# The Dual-Strategy Hypothesis Whereby Motor Control Is Assessed From a Position of Quiet Stance

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국문요약

## Dual-Strategy Hypothesis 모델 과 보행 시작시의 동작분석 고찰

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박래준

본 연구의 목적은 다음과 같이 네가지이다. 첫째, dual-strategy hypothesis 모델의 이론적인 배경을 서술한다. 둘째, 보행시작시 (Gait Initiation) 와 장애물 보행시작 (Stepping over obstacles) 시의 motor task 를 dual-strategy hypothesis 모델의 관점에서 서술한다. 셋째, 파킨슨씨 환자군과 뇌졸증 환자군을 이 모델의 관점에서 서술힌다. 마지막으로, dual strategy hypothesis 모델의 임상적용 가능성에 대해서 간단히 서술하는 것이다.

핵심단어 : Speed-sensitive-strategy, Speed-insensitive strategy, 보행시작, 장애물 보행시작, 파킨슨씨 병, 뇌졸증

## Introduction

In our everyday life we perform many types of movements in conditions of changing external environment. Individual movements will be slightly different during performances, even if the same person tries to do the same thing a couple of times. Moreover, all people are unique (for example, different dimensions of body segments,

different experience, and different abilities to learn new tasks). In order to answer "how are movements controlled?" scientists frequently try to reduce the number of parameters and variables available to the subject in performing the task. This is commonly done by investigating muscle activation patterns around a single, upper extremity joint and the resultant forces or trajectories. Even though the results from single-joint studies cannot be directly applied to multiple movements, the results from this approach provide valuable frameworks for understanding general principles of motor control.

How movement is programmed to reach a target or obtain a given force level may be simplified in terms of those variables that are controlled and those that remain invariant (Brunt et al 2000, Ghez & Gordon 1987, Gordon & Ghez 1987a and b, Gottlieb et al 1989a, 1989b and 1990). For example, if an individual is simply asked to move from target A to target B (and it is correct when an individual moves to target B), the time it takes to make it is not important for completing this task. If, however, someone is required to hit a baseball in a game, it is very important to bring a bat to the right place at the right time for successful task. These two types of movements are controlled differently. The first kind of movement is referred to as "speed-insensitive (SI) strategy", in which the rate of rise of torque profiles remains invariant and diverges only as a function of duration of movement (Gottlieb et al 1989a). In contrast, the second kind of movement is termed as "speed-sensitive (SS) strategy" in which the rate of rise of torque profiles varies with the same speed of movement, but the duration of movements remains invariant (Cocos et al 1989a).

According to the dual-strategy hypothesis model, we can control movements by sending commands to the motoneuronal pools of the agonist and antagonist muscles that

define the EMG patterns of these muscles (Gottlieb et al 1989a) (see Figure 1 for motor control system).

