Sensorimotor Control of Grasping: Physiology and Pathophysiology

STROKE

Catherine E. Lang PT, PhD¹ and Marc H. Schieber MD, PhD²

¹Assistant Professor, Program in Physical Therapy, Program in Occupational Therapy, Department of Neurology, Washington University, St. Louis MO, USA
²Professor, Departments of Neurology, Neurobiology & Anatomy, and Physical Medicine & Rehabilitation, University of Rochester, Rochester NY, USA

Abstract

Stroke results in irreversible brain damage, with the type and severity of symptoms dependent upon the location and the amount of injured brain tissue. The most common neurological impairment caused by stroke is partial weakness, called paresis, reflecting a reduced general ability to voluntarily activate spinal motoneurons. In conjunction with the reduced ability to voluntarily activate spinal motoneurons, there is often a reduced ability to selectively activate the spinal motoneurons pools, i.e. turn on some neurons while no turning on others. Together, these mechanisms result in altered movement control of many muscles, especially the contralesional hand and arm muscles used for grasping. Because of the altered muscle control, a variety of kinematic and kinetic alterations are observed during grasping in people with paresis post stroke. Impairments in grasping are related to the inability to use the hand for functional activities during daily life. In rarer instances, stroke affects the posterior parietal lobe, resulting in distinct grasping deficits that are substantially different from grasping deficits seen after corticospinal system damage. Future studies investigating grasping post stroke could include the examination of both kinematic and kinetic aspects of grasping in the same subject samples, the examination of different types of grasping (e.g. palmar, precision), and the examination of different time points post stroke.

General information about stroke

Stroke is an acute, neurological event that is caused by an alteration in blood flow to the brain. The alteration in blood flow can be either a deprivation of blood to the brain tissue (ischemic stroke) or a spilling of blood (hemorrhagic stroke) onto the brain tissue. Ischemic strokes account for about 85% of all strokes. Stroke is a major health problem. More than 700,000 new strokes occur each year in the United States, and stroke is the leading cause of adult disability (Kelly-Hayes et al., 1998).

Stroke results in irreversible brain damage, with the type and severity of symptoms dependent upon the location and the amount of injured brain tissue. The most common neurological impairment caused by stroke is partial weakness, often called paresis, reflecting a reduced ability to activate spinal motoneurons voluntarily. Total paralysis, or plegia, is the most severe form of paresis, reflecting a complete inability to activate motoneurons voluntarily. Post stroke paresis or plegia most dramatically affects the side of the body contralateral to the damaged brain tissue. Typically, the entire contralateral half of the body is weak or paralyzed (hemiparesis or hemiplegia), although occasionally smaller strokes weaken only the contralateral arm or only the leg (monoparesis or monoplegia). Note that paresis can result from a wide range
of neurological diseases in addition to stroke, such as multiple sclerosis, cerebral palsy, amyotrophic lateral sclerosis, traumatic brain injury, and spinal cord injury. In all cases, the disease has produced a lesion that results in a decreased ability to voluntarily activate motoneurons in the anterior horn of the spinal cord.

Around 80% of people with stroke experience acute hemiparesis, resulting in a diminished ability to use their affected extremities for purposeful movement (Granger et al., 1988; Gray et al., 1990). Other impairments often accompany hemiparesis, such as hemianesthesia, hemianopsia, aphasia, and dysarthria (Granger et al., 1988; Han et al., 2002; Patel et al., 2000). Only about 40% of such patients achieve full recovery (Jorgensen et al., 1995; Reding and Potes, 1988; Wade and Hewer, 1987). The remaining 60% of stroke survivors have persistent motor and non-motor impairments that significantly disrupt their ability to participate in home and community life. Even for stroke survivors who are considered fully recovered, quality of life is decreased compared to stroke-free, community dwelling individuals, after controlling for age and co-morbidities (Lai et al., 2002). As the population of the world ages and the rate of obesity increases, more individuals are expected to have strokes and to live with the disabling consequences of stroke.

