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Gait Termination and Parkinsonism: A Review

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국문요약

파킨슨 환자의 보행종료

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보행 종료(gait termination)는 인간의 보행개시(gait initiation)나 율동성 보행(rhythmical walking)에 비해 관심을 적게 받고 있다. 파킨슨 환자들이 보행종료와 방향전환 시 가지는 어려움은 이들 환자들이 보행 시 넘어지게 되는 노출 요인으로 보고되어져 왔다. 보행종료에 대한 기전의 이해는 효과적으로 걸음을 멈추지 못해 넘어지게 되는 위험요인을 가진 사람들에게 적용되어질 수 있다. 이 논문에서 우리는 보행종료에 대한 최근문헌을 고찰하고 우리 실험실에서 진행되어온 파킨슨환자의 보행종료에 관한 일련의 실험들의 결과를 요약하였다. 본 연구는 율동적인 보행상태에서 완전한 멈춤으로의 전환에서 일어나는 동역학의 변화를 검사하기위해 시행되었다. 보행속도가 증가함에 따라 따르는 다리(trailing limb; 보행종료시점에서 뒤에 위치하는 다리)의 유의한 가속력 감소가 나타나고 멈출 때 이끄는 다리(leading limb; 보행종료시점에서 앞에 위치한 다리)에서 유의한 감속력의 증가가 나타난다. 보행종료 시, 이끄는 다리의 뒤쪽에 질량중심(center of mass)을 유지시키기 위해 하지 신전근의 활성도가 오래 지속되어져야 한다. 예측하지 못한 상황에서 보행종료 시, 보행 중이나 예측되어진 상황에서 멈출 때보다

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이끄는 다리 아래에서 감속력이 빠르게 발생하는 경향을 보였다.
대상자들을 보행속도에 따라 두 군으로 나눌 때, 계획되지 않은 보행종료를 하는 동안 보행속도가 느린 군이 빠른 군보다 전방 가속력을 급속하게 증가시키는 것으로 나타났다. 이러한 결과들은 보행속도가 느린 대상자들이 따르는 다리로 힘을 생성하기 보다는 주로 이끄는 다리에 의존하여 보행종료를 하는 것을 보여준다. 따르는 다리에서 발생되는 힘과 근육활성도의 조절을 측정하기 위하여, 파킨슨 환자와 연령에 의해 짝짓기된 정상 환자군에게 표적 조건(target condition)을 추가하였다. 파킨슨 환자군은 표적에 다리를 정확히 놓기 위해 몇 발자국 전부터 보행 속도를 줄였다. 파킨슨 환자들은 보행종료를 촉진하기 위해 필요한 근육들의 활성도를 증가시키는데 큰 어려움을 가지고 있다고 생각되어진다.

핵심단어: 근전도; 보행종료; 지면반발력; 파킨슨병.

Introduction

The mechanisms of stopping the forward progression of the center of mass are less studied than both the initiation of human gait and rhythmical walking. However, the question remains; why do we need to know about how we stop? Stopping is a transitional activity; that is, a transition from rhythmical gait to standing still. Falls often occur during transitional activities such as stopping or turning. Difficulty turning or stopping is a characteristic of elderly fallers (Tinnetti et al, 1986), stroke survivors (Kirker et al, 2000) and those with parkinsonism (PD) (Gray and Hildebrand, 2000; Morris and Iansek, 1997). Thus, understanding how one stops has implications for people who may be at risk of injuring themselves as a result of their inability to stop effectively. A number of factors may come into play when considering this topic. For example, frail elderly fallers have strength deficits in the hip and ankle musculature important during stopping. Balance, peripheral sensation, and reaction times must be considered. Another consideration is that stopping may be a motor program. As such, difficulty activating this program, for example, someone with PD, will also affect a person's ability to stop.

Human gait is a complex interaction of neural and mechanical processes whereby a body is moved in space from Point A to Point B. This process, of walking, is a rhythmic cycle of limb motions that propels the body in the desired direction while ultimately maintaining upright stability. In general, however, this simple sounding maneuver has several complicated stages. First, there is a transition from a steady state posture into rhythmic gait. The mechanics of the initiation process have been well studied (Breniere and Do, 1986,1991; Breniere et al, 1987; Brunt et al, 2000; Crenna and Frigo, 1991). Gait initiation involves inhibition of tonic postural activity, after which, there is an increase in the loading of the leg that is about to take the first step

(Brunt et al, 1991). Transfer of weight off this limb is necessary in order for it to leave the ground. Subsequently, two to three steps are required for the body to reach a steady state velocity (Breniere and Do, 1986; Wearing et al, 1999). Once there, an interaction between potential and kinetic energy occurs. The center of mass rises and falls in a sinusoidal fashion. Thus, while potential energy is greatest at the high point, kinetic energy peaks at the nadir of the path of the COM. This creates a situation whereby energy conserved as the potential energy is converted into kinetic energy as the COM "falls." The kinetic energy then "lifts" the COM back to the potential energy peak.

