HEMIPLEGIA AND ITS EFFECT UPON FRACTIONATED PREMOTOR, MOTOR AND ANKLE DORSIFLEXION REACTION TIMES.

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Summary.-Ten hemiplegic subjects completed 20 rapid dorsiflexion of their afflicted and non afflicted limbs. Electrodes were attached to the tibialis anterior and the gastrocnemius muscles and electromyograms (EMG)s were recorded for their premotor time (PMT), motor time (MT) and simple reaction time (RT) during ankle dorsiflexion and plantar flexion of their lower limbs. The fractionated components of RT, namely PMT and MT, of both legs were statistically compared. It was found that the PMT of the subject's stroke, affected limb was significantly slower than the PMT of the non affected limb (control), with there being no differences being found between their associated MTs. These results supported the hypothesis that a stroke has a deleterious affect upon the central, PMT processing centers, and has no disruptive influence upon the peripheral MT. Utilizing the fractionated components of RT (PMT and MT), as compared to simple RT, they were found to be a more sensitive and valid method in order to detect possible injurious side effects of a stroke upon the brain's neuromotor transmission centers and sub-centers, and their peripheral, stimulus, response network.

Stroke is the third highest cause of death in the United States and the number one reason for resultant, crippling deformities. It is estimated by the National Stroke Association (1996) that stroke affects 400,000 to 500,000 people each year, with a substantial number of stroke survivors having disabilities that significantly interfere with activities of daily living. As about 70 percent of patients survive for a considerable time after the stroke with the prevalence of stroke-related disability being high; approximately 650/100, 100. Since the risk of stroke more than doubles with each decade after age 55, and the number of older Americans are steadily increasing, it is imperative that there should be an increase in studies involving the damaging side effects of stroke upon the possible disorganization of the brain's neuromotor, processing centers, and its chain reaction, distressful effect upon voluntary, limb movement.

A common side effect of a cerebrovascular accident (stroke) is that one side of the body is partially or totally paralyzed while the other side is often unaffected (hemiplegia). The pathophysiologic basis of the negative phenomena primarily reflect damage, to the motor coordination centers and subcenters. There is a spectrum of disability following stroke. In the lower extremity there is usually thigh adduction, foot inversion and plantar flexion.

The initial blueprint for a motor program was originated by Keele (1968), and since its inception there have been many competing and unresolved theories and concepts concerning how neurological centers and sub-centers process motor information prior to the execution of a coordinated volitional response. Henry (1980), Klapp (1980), Anson (1982), Christina, Fischman, Lambert and Moore (1985) and Christina (1991), Robert Christina (1991) compiled a scholarly review of the research literature in an attempt to unravel the controversy surrounding the competing theories that sought to explain the response complexity in skilled movement. Special attention was given to the memory motor drum theory conceived by Henry and Rogers (1960), which speculated as to the origin of neuromotor programming that precedes the onset of volitional movement. The traditional method employed by Henry and Rogers and many other researchers, in an attempt to solve this enigma has been to investigate the influence and relationships of RT's which preceded movements of varying degrees of complexity (Ito, 1997).

The early traditional experimental procedure of the aforementioned investigators in their failure to fractionate simple RT relative to its relationship to brain trauma, has continued to the present time. This questionable experimental design has masked the possible partial neurophysiological, interactions from the components of RT, namely PMT and MT. Hence in the current study the RT was fractionated into PMT and MT in order to shed new light upon the possible selective, injurious side

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effects, that stroke may have upon the central and peripheral elements of RT. Apart from a relative minor number of occasions when reflexes initiate movement simple RT precedes all other volitional limb movement. Hence, it is paramount that investigators discover and evaluate the role RT plays when its performance is impaired by the damaging, neurological after effects of stroke. Additional fundamental knowledge concerning RT should benefit rehabilitation programs that are employed following the impairment of gait, as an aftermath of stroke.

As a result of a stroke producing hemiplegia, the partial paralysis of one side of the body, is probably the result of brain damage which predominantly affects the central processing centers. Therefore the programming time of RT, which is responsible for the PMT interval, could possibly be disrupted whereas the peripheral element, MT, which is the transmission response time following the PMT, feasibly would be unaffected. Simple RT is the elapsed time between the onset of the stimulus and the initial first movement. The fractionation of RT involves PMT, which is the latency of the electromyographic onset following the presentation of the stimulus, and is determined by the subtraction of the MT from RT, MT is physical response and is the peripheral, electromechanical time delay in the execution of the program, following the heightened EMG activity above the motor point region, till the observation of the first significant physical response. It is the time required for the efferent impulses to generate enough muscular activity to initiate movement.