**Paresis post stroke**

Despite the fact that stroke often injures multiple brain areas, motor deficits due to stroke in humans can be reasonably predicted by the proportion of the corticospinal system that is damaged (Pineiro et al., 2000; Ward et al., 2006). The corticospinal system, i.e. the primary motor cortex, the non-primary motor cortical areas, and the corticospinal tract, is distributed in a proximal to distal gradient to the cervical spinal cord, such that the motoneuron pools of the distal upper extremity segments receive the greatest proportion of inputs (Clough et al., 1968; Dum and Strick, 1996; Dum and Strick, 2002; Fetz and Cheney, 1980; Palmer and Ashby, 1992; Porter and Lemon, 1993). Consistent with the disruption of this input gradient, there is a clinical perception that the severity of hemiparesis is greatest in the distal muscles and least in the proximal muscles of the upper extremity (Colebatch and Gandevia 1989). This clinical perception however, is not well-supported by systematic studies of hemiparetic severity across the upper extremity in larger patient populations (Bard and Hirschberg, 1965; Lang and Beebe, 2007). With respect to grasping, corticospinal system lesions disrupting corticofugal fibers within the posterior limb of the internal capsule result in chronic disabilities affecting dextrous movements, where the more posterior lesions (those most likely to damage fibers from the primary motor cortex) were strongly related to greater deficits in the kinematic and kinetic features of grasping (Wenzelburger et al., 2005).

Loss of corticospinal system input to spinal motoneuron pools is the neural mechanism that produces paresis post stroke (figure 1). Diminished corticospinal input causes a reduction in the number of motor units in the hemiparetic muscles that can be recruited voluntarily (McComas et al., 1973). During strong voluntary effort, fewer motor units are recruited, and the discharge rates of recruited motor units are slower than typically found in normal muscles (Frontera et al., 1997; Gemperline et al., 1995; Jakobsson et al., 1991; Jakobsson et al., 1992; Rosenfalck and Andreassen, 1980; Young and Mayer, 1982). This may account for the observation that the amount of time required to develop peak forces in hemiparetic muscles is prolonged (Canning et al., 1999). Furthermore, the ability to modulate motor unit discharge rates is impaired, such that the range of modulation and its variability are reduced (Dietz et al., 1986; Frontera et al., 1997; Gemperline et al., 1995; Rosenfalck and Andreassen, 1980).
Measurements of synchrony between motor unit pairs in hemiparetic hand muscles show either a narrowing or a broadening of the cross-correlogram peak (Farmer et al., 1993), indicating decreased overall corticospinal tract input to spinal motoneurons (narrowing), or the loss of focused input (broadening). Interestingly, the re-emergence of motor unit synchrony appears to be loosely related to the functional recovery of the hemiparetic limb.

At the muscle level, loss of corticospinal input and altered activation of motor units post stroke results in a decreased ability to recruit targeted (agonist) muscles for a given task (Fellows et al., 1994; Gowland et al., 1992; Hammond et al., 1988; Kamper and Rymer, 2001). Paretic muscles often produce more electromyographic activity (EMG activity) per unit force compared to the homologous muscle on the side unaffected by the stroke (Tang and Rymer, 1981). The decreased ability to recruit agonist muscles is frequently accompanied by co-activation of antagonist muscles (Bourbonnais and Vanden Noven, 1989; Dewald et al., 1995; Hammond et al., 1988; Kamper and Rymer, 2001; Sahrmann and Norton, 1977), although this is not always the case (Fellows et al., 1994; Gowland et al., 1992). Initiation and termination of muscle activity are delayed, i.e. the paretic muscles are slow to turn on and slow to turn off (Angel, 1975; Dietz and Berger, 1984; Sahrmann and Norton, 1977). Spatial and temporal patterns of muscle activation are disrupted across single joints (Bourbonnais et al., 1989; Canning et al., 2000), multiple joints (Dewald and Beer, 2001; Dewald et al., 1995), and even at joints not directly involved during a given motor activity (Boissy et al., 1997; Lang and Schieber, 2004).

Taken together, these findings suggest that the loss of input from the corticospinal system reduces the ability to selectively activate sets of muscles needed to perform skilled motor tasks (Lang and Schieber 2004). Understanding the underlying relationships between damage to the corticospinal system, motor unit activity, and muscle activity (illustrated schematically in figure 1) is critical for the interpretation of how stroke affects grasping.