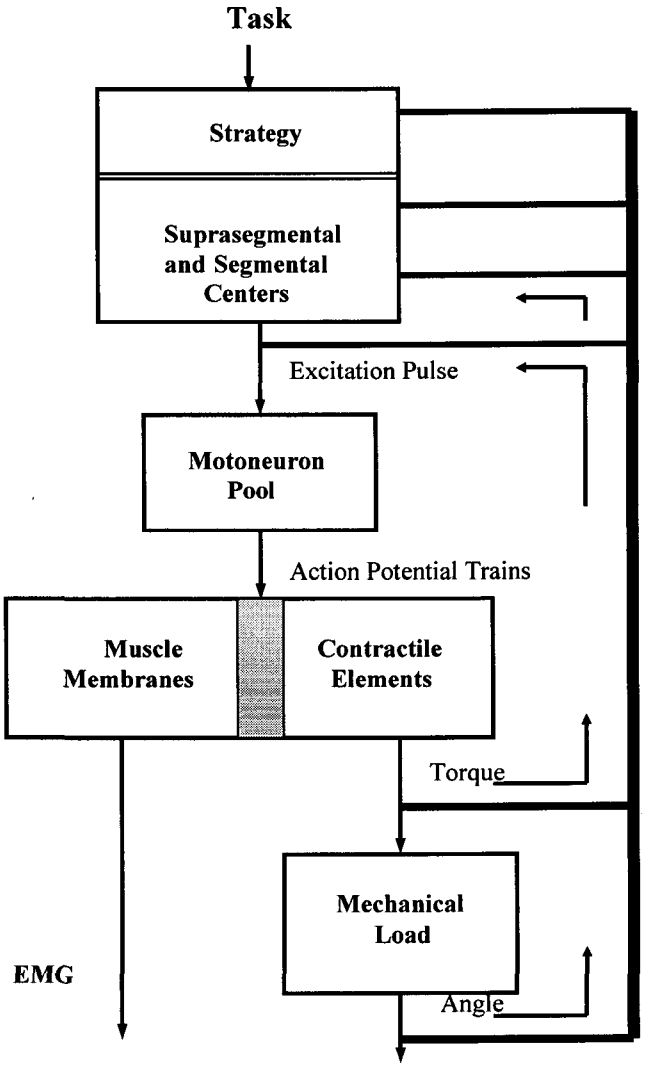



Figure 1. Selected feature of the motor control system. The figure illustrates how a movement task determines a motor control strategy that gives rise to an excitation pulse, which is an input to the motorneuron pool and produces EMG signals. The motoneuron pool is the final common pathway to the muscle on which the excitation pulse acts. It is the net convergence of descending excitation and inhibition from all sources. Two different kinds of peripherally observable phenomena are caused by action potentials produced by the excitation pulse, which can be

induced by input from both suprasegmental commands and feedback (for example, signals from peripheral receptors that induce reflex changes in the activity). The electric responses in the muscle membranes: forces and movements arise from mechanical responses of the contractile elements are all reflected in the EMG activity (Gottlieb et al 1989a).

In movements performed at the same speed (SI strategy in the first case), the duration of excitation to motoneuron pools is prolonged and the muscles are activated later for longer movement or movement made against larger loads (Corcos et al 1989a). Whereas, in case of movements performed at different required speed (SS strategy in the second case), movements are controlled by changing the intensity of activation to motoneuron pools rather than by changing the duration of activation (Gottlieb et al 1989a).

In SI strategy the initial excitation to the motoneuron pool is insensitive to the speed at which the movement is performed, distance or load (Gottlieb et al 1989a). Therefore, once the acceptable level of excitation to the motoneuron pools are selected, the motor system only changes the width of the agonist and antagonist pulses and the latencies of the early (LAGe) and late antagonist bursts (LAGl ) to meet different distances and load requirements (Gottlieb et al 1989a) (See Figure 2). In SS strategy, in contrast, the initial excitation to the motoneuron pool is proportionally selected to the speed at which the movement is performed, so this appropriately scales the initial rise in torque (Corcos et al 1989a). Therefore, the motor system modulates the amplitude of excitation pulses to the motoneuron pools and the latency of the antagonist burst (which is inversely proportional to speed or to peak initial torque) while the duration of movement remains invariant (Corcos et al 1989a) (See Figure 2).

