is warranted, especially in respect to extensor versus flexor muscles.

REFERENCES

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