**Kinematic and kinetic alterations in grasping post stroke**

The corticospinal system is the major neuroanatomical substrate for control and execution of hand and finger movements, such as grasping (for comprehensive reviews see Porter and Lemon, 1993; Wing et al., 1996). While grasping has been studied extensively in healthy, neurologically intact individuals, it is only beginning to be studied in people with stroke. Grasping movements in healthy controls are highly consistent and reproducible, both within and across individuals. This is not the case for grasping movements in people with hemiparesis post stroke. Grasping, like many other movements, is highly variable after stroke, both within subjects and across groups of subjects. Note that increased variability is a hallmark of movement in many diseases affecting the central nervous system, and is not unique to those individuals who have had strokes.

Within any given sample of subjects with hemiparesis, there will be those whose grasping movements appear normal or close to normal, and those whose movements are very far from normal or absent altogether. This means that generalizing group data to specific individuals should be done with caution, since one individual may be quite far from the group mean, or from other individuals within the group. A handful of studies now report on how grasping is altered and how it may recover in the affected, contralesional hand of people with hemiparesis post stroke. As in healthy subjects, hemiparetic grasping typically has been studied either with kinematic analyses during reach-to-grasp movements or with kinetic analyses during a precision grasp once the fingers are already touching the object. Below, we review the kinematic and kinetic data on hemiparetic grasping.
with respect to how it is similar to and/or different from grasping in healthy, neurologically-intact individuals.

Grasps often occur in the context of a reach-to-grasp movement in everyday life. In healthy individuals, the fingers open and close in a single smooth movement (Figure 2A) and the grasp component is temporally coupled with the reach component such that the fingers start opening as the hand starts to move toward the target object (Jeannerod, 1984). [Reference other chapters in this volume here.] In people with hemiparesis post stroke, the ability to open the fingers during a reach-to-grasp movement is altered because stroke impairs the ability to activate finger muscles and/or selectively activate them in the appropriate temporal patterns (see mechanisms outlined in figure 1). The diminished ability to activate the finger extensor muscles (Kamper and Rymer, 2001; Trombly and Quintana, 1983; Twitchell, 1951) and to coordinate activation of flexor with extensor muscles results in highly variable and inefficient patterns of finger opening (Lang et al., 2005). Figure 2B shows a typical example of finger opening during a reach-to-grasp movement in a person with hemiparesis 2 weeks after stroke. As the ability to extend the fingers improves over time, finger opening becomes smoother (figure 2C) but does not quite approach normal in this individual (Lang et al., 2006a).

The temporal coupling of the reach component and the grasp component during a reach-to-grasp movement is relatively preserved both early (Lang et al., 2005) and later (Michaelsen et al., 2004; Raghavan et al., 2006) after stroke. Those people with hemiparesis who do show alterations in the temporal coupling between the reach and the grasp components are unable to sufficiently activate the finger extensor muscles, resulting in several peaks in the aperture profile, where the largest of these several peaks may occur earlier or later than normal (Lang et al., 2005). As the ability to extend the fingers improves, peak aperture during reach-to-grasp movements becomes closer to normal, and time of peak aperture becomes less variable (Figure 3) (Lang et al., 2006a). In people with hemiparesis, hand shaping occurs gradually as the hand is decelerated during the later part of the reach, and not from the initiation of the reach, as happens in healthy individuals (Raghavan et al., 2006a). Normally, hand orientation at the end of the reach component is accomplished via rotational control of the upper extremity segments, but in people with hemiparesis, flexion and rotation at the trunk can assist in orienting the hand to grasp the object (Michaelsen et al., 2004).

Once the fingers are in contact with the object, people with hemiparesis post stroke have difficulty producing appropriate finger forces for object manipulation (Hermsdorfer et al., 2003). The ability to maintain low grasp forces (< 2N) accurately for short periods of time may be relatively preserved, but the ability to modify fingertip forces, i.e. rapidly grasp and release, is substantially slowed (Hermsdorfer et al., 2003; Mai, 1989). This again reflects the underlying mechanisms of hemiparetic movement control (figure 1), a decreased ability to recruit, modulate, and turn off motor units and muscle activity. Possibly as a compensatory strategy to prevent an object from slipping, people with hemiparesis often produce higher grip forces relative to load forces in the contralateral hand (Hermsdorfer et al., 2003), and in the ipsilesional hand as well (Quaney et al., 2005). Higher force ratios (grip:lift) however, are not a consistent finding across studies (Blennerhassett et al., 2006; McDonnell et al., 2006), but when present, have been attributed to altered somatosensory inputs or altered somatosensory processing (Hermsdorfer et al., 2003). Interestingly, enhanced somatosensory input can lead to better regulation of these forces (Aruin, 2005).