Gait termination in healthy adults

Stopping is the result of decelerating the forward motion of the body and finally coming to rest. During a planned stop, there occurs a volitional decision to stop at a certain position in space. This implies that not only is there enough time available to appropriately identify a suitable, satisfactory place to stop free of objects, obstacles, and other impediments, but also to determine how much braking force and how rapidly it is needed to be produced to slow the body and maintain dynamic balance.

Jian et al (1993) identified three phases of gait termination: preparatory brake, fast brake, and final brake. The goal of preparatory braking is described as the reduction of forward velocity and adjustment of posture for ensuing phases. During fast braking, a rapid reduction in kinetic energy occurs. In the final brake phase, the remainder of the forward velocity is reduced as the COP moves quickly ahead of the COM due to increased activity of the plantar flexors, and, finally, the COM is brought within the base of support. Hirokawa (1989) noted a decrease in forward velocity over the last three steps during stopping. Similarly, Wearing et al (1999) showed that subjects had already slowed by the second to last step.

An unplanned stop may be defined as, in a working manner, a termination of gait that occurs without prior planning. That is, an event occurs, that requires the rapid termination of forward gait. Perhaps more commonly, the occurrence has always been present (a pothole in the foot path) and it suddenly comes to our notice. Jaeger et al (1992) tested the ability of healthy adults to stop suddenly in response to an auditory tone. The predominant mechanisms identified to reduce forward velocity were a diminution of acceleration forces at push-off of the trailing leg and an increase in deceleration forces of the leading leg. These findings were later replicated by Jian et al (1993) and in our laboratory (Bishop et al, 2002; 2003). That is that there is an interaction between the leading and trailing limbs that occurs. This interaction becomes

more evident as the velocity of gait increases (Figure 1).

Jaeger et al (1992) found what they observed to be a critical periods during the stance phase of gait. That is, as long as the signal stop occurred before 18% of stance time, subjects were able to stop on the next step. After this, the time to stop increased, as subjects had to take an extra step. Based on the timing of the signal, three mechanisms have been identified to stop the forward motion of the body (Hase and Stein, 1998). First, the subject could increase the extension of the leading limb, thereby keeping the COM behind the lead foot. This was achieved predominantly by strong activation of the soleus and activation of the quadricep and gluteus medius muscles. Second, there was a reduction in push-off from the stance limb (soon to become a swing limb). A large burst of activity from the tibialis anterior muscle reduces the effect and output from the ankle plantar flexors. In this instance, gluteus medius and hamstrings slow and limit hip flexion, in effect holding the swing leg back. Hase and Stein (1998) describe a third scenario that occurs when the first two mechanisms described are ineffective in controlling the motion of the center of mass. If the signal to stop occurred after midstance there was insufficient time for subjects to reduce push-off power and ready the swinging limb to maintain extension. In this case, subjects either rose onto their toes to convert kinetic to potential energy, or took another step. This process

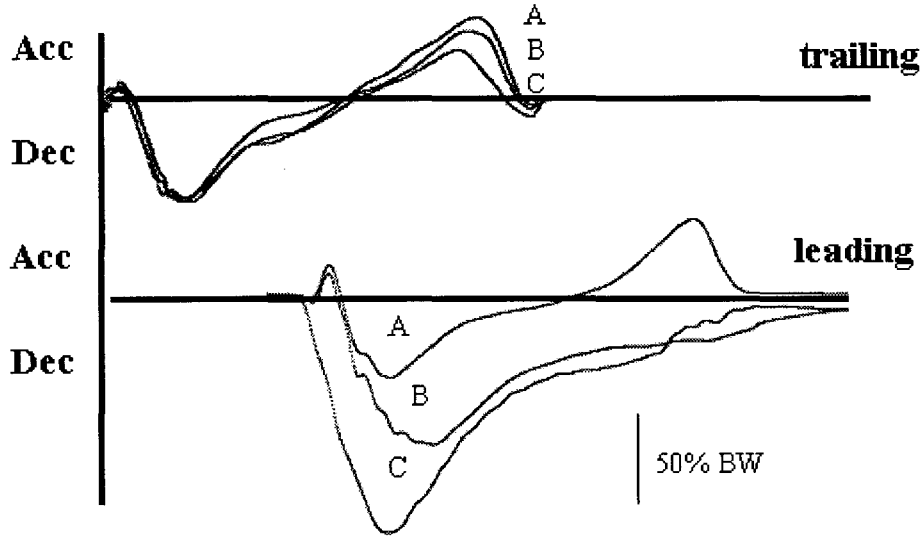

Figure 1. The interaction between lead and training limbs during planned stopping
This figure represent anterior-posterior ground reaction force changes.
A:100%, B:125%, C:150% of normal cadence

was summarized by Crenna et al (2001) as distal to proximal activation in the lead limb, and proximal to distal activation in the trailing limb.

