SUMMARY
Discoordination following stroke imposes significant limitations in the performance of activities of daily living. We examined upper limb discoordination in severely impaired chronic stroke survivors by applying non-negative matrix factorization to EMG data collected from major shoulder and elbow muscles during generation of 3-D isometric forces at the hand. Specifically, we identified the minimum number of muscle synergies (muscular coordination patterns) that captured most of the total variance of EMGs across tasks and compared the composition of the synergies to those of healthy control subjects. In both groups, spatial patterns of muscle activation could be explained by task-dependent combinations of only a few (typically 4) muscle synergies. Broadly speaking, the four muscle synergies in the control group were composed of elbow flexors, elbow extensors, shoulder abductors/extensors and shoulder adductors/flexors, respectively. The composition of muscle synergies exhibited consistent alterations following stroke. Unlike control subjects, the anterior deltoid was coactivated with medial and posterior deltoids within the shoulder abductor/extensor synergy. In addition, the shoulder adductor/flexor synergy in stroke was dominated by activation of pectoralis major, with limited anterior deltoid activation. Our results suggest that an impaired control of the individual deltoid heads may contribute to post-stroke deficits in upper limb function.

INTRODUCTION
Previous studies in neurologically intact subjects have shown that motor coordination is accomplished by assembling flexible combinations of a few intermuscular coordination patterns, or “muscle synergies [1].” As defined here, a muscle synergy is a fixed pattern of activation across a set of muscles. The impact of stroke on the low dimensional control of upper limb muscle activation remains unclear. Recovery of upper limb function following hemiparetic stroke is characterized by the emergence of abnormal, stereotypical movement patterns that adversely impact the performance of activities of daily living [2]. Accordingly, we hypothesized that in more severely impaired stroke survivors, stroke induces muscular discoordination by altering the composition of muscle synergies available for motor control.

METHODS
We examined the spatial patterns of elbow and shoulder muscle activation in the paretic upper limb of five severely impaired stroke survivors (Fugl-Meyer assessment score < 26 [3]; age 67.6 ± 8.8 years, two females) with chronic hemiparesis (occurrence of stroke, 207.2 ± 65.0 months prior to the study) and in the dominant upper limb of six neurologically intact control subjects, as they performed a precisely controlled isometric force matching protocol at a single limb posture. Hand position and 3-D forces generated at the hand were recorded using the Multi-Axis Cartesian-based Arm Rehabilitation Machine (MACARM), a cable robot for upper limb research and rehabilitation (Figure 1) [4]. The end-effector for this robot incorporates a gimbaled handle mounted on a 6-degree of freedom JR3 load cell. Subjects generated voluntary forces (magnitude: 40% of their maximum lateral force) in 54 different directions presented in random order, uniformly distributed in 3-D force space, with their hand positioned directly in front of the shoulder at a distance of 60% of arm length.

Surface electromyograms (EMGs) were recorded from eight muscles of the upper extremity: brachioradialis (BRD); biceps brachii (BI); triceps brachii, lateral and long heads (TRIlat and TRIlong, respectively); deltoid, anterior, medial and posterior fibers (AD, MD, and PD, respectively); and pectoralis major (clavicular fibers; PECT).

We modeled EMG patterns collected from each ($EMG_{isometric}$) as linear combinations of a set of $N$ muscle synergies ($W_{isometric}$), each of which specified the balance of activation across 8 muscles. We applied non-negative
matrix factorization to identify the minimum number of muscle synergies that captured most of the total variance of $EMG_{\text{isometric}}$ [5]:

$$EMG_{\text{isometric}} = W_{\text{isometric}} \cdot C_{\text{isometric}}$$

where $W_{\text{isometric}}$ was a 8 by $N$ matrix containing the $N$ synergies (of unit magnitude) in each column and $C_{\text{isometric}}$ was a $N$ by 54 (number of trials) matrix, with each column containing the synergy activation coefficients for a specific trial. The number of muscle synergies for each subject was initially defined as the smallest number of synergies whose combination explained approximately 90% of the total variance for a given data set; see Figure 2 for an example.

RESULTS AND DISCUSSION

A set of about four synergies was sufficient to explain ~90% of total variance of EMG data collected in both groups (Figure 2). In controls, two of the four synergies consisted of relatively isolated activation of elbow flexors (brachioradialis and biceps; “E Flex”) and extensors (triceps long and lateral heads; “E ext”), respectively (Figure 3). Additionally, the analysis identified a “shoulder abductor/extensor” synergy (“S AB/E”) dominated by activation of medial and posterior deltoids, and a “shoulder adductor/flexor” synergy (“S AD/F”) involving activation of anterior deltoid and pectoralis major (clavicular fibers). In contrast, alterations were found in synergy composition in stroke (Figure 4). Anterior deltoid tended to be coactivated with medial and posterior deltoids (“Delt” in Figure 4), suggesting impaired control of the individual deltoid heads. In addition, the shoulder adductor/flexor synergy in stroke was dominated by activation of pectoralis major, with limited anterior deltoid activation.

REFERENCES