The ability to proactively and reactively adjust grip forces also is altered post stroke (Blennerhassett et al., 2006; Grichting et al., 2000; Raghavan et al., 2006b). This disability can
manifest as prolonged times to obtain peak forces, fluctuating force profiles, and/or disordered sequencing of grip and load force production. Such kinetic alterations post stroke may be attributed to altered motor unit activation, motor unit modulation, and selective muscle activation post stroke (figure 1). Some evidence, however, suggests that deficits in force production during grasping are not due solely to a motor execution problem. Raghavan and colleagues recently showed that anticipatory grip force control can be improved if the ipsilesional hand experiences the load prior to the contralesional hand (Raghavan et al., 2006b). This result implies that, in addition to an execution problem, some people with hemiparesis post stroke may have a higher-order motor planning deficit.

In neurologically-intact subjects, somatosensation is important for grasping. [Reference other chapters in this volume here.] In people with stroke, relationships between somatosensation and grasping are less straightforward. In our own hemiparetic subjects with and without somatosensory loss, the loss of somatosensation has not been shown to be related to the ability to grasp or the ability to use the hand for functional activities (Lang and Beebe, 2007; Lang et al., 2005; Lang et al., 2006a). Other literature that has more specifically examined somatosensory modalities needed for grasping has shown that, after controlling for motor deficits, the degree of somatosensory loss was related to 2-fingered grasp function in people with mild hemiparesis post stroke (Blennerhassett et al., 2007). And in cases of complete somatosensory loss with normal motor function, visual feedback can partially but not completely compensate for deficits in grasping (Jeannerod et al., 1984). Looking across these studies, we hypothesize that the importance of somatosensation for grasping is dependent upon the presence of adequate motor function. In other words, if motor abilities are diminished enough to substantially disrupt grasping behavior, then loss of somatosensation may not worsen the deficit. If motor deficits are relatively mild, however, then loss of somatosensation becomes an important factor impeding grasping and hand function.

Recovery of grasping

While much is known about general motor recovery post stroke, far less is known about recovery of grasping post stroke. Motor recovery post stroke is fastest in those individuals who are most mildly affected and slowest in those individuals who are most severely affected (Duncan et al., 2000; Jorgensen et al., 1995). The majority of motor recovery is achieved by the first 3 months post stroke, with only small changes in motor ability occurring after three months, usually in the more severely affected individuals (Duncan et al., 2000; Jorgensen et al., 1995). Functional recovery typically lags motor recovery by approximately 2 weeks (Jorgensen et al., 1995). This may be because it requires practice to discover and then incorporate newly emerging motor abilities into daily functional activities.

For the upper extremity, best possible function was achieved within 3 – 6 weeks post stroke for those with mild paresis (Nakayama et al., 1994). For those with severe upper extremity paresis, best possible function was achieved within 6 – 11 weeks (Nakayama et al., 1994). The severity of upper extremity paresis within the first few weeks after stroke is consistently the strongest predictor of eventual upper extremity motor ability and function (Kwakkel et al., 2003). In general, people who are more mildly affected initially are most likely to have full recovery and people who are more severely affected initially are most likely to have little or no recovery. Early observational data from Twitchell showed that about one third of people with motor
deficits after stroke will have complete upper extremity recovery, one third will have partial upper extremity recovery, and one third will have little-to-no recovery of function in the contralesional upper extremity (Twitchell, 1951). These observations are generally consistent with modern clinical experiences.