PMT is the period involving the processing of the stimulus and the interpretation and preparation of the response, and is the latency of the initiation of the EMG. It mirrors the elapsed time in order to organize and interpret the program for subsequent execution, and is a more accurate reflection of the central, processing time than the RT. Also the PMT reflects the monitoring time of the specific neuromotor, centers and subcenters and analyzes and interprets the stimulus initiated programs, coding them to the target group of skeletal muscles. Recent experimental progress has been achieved relative to resolving the problem concerning the specific information that is precluded when RT is used as a dependent variable in studies which involve the programming that is generated in the brain's processing centers and sub-centers. This leap in methodology has been achieved by fractionating RT into its two components of PMT and MT. Weiss (1965), Maria (1970), Lagasse and Hays (1973), Christina and Rose (1985), Sidway (1988), Siegal (1988), Ito (1990), Kasai and Seki (1992), and Ito (1997). It was therefore postulated that because of brain trauma following a stroke, the disruption caused within the neuromotor, coordination centers would only result in the impairment of the PMT component of RT and would not interfere with the affected limb's MT element of RT.

In reference to the sparsity of research findings concerning the study of RT of disabled stroke persons it was hypothesized that neurological damage that had resulted in the coordination of one of their lower limbs (hemiplegia) being significantly impaired, the neuro processing time component of fractionated RT, PMT, would be disrupted resulting in a slower programming time of the stimulus, coordination information, prior to the execution of the RT response. Hence the main purpose of the study was an attempt to ascertain what was the possible effect of brain damage, resulting from a stroke, upon the central processing of neuro motor information and the subsequent time interval elapsing before initial, motor movement.

METHOD

Subjects

The ten subjects were volunteers from a hospital stroke club with the average age of the three females and six male subjects being 60 and 61 years respectively. The tenth subject was a male aged 26. Three subjects had a diagnosis of a left cerebrovascular accident (CVA) and seven subjects had a right CVA. The subjects strokes had all occurred more than one year prior to being tested.

Procedure

Prior to the testing no subject had prior information relative to the main objectives of the study. Before commencing the trials the tester reviewed the test procedure with each subject, who were seated in front of the computer, and it was emphasized that the subject should react to the visual stimulus as fast as possible. Each subject completed five practice trials prior to their twenty test trials, for both limbs, interspersed with a 10 sec. rest period between each trial. The RT foreperiod was a random, delay range of one to four sec., which occurred immediately prior to the presentation of the visual cue, which was a 5 cm. diameter, blue disc on the computer's monitor.

A Toshiba 105CS laptop computer was utilized as the foundation of the data acquisition process. A custom program was written in Labview for Windows to control the process. LabviewÒ is a Windows based, graphical programming language from National Instruments, Austin, Texas. A National Instruments DAQCARD 1200 was used to interface the computer to record the subject's PMT, electromyographic reactions.

When the blue disc, the visual cue, was first displayed on the computer's monitor the RT counter was started. A push button switch, activated by the subject's foot, signaled the subject's movement, which in turn stopped this counter. Two seconds of surface EMG was recorded using Uni-Patch, Wabasha, MN, free form, rectangular, TENS electrodes, which were attached to the tibialis anterior and gastrocnemius muscles. Following the subject receiving the visual cue, the EMG was amplified, (Leaf Electronics QT-5B, low noise preamplifier, gain: 1000), bandpass filtered (ONE to 475Hz 40 dB/Dec.) and digitized (12 bits 1ms. sampling time). Following the recording, the digitized EMG was saved to a computer file for future reference.

The EMG electrodes and a ground electrode were smeared with conductive paste and were positioned approximately .5 cm. apart over the motor points of the tibialis anterior and the gastrocnemius muscles. Prior to applying the electrodes the skin was shaved, cleaned and rubbed with abrasive paper to remove any surplus dead skin in order to lower the impedance level prior to the recording of the EMG action potentials. The computer EMG traces indicated when the changes occurred from the recorded baseline to increased muscle activity at the motor point regions of the tibialis anterior and the gastrocnemius muscles.

Whenever possible the end of the PMT and start of the MT was denoted by observing when the EMG first became greater than twice the average of the EMG prior to significant activity. MT was calculated from this beginning of significant EMG activity to the opening time of the electronic circuit. The PMT was derived by subtracting the MT from RT. GRABINER (1988) in a discussion of the variability and possible error in the accurate recording and measurement of the onset of the initial EMG, suggests that the signal can be susceptible to temporal and spatial summations of myopotentials. In the present study, to heighten objectivity, the investigators each evaluated the specific EMG's and then cooperated in deciding upon the final target, EMG crest to be measured. The anterior tibialis and the gastrocnemius muscles were selected to be studied as they are the principal muscles employed in the dorsiflexion and the plantarflexion of the ankle joint.

RESULTS

For the data recorded from the active and inactive limbs, Table I displays the Means and Standard Deviations for the PMT, MT and RT. Five subjects were unable to move the affected limb so it was not possible to record data from their affected limbs.

Table I. Means and Standard Deviations of PMT, MT, RT

Limb		Unaffected	Affected
PMT	M	275.9	346.2
	SD	126.2	146.3
MT	M	91.9	171.4
	SD	49.17	103.6
RT	M	368.3	518.4
	SD	151.7	191.1

The Coefficient of Variation (Cv) is an absolute measure of the dispersion of the data (Miller, Freund & Johnson, 1990). The smaller the Cv the closer the grouping of the data.

Table II. Coefficient of Variation statistics.