The study performed by Hase and Stein (1998) did not place velocity constraints upon the subjects. Presumably, if subjects were to walk more quickly the signal to stop would have to occur earlier in the gait cycle. This time between signal and event has been labeled 'available reaction time' and has predominantly been used to in studies of obstacle clearance and turning. We have confirmed that as the velocity of gait increases, the timing of the signal to stop must come earlier within stance phase and the required available reaction time or (warning time) increases.

There is evidence that cutaneous input is involved in reflexes that modify swing limb trajectory during stumbling (Zehr, 1998). Zehr et al (1998) suggest that sural nerve input assists in stabilizing the human gait against unexpected perturbations in stance and swing phase. Given that the lateral and inferior surfaces of the foot are the initial areas of contact in the stance phase of gait, sensory input from this area is very important in the regulation of the forces required during walking, and even more so during stopping.

When forward gait was studied in patients with diabetic neuropathy, Dingwell et al (1999) showed an increase in variability of gait parameters such as stride length and width of base of support and implied that this increased variability may indicate an instability in those with diabetic neuropathy. Other studies have examined the incidence of falls and injury in individuals with sensory deficits in the plantar aspect of the foot (Lord et al, 1994; van Deursen et al, 1999). A decrement in plantar sensation correlates with an increased risk of falls and injury.

Perry et al (2000) demonstrated that cutaneous sensation from the foot was essential during recovery from perturbation. Subjects in this study showed a decrease in sensory and vibratory sensitivity with no concomitant loss in strength or proprioception, after exposure to an ice bath. When presented with an unstable surface, subjects showed a marked increase in the timing of stepping reactions and had greatest difficulty with backward translation. What is more important to note, is that during the test condition, subjects required several extra steps to recover their balance. The absence of feedback resulted in extra steps and increased loading of the stepping limb after ground contact perhaps due to decreased load-sensing ability. Our work in experimentally induced neuropathy, demonstrates that subjects alter characteristics of rhythmic gait (Fiolkowski et al, 2002) demonstrating increased variability in the temporal components of gait, and report difficulty turning (Fiolkowski, 2000).

Gait termination in parkinsonism

We have studied gait termination by those with PD under both planned and unplanned conditions. The series of experiments to be reviewed have common nomenclature, experimental design and data analysis, which will be summarized here. Note that we also include the data from trials in which subjects had to stop to a target.

Subjects

Patients with any concomitant neuromuscular or musculoskeletal disabilities that affect their ability to participate in this study were excluded. All patients were tested in the "on" medication state approximately 1.5 hours following the administration of their medication. Age (2 years) and gender-matched control subjects were recruited from community dwelling residents in Florida, USA. All subjects read and signed an informed consent form approved by the University Institutional Review Board.

Equipment

Surface electrodes were applied to the muscle bellies of the tibialis anterior (TA), soleus (S), and gluteus medius (GM) of both lower extremities. Recording electrodes consisted of two silver-silver chloride 1 cm diameter electrodes embedded in an epoxy-mounted preamplifier system (x35) whose centers were spaced 2 cm apart. A reference electrode was attached to the medial aspect of the tibia. The EMG signals were band-pass filtered (20 Hz to 4 kHz) (Therapeutics Unlimited, Iowa City, Iowa), and full-wave rectified and low-pass filtered (350 Hz) on-line. Two force platforms, embedded in a level walkway (10 m in length and 1.22 m in width), measured ground reaction forces. Processed EMG[1)] and amplified force platform signals were sampled on-line at a rate of 1,000 Hz. A reflective marker was placed on a sacral wand to estimate forward velocity of the center of mass using the MacReflex motion tracking system[2)].

Procedures

A walkway was arranged to position force plates such that both feet hit the center of a force plate during normal walking and during gait termination (Figure 2A). The position from which each subject started on the walkway was determined prior to data collection.

There were two conditions: planned stopping and unplanned stopping. First, the subject walked the entire length of the walk way five times. Kinetic and kinematic data from these trials were used for norm BIOPAC Systems, Goleta, CAalization and data reduction. For planned stopping, there were five trials in which the subject was instructed to stop on the force plates (Figure 2B). There

1) BIOPAC Systems, Goleta, CA
2) Qualisys Inc. Glastonbury, CT

were five additional trials where the subject was asked to strike a 10 cm diameter target taped in the center of force plate two with the heel of the leading limb when stopping.