Less than a handful of studies have examined recovery of grasping after stroke. Palmar grasp and release movements have been used as outcome measures during a small trial of specific therapeutic exercises in patients an average of 6 weeks post stroke (Trombly et al., 1986), but grasping was rated by the number of movements made and not examined in a more quantitative or qualitative manner. Grasp movements have also been used in acute imaging studies to see how cortical activation changes after stroke (Staines et al., 2001). Unfortunately for the purpose of examining recovery of grasping, the parameters of movements made during scanning had to be experimentally controlled and thus the behavioral data do not permit the evaluation of changes in grasping during recovery after stroke. Kinematic changes in reach-to-grasp movements over the course of recovery were recently examined in a sample of people with mild-to-moderate hemiparesis post stroke (Lang et al., 2006a). Consistent with the time course of recovery of general upper extremity function, most of the recovery in grasping occurred within the first 3 months post stroke, with little change occurring from 3 to 12 months post stroke. Within the first few months after stroke, recovery of grasping was due to improvements in: the ability to extend the fingers, as measured by larger peak apertures; the ability to open the fingers faster, as measured by higher peak aperture rates; and the ability to efficiently close the fingers in a single smooth movement, as measured by lower aperture path ratios (Lang et al., 2006a). [Aperture path ratio is measure of how directly the thumb and index fingers close. It is calculated as the ratio of the length of the aperture curve actually traveled to an ideal straight line between the first peak of the aperture trace and the aperture at the end of movement. An aperture path ratio of 1 represents a single, smooth closing of the thumb and index fingers (ideal) and an aperture path ratio of >1 represents abnormal closing of the fingers, typically seen when subjects make multiple attempts to open and close the fingers as in figure 2B and 2C.]

Relationships between the ability to grasp and daily function

Grasping is an important movement for function, where hundreds or perhaps thousands of grasp and release movements are made throughout the day. People automatically adjust the type of grasp used and the amount of force applied based on an object size, weight, surface texture, and intended use [Reference other chapters in this volume here.] Thus, the ability to grasp in people with hemiparesis post stroke may be an important indicator of the functional use of the upper extremity.

A recent paper quantified the importance of grasping for upper extremity function post stroke (McDonnell et al., 2006). Seventeen subjects with subacute hemiparesis post stroke were studied performing a 2-finger grip-lift task and then tested on the Action Research Arm Test (ARAT), a well-established standardized clinical measure of upper extremity function. Of the eight kinetic grasp parameters measured, three parameters were correlated to upper extremity function as measured by the ARAT. Preload duration, i.e. the time between the onset of grip force and the onset of the upward lift force, was negatively correlated with ARAT scores (Spearman’s rho = -0.72), indicating that those subjects who took longer to establish grip forces had poorer upper extremity function. The maximum rate of grip force production was positively correlated with ARAT scores (rho = 0.83), indicating that those subjects who could develop grip force rapidly had better upper extremity function. And lastly, the strength of the temporal
relationship between the rate of grip force production and the rate of lift force production was positively correlated with ARAT scores (\(\rho = 0.83\)), indicating that those subjects who had more synchronous rates of grip and lift force production had better upper extremity function.

Another paper has looked at the relationships between grasping and upper extremity function in the early stages of stroke recovery (Lang et al., 2006b). Fifty subjects with hemiparesis were studied performing a reach-to-grasp task and then tested on the ARAT at three time points, an average of 10 days, 26 days, and 111 days after stroke. Within a few weeks, within the first month, and around 3 months after stroke, the kinematic parameters of grasping were correlated to upper extremity function as measured by the ARAT. The strength of the correlational relationships at each time point are illustrated in figure 4. Although there were some fluctuations in the strength of the relationships, faster (higher peak aperture rates) and more efficient (lower aperture path ratios) grasp performance and a greater ability to open the fingers wider (higher peak apertures) were associated with better function (higher ARAT scores). The strength of these correlations is moderate because the ARAT measures the broad construct of upper extremity function whereas the kinematic measures capture a specific parameter of one movement essential for UE function. In this same cohort, we found similar relationships between grasping and upper extremity function when function was measured with the Wolf Motor Function Test instead of the ARAT (Edwards et al., in review). Others have reported moderate to strong relationships between kinetic grasp parameters and grip strength (Hermsdorfer et al., 2003), often considered a surrogate measure of upper extremity function in the clinic.

Thus, available evidence indicates clearly that the ability to grasp is related to the ability to use the upper extremity for functional activities in people with hemiparesis post stroke. In many studies, however, just a few of the many tested grasp parameters are significantly correlated with function (Hermsdorfer et al., 2003; McDonnell et al., 2006; Raghavan et al., 2006b). Many measurable parameters of grasping may be uninformative in the post stroke population. Future studies of grasping in this population should focus on those grasp parameters that are consistently correlated with function.