Limb		Mean	SD	Min	Max
Unaffected	PMT	0.2968	0.0938	0.1482	0.4921
	MT	0.2202	0.0763	0.1114	0.3575
	RT	0.2319	0.081	0.1292	0.411
Affected	PMT	0.2676	0.1039	0.1855	0.448
	MT	0.2375	0.1124	0.1375	0.378
	RT	0.196	0.0639	0.1536	0.309

All correlations were achieved using the Pearson Product Correlation. Correlations for PMT VS. MT on the affected and unaffected limbs were .1476 and .3825 respectively. The correlations between limb times of the affected and unaffected limbs relative to PMT VS. PMT, MT VS. MT and RT VS. RT were .5426, .6627 and .5447 respectively. The correlations were quite similar in magnitude ranging from r= .5426 for PMT VS. PMT to r = .6627 for MT VS. MT. As the percentage of common variance only varies from 29% to 44% their low magnitude are not high enough for valid prediction purposes.

The reliabilities for the dependent variables RT, PMT and MT were determined by calculating the intraclass correlations for each subject's twenty trials. The intraclass correlations for the unaffected and affected limbs for PMT, MT and RT are depicted in Table III. The reliabilities for the MT's of the unaffected and affected legs were .9338 and .8471 respectively. This is in high contrast to the low reliability recorded for the PMT's namely .6578 and .5956 which indicates the greater inconsistency in performance as displayed by the central processing system as contrasted against the peripheral, efferent network.

Table III. Intra-class Correlations

Limb	Unaffected	Affected
PMT	0.6578	0.5956
MT	0.9338	0.8471
RT	0.7368	0.7375

For each of the dependent factors (PMT, MT, RT), a two-way randomized block design analysis of variance (ANOVA), with block on subjects (Hicks 1993), was performed on affected and unaffected factors from the five subjects which could volitionally move their affected and unaffected limbs. The experiment was not completely randomized since we always studied the unaffected limb first. Due to learning curve type effects, this may have biased our data in such a way that the unaffected limb times are longer due to learning and the affected limb times may be shorter.

Even with this possible bias decreasing the separation of affected and unaffected limb data the ANOVA revealed that the mean PMT between limbs was significant (F1,4 = 7.95, p < .05) and as expected there was a significant interaction between subjects; (F4,4 = 85.35, p < .005). Also the ANOVA showed that there were no differences between the Limb's MT's (F1,4 = 2.61 > .05) and subjects (F4,4 = 2.12 > .05. The ANOVA for RT disclosed the difference between subjects to be significant (F4,4 = 7.33, p < .05), but not an interaction between limbs; (F1,4 = 6.383, p < .10).

Figures 1 and 2 depict the two seconds of EMG recorded during the RT test. Note the longer quiet signal at the start of the affected figure as compared to the unaffected figure.

Figure 1. Two second recording of EMG from affected limb, subject H trial 12, PMT = 343 ms.

Figure 2. Two second recording of EMG from un-affected limb, subject H trial 10, PMT = 154 ms.

DISCUSSION

Results signified that each group was not equally affected by the stroke, with the PMT of the affected limb being found to be significantly slower than the PMT of the control's non affected leg. Also there was a greater disparity of time between the PMT and MTs of the affected limbs as contrasted with a similar comparison of the same two fractionated times of the non affected limbs. Differences in the variance of the RT's of the two limbs occurred chiefly in the premotor component of the affected limb and is interpreted as being principally a central function rather than a peripheral phenomena. Therefore, it appears reasonable to conclude that the

PMT, which is mainly responsible for RT response programming, is adversely damaged from the side effects of a stroke. The fractionation of RT showed that PMT, the central programming factor, and not the peripheral element MT, was predominantly responsible for the increase and fluctuation in RT. In general, the present findings parallel the conclusions of Ito (1997) who, in a study of RT as being a function of the number and similarity of sequenced elements in rapid force production, reported that the observed RT effects were mainly mediated by the central component of the total RT, namely PMT, as compared to the peripheral process, MT. Ito's conclusion that the main RT influence was a central effect is in agreement with the previous results of Fishman (1984) and Christina and Rose (1985).

In summary, the unresolved issue was investigated concerning the potential, injurious outcome of stroke upon the brains, neurological organization, and the subsequent peripheral response, as typified by the PMT and MT components of RT. The diverse findings which have been previously reported concerning the association between brain injury and its effect upon RT have resulted in there being no valid principle of generality or specificity being established relative to the influence of brain trauma upon the RT response. By only examining RT and ignoring the fractionation of RT into PMT and MT, invalid conclusions have been previously deduced from investigations concerning response programming relative to brain trauma. Although the RT of the unaffected limb was significantly slower than the affected leg's RT following fractionation of the RT, it was discovered that there were no differences between the MTs of both legs.

It was further revealed that PMT, has a more variable function, as compared to MT, relative the role that RT plays in central processing and peripheral relay. PMT, as compared to RT, was found to be a more valid measurement of the programming and processing activity of the central neuro motor centers, and in the future should be employed in studies to monitor possible traumatic side effects following the onset of a stroke. Results of the study demonstrated, that following a stroke the resultant, traumatic side effects which cause a slower RT was predominantly the disruptive effect of the disorganization of the central, processing PMT.

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