For unplanned stopping, the subjects started, as during the walking condition, however, an unexpected light signal cued them to stop (see D in Figure 2C). To discourage any planned strategy to stop on the force plates the light signal occurred during random trials and at different locations on the walkway. Onset of the light occurred either during single limb stance on force plate 1 or immediately before or at heel strike of the next step on force plate 2. Timing of this signal was accomplished using an infrared switch placed near the start of the walkway to trigger a time delay relay attached

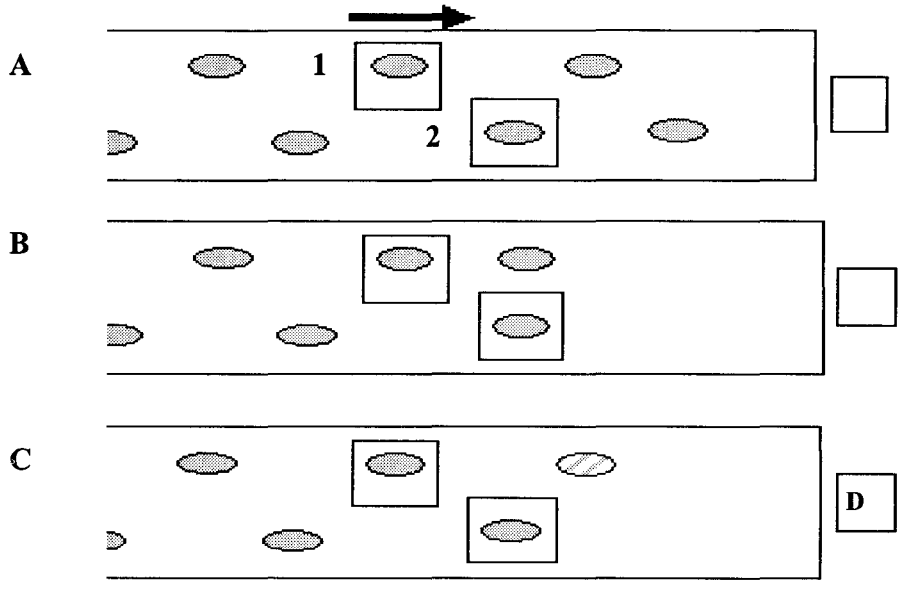

Figure 2. Foot placement and force data during walking, stopping trials
The first three figures (A, B, C) represent the foot locations during walking, planned stopping, and unplanned stopping, respectively. The arrow indicates the direction of travel. 1=first force plate. During stopping trials, foot strikes at this location were considered trailing limb. 2=second force plate. Foot strikes at this location were considered lead limb. For unplanned stopping trials, the light signal (D) occurred before, at, or after the force plates to minimize targeting. Foot placements shown in C are to assist with nomenclature only. The mottled foot strike shows the approximate location of foot placement if subjects were to take an extra step during stopping.

to the light. Only trials where the trailing stance limb landed on the force plate were used in data analysis.

The limb in stance when the light signal was activated was considered the trailing limb. The limb in swing as the light was activated was considered the lead limb. Following the light signal, trials in which the subject was able to stop within the next foot-strike of the swing limb were termed "next step" (NS) trials. If the subject had required an additional step, and finished with the trailing limb at shaded position shown in Figure 1C, the trial was considered an "extra step" (ES) stopping trial.

Data reduction

Subjects with PD were grouped according to free walking velocity. Those with an average free walking velocity less than 1.3 m/s were considered "slower" walkers. The remainder of the subjects with PD walked at 1.5 m/s or greater. Gait velocity was determined via kinematic data of a sacral marker. Raw velocity data was low-pass filtered at 10 Hz. Velocity during stopping trials was also normalized to each subject's overall mean COM velocity during walking to allow between group comparisons. This normalization method provided a comparison that allowed us to consider relative change in velocity made by subjects as the slowed and then stopped.

The EMG dependent variables were the timing of muscle activity onset and offset, and the amplitude of muscle activity relative to heel strike during walking and planned stopping. The level of muscular activity was determined by normalizing integrated EMG (iEMG) during stopping to that during walking. The stance phase of walking was divided into phases based on the vertical ground reaction force (Fz). These phases were operationally defined as being from initial contact to peak loading (Pk1), peak loading to midstance (MS), midstance to second peak loading (Pk2), and second peak loading to toe-off (PO). The amplitude of EMG of the stopping trials was then expressed as a percentage of the iEMG during walking trials for each of these four phases.

The onset and offset of muscle activity was determined as the point at which EMG activity rose or fell two standard deviations from the mean of back ground EMG activity at rest for 30 ms. Timing data were normalized to the time to the first peak loading calculated from Fz during walking. These normalized data were used to control for the velocity differences between groups. After analysis of EMG amplitude, the EMG data were low-pass filtered at 40 Hz to be used for illustrative purposes.

Force plate measures included peak deceleration and acceleration (Fx), and the average slope of the deceleration curve from both limbs. Examples of raw force data may be seen in Figure 1. Analysis of variance (ANOVA) techniques with repeated measures were used to detect dif-

ferences between the groups and among conditions with Bonferroni post-hoc tests for differences in means. Any associations between age, UPDRS, and H&Y scores were determined by nonparametric statistical method (Kendal, Freidman, Mann-Whitney). Type 1 error was maintained at 5%.