**Unique grasping deficits when stroke damages only the posterior parietal lobe**

The above discussion has focused on deficits in grasping due to damage to the corticospinal system. In rare cases, a stroke can cause isolated damage to the posterior parietal lobe. The effects of these lesions on grasping are quite distinct, as illustrated by 2 cases of unilateral posterior parietal lobe damage (Jeannerod, 1986). When visual feedback is present during the movement, the reach component of reach-to-grasp movements on the contralesional side is relatively normal. When visual feedback is not present, then the reach component is characterized by slow and inaccurate movements that often overshoot the target. Unlike the reach component, the shaping of the hand during the grasp component is impaired regardless of whether visual feedback is present or absent. The impairments are most pronounced when visual feedback is absent. Instead of extending the fingers and then quickly closing them on the object, the fingers stay extended and do not shape into a formation that conforms to the shape of the object. Finger closure can be incomplete such that the object is either grasped inefficiently with an unintended portion of the hand or is not grasped at all (Jeannerod, 1986). Thus, grasping deficits after isolated posterior parietal lobe lesions tend to be the opposite of what is seen after corticospinal system lesions. Patients with posterior parietal lobe lesions tend to hold their fingers in extension and have difficulty flexing their fingers to match an object’s dimensions,
whereas patients with corticospinal system lesions tend to hold their fingers flexed and have difficulty extending them. The observed grasping deficits after isolated lesions to the posterior parietal lobe are consistent with the important role that this region plays in the visuomotor transformations required for grasping (for review see Andersen and Buneo, 2002; Galletti et al., 2003). [Reference other chapters in this volume here.]

Conclusions

Grasping deficits after stroke are generally a result of a lesion to the corticospinal system. Damage to this system disrupts the descending control of motor units, and consequently the voluntary activation of muscles on the contralesional side of the body. The varied kinematic and kinetic deficits observed after stroke are a consequence of these mechanisms. Future avenues of investigation regarding hemiparetic grasping include the examination of both kinematic and kinetic aspects of grasping in the same subject samples, the examination of different types of grasping (e.g. palmar, precision), and the examinations of different time points post stroke.
Figure 1. Schematic of relationships between stroke, damage to the corticospinal system (CSS), motor unit (MU) activity, muscle activity, and grasping behavior. Note that damage to other brain areas results in alterations in other domains besides the motor system and that movements other than grasp are affected by this same cascade.
Figure 2. Aperture (distance between the thumb and finger tips) traces for a healthy, neurologically-intact control subject (A), a subject with hemiparesis 2 weeks post stroke (B), and then again at 3 months post stroke (C). Time 0 indicates start of hand transport toward the target object. The end of the trace indicates the moment of contact with the target object. Note that the y-axis range in panel A is twice as large as the y-axis range in panels B and C. From Lang et al. 2006a.
Figure 3. Hemiparetic and control group means and SDs for peak aperture (A) and time to peak aperture (B) during a reach-to-grasp movement. Time points are within the first few weeks (acute), three months (day 90) and 1 year after stroke. Modified from Lang et al. 2006a.
Figure 4. Pearson correlation coefficients between kinematic measures of grasping and Action Research Arm Test scores. T1: 10 ± 5 days post stroke; T2: 26 ± 11 days post stroke; T3: 111 ± 21 days post stroke; Speed: peak aperture rate, higher is better; Grasp efficiency: aperture path ratio, lower is better; Peak aperture: higher is better. Modified from Lang et al. 2006b.
References


Clough JFM, Kernell D, Phillips CG. The distributions of monosynaptic excitation from the pyramidal tract and from primary spindle afferents to motoneurons of the baboon's hand and forearm. J Physiol 1968; 198: 145-166.


Dum RP, Strick PL. Motor areas in the frontal lobe of the primate. Physiol Behav 2002; 77: 677-82.


Edwards DF, Lang CE, Wagner JM, Birkenmeier R, Dromerick AW. Validation of the Wolf Motor Function Test in the acute stage of stroke recovery. in review.