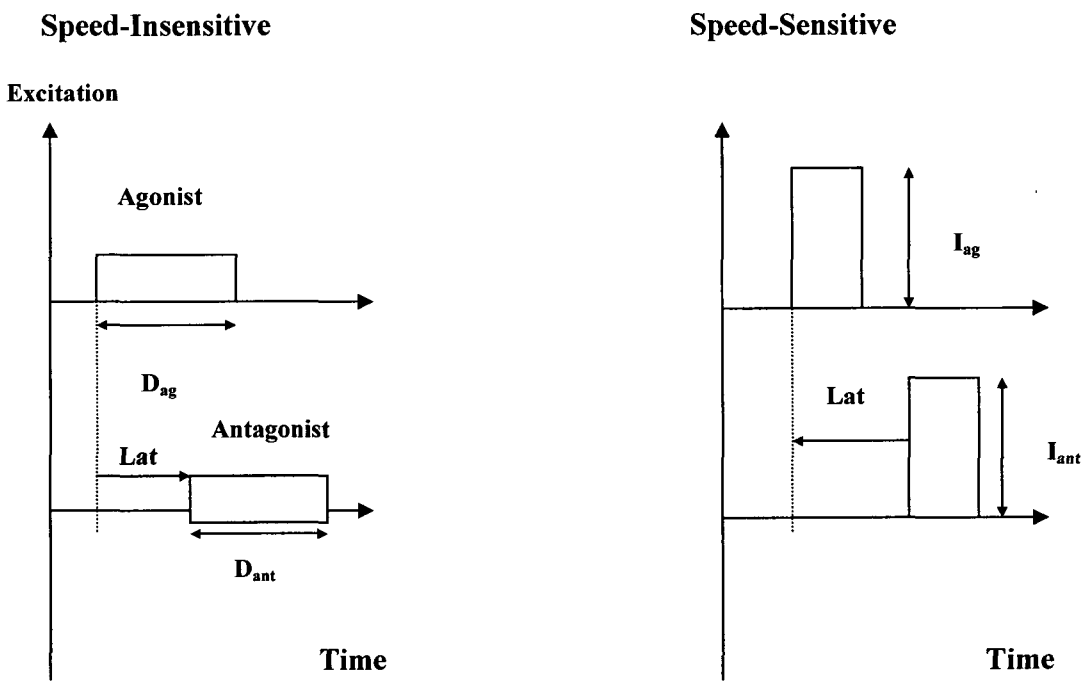



Figure 2. Schematic diagrams illustrating how speed-insensitive and speed-sensitive strategies may be operated by controlling (1) the duration ($D_{ag}$) or intensity ($I_{ag}$ ) of the excitation pulse to the agonist muscle. (2) the latency (Lat) of antagonist activation, and (3) the duration ($D_{ant}$) or intensity ($I_{ant}$) of the antagonist. (Gottlieb et al 1989a)

**Overview of the model incorporating gait initiation and stepping over obstacles**

In the above experiment, acceleration and force profiles vary according to the accuracy constraints of the task. That is, profiles changed at rates proportional to the target size (Corcos et al 1990b). Without any accuracy constraints acceleration and force profiles would rise along a common path (pulse-width control), but the duration of movement is controlled (Gottlieb et al 1990). The hypothesis for the single joint control of upper extremity (Corcos et al 1989a, Corcos et al 1990b, Ghez & Gordon 1987, Gordon & Ghez 1987a, Gordon & Ghez 1987b, Gottlieb et al 1989a, 1989b, 1990) and non-weight bearing lower extremity movements (Monohar et al 1998) has been thoroughly tested. Definitive evidence has yet to emerge that explains the degree to which principles of upper extremity control can be compared to volitional tasks from a position of upright stance. However, recent studies have provided preliminary data to suggest that gait initiation (GI) may be an appropriate task with which to make this comparison. The rationale for using this task is two-fold. First, as in the upper extremity studies, GI is motion about a single joint axis (at the ankle) where moments of force are generated that accelerates the center of mass like an inverted pendulum (Breniere & Do 1986, 1991, Breniere et al 1987, Brunt et al 1991, 1999, Elble et al 1994). Second, the center of pressure in the sagittal plane, or the location from where the ground reaction force vector originates, is controlled by the interaction of the antagonist muscles at the ankle (Breniere & Do 1991, Brunt et al 1991, Rogers & Pai 1991).

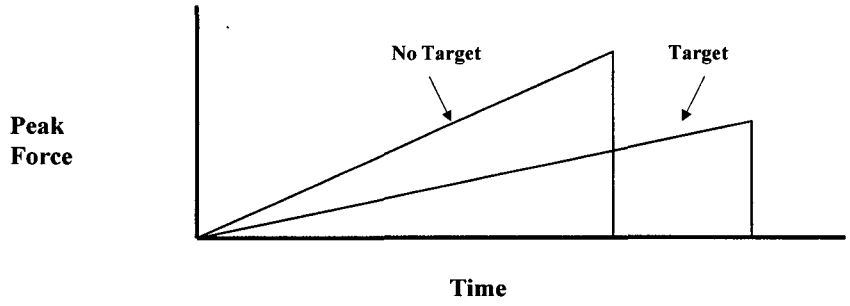
When subjects are asked to initiate gait and stepping one peak and the swing and the stance limb generate two distinct peaks of acceleration forces respectively (Brunt et al 1999, Brunt et al 2000). The first peak coincides with stance limb loading and