Planned stopping

During planned stopping, older adults perform the predominant amount of COM deceleration during the final step of the stopping task regardless of the presence of the target. Decreasing SOL activation and concurrently increasing activation and duration of the TA of the trailing limb, thereby decreasing the push-off impulse and acceleration, accomplished this. When attempting to strike the target, the TA activity increased in conjunction with GM. This GM activity is most likely related to

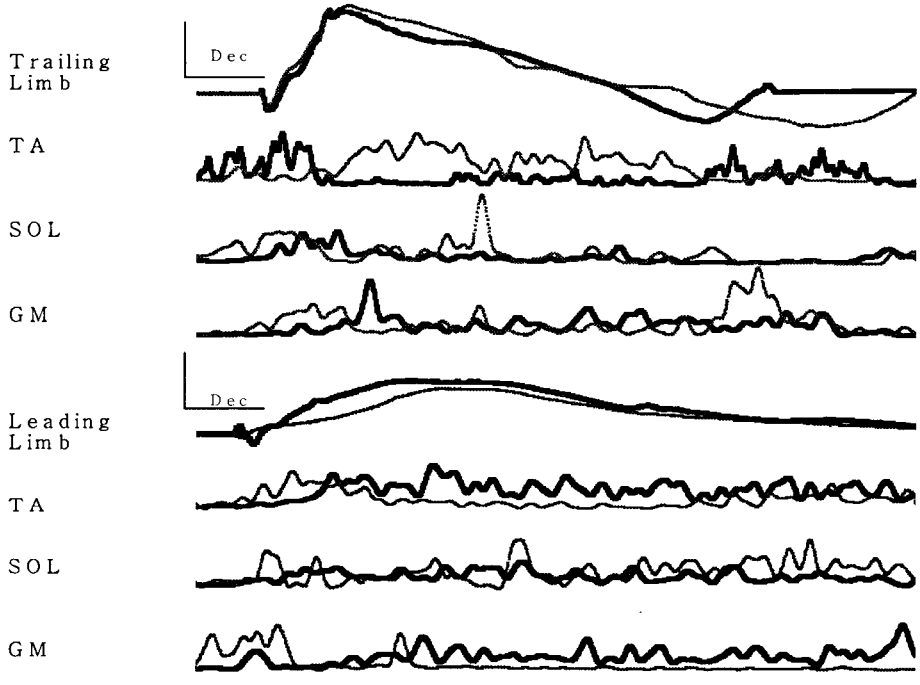

Figure 3. Raw data from a single subject for single trials during planned stopping with (thin lines) and without a target (dark lines)
TA: tibialis anterior, SOL: soleus, GM: gluteus medius.
Vertical bar for trailing limb represents 30%BW and .1 mV.
Vertical bar for lead limb is 60% BW and .1 mV.
Horizontal bars show 125 ms. Vertical arrow over the top tracing indicates the timing of the lead limb ground contact.

slowing the pelvis and prolonging single leg stance while the subject ensures a direct hit on the target. All subjects showed similar patterns of muscle activity during planned stopping. Raw data from one subject in the PD group shown in Figure 3. Note the decrease in peak lead limb forces, and the reduction in acceleration force under the trailing limb. Additionally the changes in EMG activity can be seen in the figure.

However, the addition of the target to the task resulted in the slower walking subjects with PD further slowing their overall approach to the target area. It is known that there is a visual impact on the gait of those with PD. Objects on the floor or doorways can limit the gait of a person with PD to the point of causing "freezing" of gait. As slower subjects with PD slowed COM velocity early, they were not required to decelerate as rapidly within the final step. In contrast to the control group, slower subjects with PD increased SOL activation during the final step, probably controlling forward advancement of the leg over the foot. Lead limb activity of TA appears to be related to the accuracy of foot placement as all groups increase TA activity possibly to make sure that the target was struck by the subject's heel and not the mid-foot.

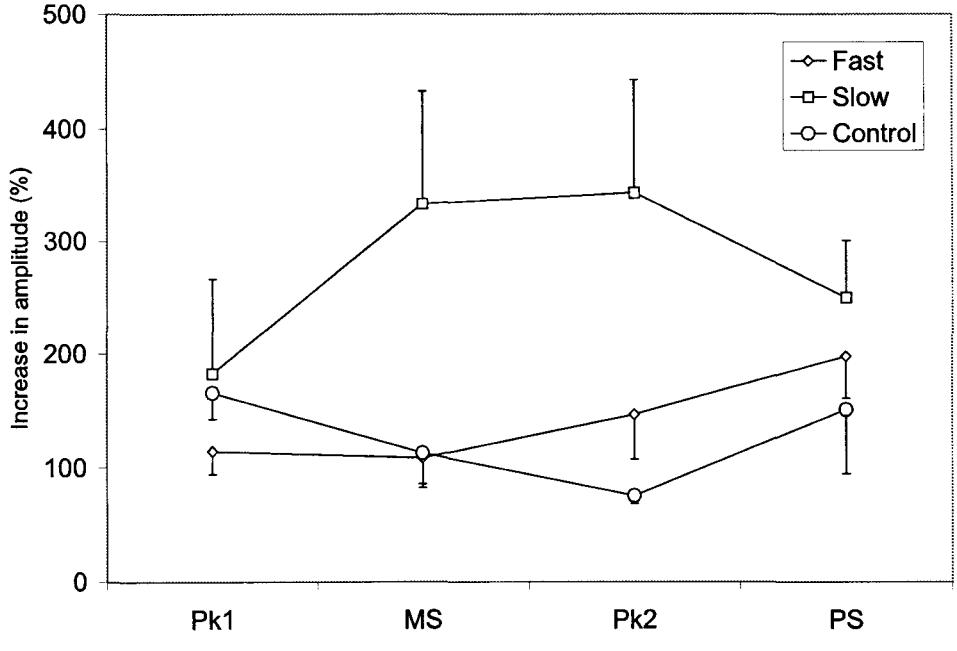

Figure 4. Changes in soleus iEMG amplitude during planned stopping trials
Pk1: initial contact to peak loading, MS: peak loading to mid-stance
Pk2: mid-stance to second peak load, PS: second peak load to toe-off

There is moderate evidence from the results of our studies that slow walking subjects with PD modulate force and EMG amplitude differently to faster walking subjects. Amplitude changes were shown in SOL. The slow group increased activation of SOL throughout stance phase of the trailing limb during planned stopping while the fast group was not significantly different from the matched control group (Figure 4). The concurrent increase in TA amplitude during the latter part of the trailing limb stance phase resulted in the slower walking group co-contracting around the ankle.

Particularly noteworthy were the alternating bursts of TA and SOL activity demonstrated by a significant proportion of the slow group (Figure 5). The alternating muscle activity may have contributed to the co-contraction strategy demonstrated by the subjects in the slower group by increasing the average amplitude through each subphase of stance. These alternating bursts are similar to those noted by others during postural adjustments prior to arm motion and gait initiation. A common mechanism to initiate gait is to inhibit SOL activity and pull the center of pressure behind the

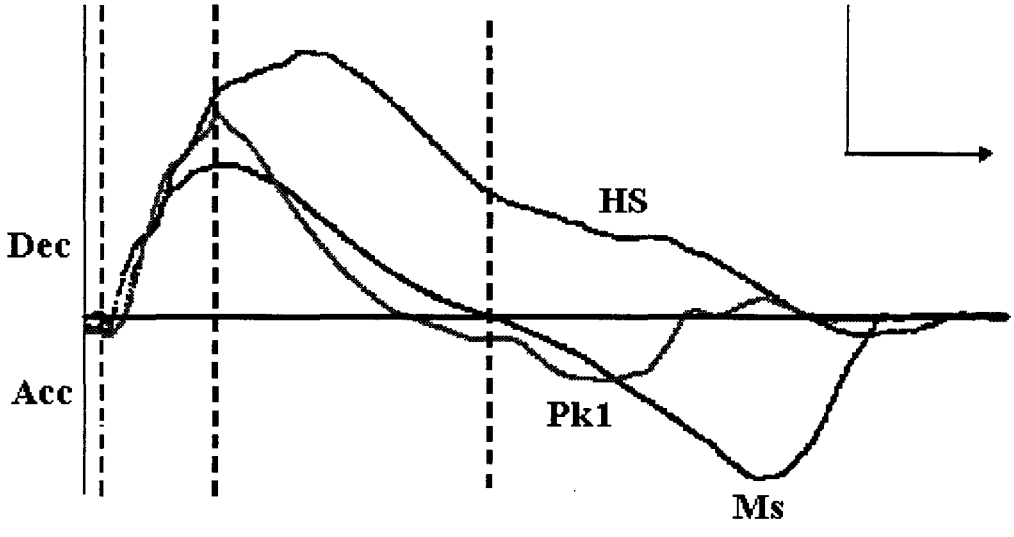

Figure 5. Mean anterior-posterior force graphs for all trials in which the signal to stop occurred at heel-strike (HS), first peak loading (Pk1), and midstance (MS)
The vertical dashed lines indicate the approximate timing of the light signal.
The vertical bar represents 40% body weight, the arrow 100 ms.
For the HS trial, note the increase in area above the zero line.
This is indicates an increase in the braking impulse.

center of mass using TA contraction or gravity. During gait initiation, it is hypothesized that the alternating TA and SOL activity arises from an inability to activate or superimpose the central program to destabilize the subject's posture. Perhaps the bursting of activity, noted in the subjects in this current study, reflects an inability to superimpose a stopping program onto that of walking.