swing toe-off while the second peak approximately coincides with swing heel-strike and precedes stance toe-off. It has been shown that the slopes to these peaks can be modulated differently. For example, when stepping over an obstacle compared to GI the slope to the first peak remained invariant but the slope to the second peak increased for stepping (Brunt et al 1999). A similar result was noted when the accuracy of swing limb heel-strike was constrained during GI (Brunt et al 2000). That is, subjects slowed the velocity of the movement due to the demands of accuracy with a corresponding decrease in the second slope. The first slope, however, remained unchanged. It was thought that these results were not unlikely upper extremity experiments (Corcos et al 1989a, Corcos et al 1990b, Fitts 1954, Fitts & Peterson 1964, Freund & Budingen 1978, Ghez & Gordon 1987, Gordon & Ghez 1987a, 1987b) and non-weightbearing lower extremity movements (Gottlieb et al 1989a, 1989b) where the modulation of the slope of force has explained how individuals may program a given movement. That is, peak force could be reached if rate of force remained invariant but time to peak force was controlled. This seemed to be the case for the first slope of the ground reaction force of the stance limb. Alternatively, if time to peak force remains invariant then the correct peak force would be achieved by controlling the rate of rise of force, as was the case for the second slope of the ground reaction force of the stance limb.

Upper extremity experiments have shown that a subject's response may be determined by variables that constrain the velocity of movement. That is, variables such as accuracy or an explicit instruction of speed will affect the kinetics of the movement. That is not the case with other variables such as distance. It appears that the forces associated with stepping should be analogous to those of moving different distances in

the upper extremity experiments. With stepping, ground clearance is greater and swing time longer. That being the case then the invariance of the slope to the first peak Fx is in accordance with the upper extremity model. However, the finding that the slope to the first peak Fx of swing remained invariant is not consistent with the upper extremity model. It is because the target used for swing heel-strike in the previous study has been too large to dictate the velocity of movement (Brunt et al 2000). Recent study that used much smaller target size (50 % smaller than the one used in that study) found that the stance and swing peak Fx and the slopes to the peak Fx decreased with the target condition (Brunt et al Unpublished). In contrast, no differences between gait initiation and stepping were noted for the peaks and slopes of the swing and stance limb. Time to swing toe-off was significantly longer for the target condition and shorter for the stepping condition. The relative changes in time and slope to peak Fx of the swing and stance limb for the target condition do in fact suggest that subjects modulated the rate of rise of force and kept time to peak force relatively constant to achieve the desired initiation velocity. The relative changes in time and slopes of the swing and stance limb are shown in Figure 3.

a

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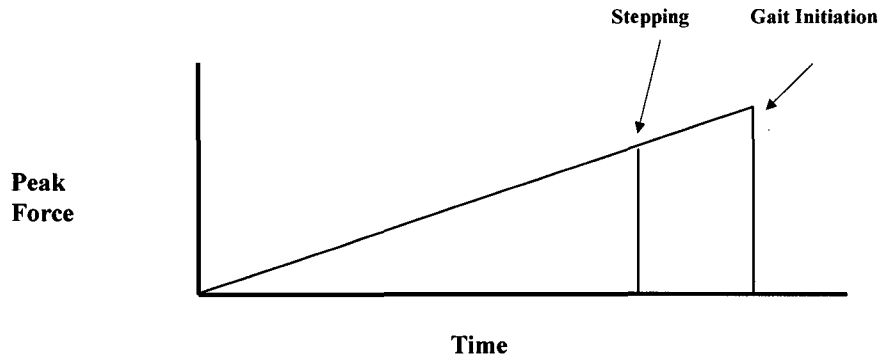


Figure 3. The relative changes in time and slopes of the swing and stance limb. For both the slopes of swing and stance limb forces (a) the target condition is greater than no target condition (b) no difference is found between gait initiation (GI) condition and stepping condition.

The velocity of GI and stepping decreased when accuracy constraints were placed on swing limb heel-strike. This decrease was a result of the modulation of both the stance and swing limb ground reaction forces prior to toe-off. The time to these peak forces remains relatively constant. This notion is supported by the strong relationship between the slope to swing limb Fx and time to swing heel-strike ($r = 0.84$) and a more modest relationship ($r=0.64$) for the slope to stance peak Fx. In addition, there were no differences in the slopes to either swing or stance peak Fx between GI and stepping. This concurs with the upper extremity literature where distance moved did not affect the rate

of rise of force. Based on the above data it does appear that GI and/or stepping may prove to be appropriate tasks by which to evaluate strategies of motor control and not just performance.