No group differences were noted for the rate at which forces were generated although the target resulted in all subjects loading the lead limb more slowly during planned stopping. A methodological limitation that may contribute to this is related to the normalization process whereby forces during stopping were normalized to those generated during walking. If a subject walked slower during stopping trials, the deceleration values would be lower because of the decreased velocity.

Unplanned Stopping

For our first set of experiments, we had initially hypothesized that the more 'involved' the PD, as determined by a subject's score on the Hoehn and Yahr

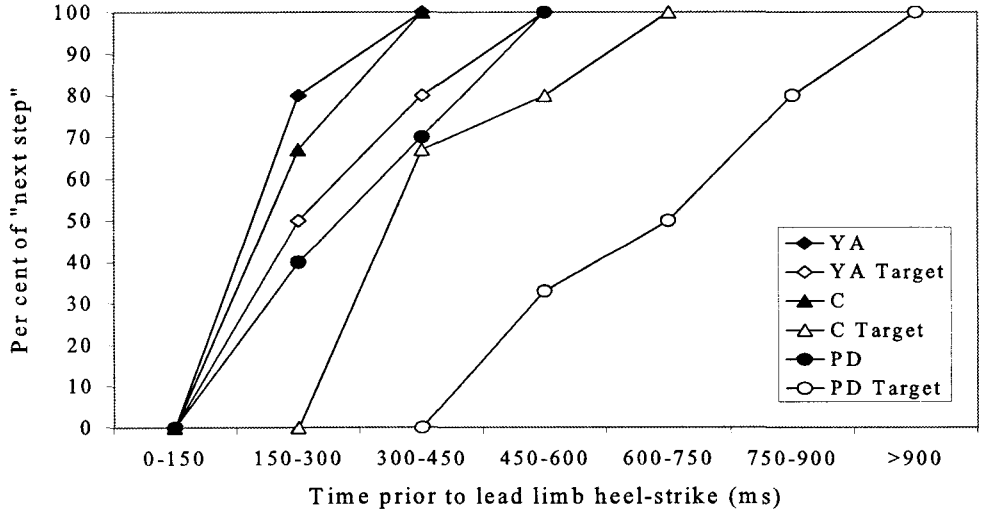

Figure 6. The percentage of all unplanned stopping trials that were next step (NS) trials for different amounts of warning time

Time represents the time before heel-strike of the leading limb.

YA: young adult, C: control, PD: parkinsonism, NS: subject was able to stop in next step, ES: subject required extra steps, T: target trial, NT: no target. When faced with a target the parkinsonism group required more than 900 ms to be able to achieve a 100% rate of NS trials. This indicates that it is imperative that those with PD be instructed in environmental scanning procedures.

scale (H&Y), the more difficulty they would face in stopping. Additionally, we had expected, based on our work in healthy adults, that the leading limb would be of the greatest significance. However, there were no differences noted for any of the dependent variables, among groups based on the H&Y score. A second analysis was performed based on functional characteristics. In this case, the speed at which subjects walked was used to form two groups: fast and slowly walking. Under this analysis, a most intriguing and unexpected result was that during unplanned stopping subjects in the slow group generated deceleration force at a significantly faster rate than the fast group. That is, although they walked more slowly, forces were generated very rapidly indicating an increase in limb stiffness. We hypothesized that these subjects had been less able to modulate force under the trailing limb and were totally reliant on the leading limb to stop. That is, slower walking subjects were unable to alter the stopping strategy that they used, decreasing the degrees of freedom in planning. This was confirmed when reviewing the video data for trials in which subjects stopped to a target; that is, subjects tended to stop on the same leg independent of when the signal to stop occurred.

Overall, the EMG amplitude changes during unplanned stopping are similar to those during unplanned stopping except in magnitude. We had initially hypothesized that subjects with PD would not be able to modulate EMG amplitude to the same extent as their matched peers. Differences did exist in the use of the lead limb. Subjects in the slower walking group increased activation of the GM and SOL of the lead limb during unplanned stopping, presumably increasing lead limb stiffness. Matched subjects tend to increase SOL activity only. What we have not yet assessed is the rate of activation of the muscles. Goudaux et al (1992) described upper extremity experiments in which subjects with PD achieved the same maximal muscle activation but the rate of production was reduced when compared to those without PD. In our studies, the PD groups produce the same relative changes in iEMG amplitude as controls but may take longer to reach the amplitude levels.

The timing of the signal to stop also differentially affects those with PD. We have demonstrated that the braking impulse generated under the trailing limb is modulated differently by those PD. Figure 5 contains mean data from control subjects and those with PD and demonstrates that the control subjects are better able to modify braking impulse in response to a signal that occurs later in stance phase (Bishop et al, 2003). Those with PD are not able to modulate the trailing limb as effectively as control subjects. This is in complete agreement with our earlier work indicating that those PD rely predominantly on the leading limb.