**Application of dual-strategy hypothesis to movement disorders**

Two types of movement disorders will be discussed in which the primary cause is a supraspinal deficit in the ability to control movement. Parkinson's disease (PD) will be discussed from the perspective of the model, but cerebrovascular accidents (CVA) in more general terms; it cannot be clearly identified within the framework of the above model because data from previous studies lacks for this application.

Parkinson's disease

Parkinson's disease (PD) patients have difficulty in initiating gait. When trying to voluntarily initiate the first step for walking, PD patients show hesitation and freezing. Although a few studies found gait initiation problems with PD patients, the difficulty that patients with PD patients is not well documented. The impairments associated with PD during GI include; increased postural sway due to the inability of PD patients to both inhibit postural soleus muscle and recruit sufficient synchronous tibialis anterior muscle (Crenna et al 1990, Gantchev et al 1996), the decrease in the lateral and posterior displacement of center of mass and reduced force and prolonged, and variable duration of postural phase and push-off phase (Burleigh-Jacobs et al 1997).

One deficit that can be applied to the above model includes an inability in PD to modulate the rate of rise of force and/or an inability to accelerate the ground reaction forces. That is, PD patients showed diminished vertical force production and reduced rate of rise of force (Burleigh-Jacobs et al 1997) due to the impairment to inhibit the

postural soleus muscle and recruit sufficient synchronous tibialis anterior muscle (Crenna et al 1990). Similar findings are found in the upper extremity studies. Godaux et al (1992) found that a prolonged movement time commonly observed in PD patients is due neither to inappropriate trajectories nor to difficulties with aiming at the target button, but to the depressed slope of development of muscle activity (deltoid, biceps, triceps and extensor indicis muscle). In addition, depression of the magnitude of the peak of EMG activity is not the reason for slowness of movement because the peaks of EMG activity were not less in PD patients than in healthy subjects (Godaux et al 1992). Similar findings were provided by Stelmach and Worringham' works (1988) and the work of Wing (1988). They found that major difference between PD patients and healthy subjects was not in the maximum levels of force that could be generated, but in the rate at which the force could be developed. PD patients are likely to follow the same trajectory even for different types of movements. It is because they have an impairment to modulate the rate at which different levels of force are generated in isometric task or an inability to accelerate at different rates in isotonic tasks. The healthy subjects showed an early divergence of the angle trace when healthy and PD subjects attempted movements over different distances, whereas PD subjects followed the same trajectory for both short and long movements (Hallett & Khoshbin 1980).

Cerebrovascular accidents

Following a central nervous system (CNS) lesion such as stroke, sensorimotor dysfunctions contralateral to the brain lesion occur in a large percentage of patients. Several types of movement impairments associated with stroke have been described. These include weakness, slowness, clumsiness, spasticity, and abnormal movement

synergies. Movement time during goal directed arm movements and gait could be prolonged up to four times that of healthy subjects (Knutsson & Richards 1979, Levin 1996). In the upper limb the disruption in the agonist and antagonist EMG activity compared to that of healthy subjects has been reported (they were prolonged) (Hammond et al 1988). Prolonged duration of muscle activity appears to be associated with a compensatory mechanism for generating efficient force to perform the task. It implies that the intensity of activation is less in patients with stroke than in healthy groups because these individuals do not have the ability to create excitation pulses. However, how individuals with stroke modulate duration of activation to accomplish movements over different distances and/or targets is not clear. Further research is required in order to interpret the data within the framework of the dual strategy hypothesis.

**Conclusions**

As explained earlier, the most frequently observed characteristics of movement deficiencies are the inability of patients to appropriately modulate the intensity duration, or latency of muscular activity. Others involved are slow reaction times, slow movement times, and increased variability in performance. Dual strategy hypothesis may be used to form a rational basis for designing appropriate treatment programs. It appears that GI and/or stepping may prove to be appropriate tasks by which to evaluate strategies of motor control. It also suggests that voluntary movement from upright stance may be a useful task when evaluating changes in performance of patients following therapeutic intervention.

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