When confronted with a target task the warning time needed to stop for those with PD dramatically increases to the point that subjects with PD require the signal to stop to occur two steps prior to the target. Figure 6 shows the dramatic increase in warning time required by those in the PD group to effect 100% NS trials. When this data is converted to represent stance times (Figure 7), it becomes obvious that the increased time necessitates the signal to stop occur prior to heel-strike. We believe these data are consistent with both our own findings of preferred stopping strategy and changes in force/impulse generation, in addition to those studies showing slower rates of muscle activation and longer reaction times.

In summary, stopping requires a simul-

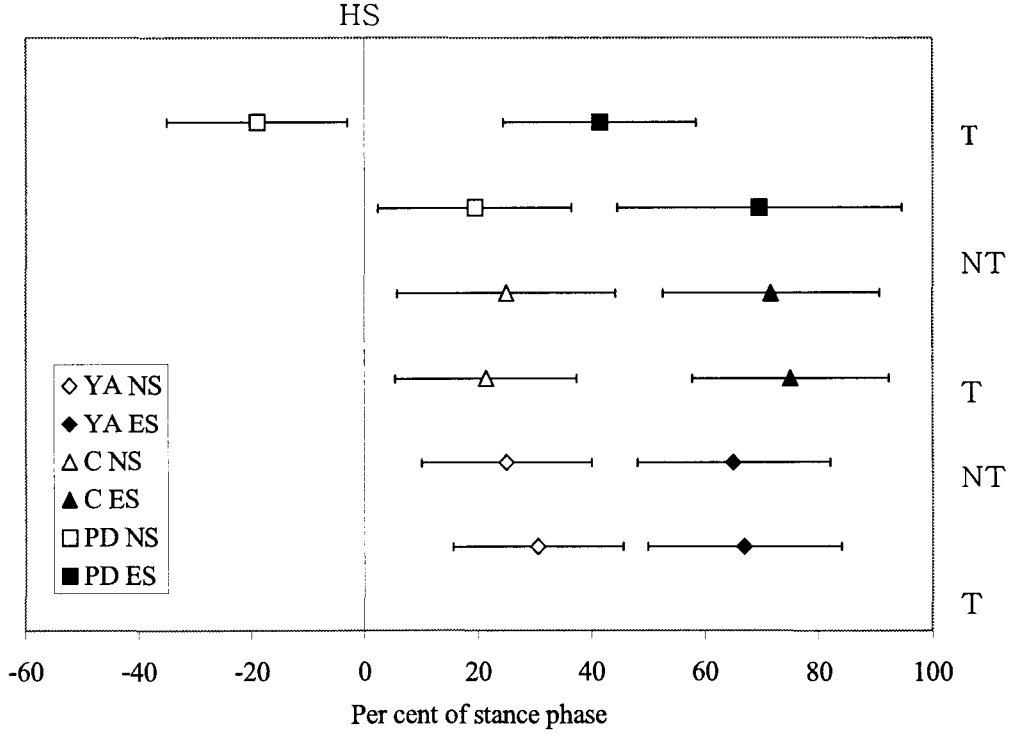

Figure 7. Relative timing of the signal to stop relative stance phase
HS: heel-strike, YA: young adult, C: control, PD: Parkinsonism, NS: subject was able to stop in next step, ES: subject required extra steps, T: target trial, NT: no target
Note that when confronted with a target, those with PD required that the signal to stop occur BEFORE heel-strike if they were to stop. In all other conditions, the signal occurred after heel-strike.

taneous decrement of acceleration from the trailing limb and increase in braking force under the lead limb. The most common strategy used was to increase TA activation while inhibiting SOL, an effect made more pronounced by adding a target. The caveat to this was those subjects in the slower gait velocity group tended to increase SOL activity during planned stopping, effectively co-contracting throughout the stance phase of the trailing limb. Further, a significant proportion of the slow subjects with PD showed alternating bursts of activity in TA and SOL throughout mid-stance of the trailing limb. This finding is interesting in light of other findings related to the TA/SOL interaction in those with PD during GI, sit to stand and standing. During unplanned stopping, subjects with PD tended to increase activation of the extensor muscles of the leading limb to a greater extent than the control group. This most likely is to make use of a stiffer lead limb to rapidly generate deceleration forces rather than modulate the muscular activity of the trailing limb. Reliance on a single strategy to stop would place a subject at greater risk of failure to stop should an alternate strategy be indicated. Subjects with PD in our studies required increased warning to be able to stop suddenly. These subjects, therefore, must be instructed in techniques such as scanning so that they may have earlier warning to change direction or stop. Earlier warning will be required to avoid any obstacles that may become hazardous to the subject. Finally, our research indicates that key muscles are TA and GM. Perhaps rehabilitation programs should emphasize conditioning of these muscle groups to facilitate the ability to stop suddenly.

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