

## RECOVERY OF MUSCULAR PARALYSIS IN CEREBRAL STROKE PATIENT: A CASE STUDY USING SURFACE ELECTROMYOGRAPHY & DYNAMOMETRY

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### ABSTRACT:

**Objective:** To evaluate the recovery from muscular paralysis in Cerebral Stroke Patients using Surfaced Electromyography (SEMG) and hand dynamometry.

**Design:** SEMG & muscle force recorded from a patient of Cerebral Stroke suffering from hemiparesis.

**Setting:** The records were obtained from various affected and normal muscles of the patient on three consecutive visits at Neuromuscular Lab, Department of Physiology, University of Karachi.

**Subjects:** As case studies a patient who met cerebrovascular accident and was treated at a local hospital was examined for SEMG and hand dynamometry.

**Main outcome measures:** SEMG and hand dynamometry are useful tools to assess recovery in affected muscles in cerebral stroke patients.

**Results:** The muscle force was recorded only from Flexor Digitorum Profundus (FDP) that demonstrated significantly lesser values in the affected FDP than its contra-lateral normal one. While, SEMG parameters have demonstrated significantly higher values in the affected Trapezius (TPZ) and Biceps Brachii (BB) while lesser values in FDP, DI and Gastrocnemius (GAS) on first visit. All of these parameters showed significantly lesser values on third visit, in all the affected muscles.

**Conclusions:** Changes in SEMG and muscle force parameters during recovery from hemiparesis, follows the reorganization in cerebral cortex for the revival of muscular performance.

**KEYWORDS:** Surface Electromyography, Cerebral Stroke, Hemiparesis, Dynamometry, Recovery.

Pak J Med Sci October-December 2003 Vol. 19 No. 4 322-329 www.pjms.com.pk

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- \* Received for publication: March 15, 2003  
Revision received: July 1, 2003  
Revision accepted: Sept. 17, 2003

## INTRODUCTION

It has been reported that motor representations in cerebral cortex reorganize rapidly as a consequence of experience or peripheral lesions. This reorganization may arise through modifications in synaptic coupling among neurons of motor cortex, and indicate the role of motor cortex in learning and in performing voluntary movements<sup>1</sup>. Coherence in the frequency range between cortical slow wave recordings and rectified Electromyographies (EMGs) of hand and forearm muscles has also been reported during the task. They have been shown to represent a consistent task dependent modulation<sup>2</sup>. Similarly, Remy et al<sup>3</sup> found that the bilateral (BL) deficit in force and EMG is associated with reduced-movement-related cortical potentials. They suggested that BL force

and EMG deficit compared with unilateral handgrip contractions is caused by a mechanism of inter-hemispheric inhibition. On the basis of ipsilateral responses from the undamaged hemisphere, prevalent in poorly recovered patients<sup>4</sup>, it was suggested that underlying mechanism might not be beneficial for recovery. The ipsilateral motor pathway has been reported<sup>5</sup> as an important mechanism for functional recovery after focal brain injury during infancy. However, in adult it has not yet been demonstrated.

In the light of above cited literature it is evident that EMG signal analysis had been used for correlating the corticospinal activity with muscular performance. However, there is a scarcity in the literature for the use of Surface Electromyography (SEMG) and hand dynamometer in such studies. Further, it is expected that SEMG is more relevant with the recovery of the limb movements and task related force. Therefore, the present work is carried out to evaluate the SEMG and hand dynamometry as useful tool for the determination of recovery after cerebral stroke.

This case reports is based upon recovery of a 27-year-old male patient from cerebral stroke (Hemiparesis), evaluated through surface electromyography and Dynamometry.

Obtained results demonstrated a change in both the force and SEMG parameters, during recovery. It is our observation that SEMG and

Dynamometry are useful in correlating the corticospinal activity with muscular performance.

**PATIENT AND METHOD**

For SEMG recordings, standard disposable surface electrodes were used. The recordings of SEMG signals were obtained on a chart recorder (Lafayette 76107 TMG) through preamplifier. The muscle force was recorded by using hand dynamometer (Lafayette 76618, 0-100 Kg). The SEMG recordings were obtained from Gastrocnemius(GAS), Dorsal Interossei(DI), Biceps Brachii(BB) and Trapezius(TPZ) muscles of the affected side (right) and were compared with their contra lateral normal ones. In addition, SEMG and force records were also obtained from Flexor Digitorum Profundus (FDP) of the affected and contra lateral normal sides. The force records for GAS, DI, BB & TPZ could not be taken due to the limitation of hand dynamometer.

The table-I shows action performed and the position of electrodes during the recordings from different muscles of affected and contra lateral normal sides.

Before recordings, the cerebral stroke patient (27 year old male) was asked to sit comfortably in chair. The skin was cleaned with rectified spirit and disposable electrodes (adhesive) were applied for recordings from respective muscles.

SEMG and force parameters were measured

TABLE - I

Muscles	Action performed during recording	Position of bipolar surface electrodes	Ground electrodes
Trapezius (TPZ)	Flexion & Extension at shoulder joint	Between Spinous process of C-7 and the acromion	Over the Spinous process of C-7
Biceps Brachii (BB)	Flexion & Extension at elbow joint.	Over the belly of Biceps.	Over the belly of Triceps.
Flexor Digitorum Profundus (FDP)	Force full pulling of hand Dynamometer.	Medial Fore arm.	Laterally, at the surface of Brachialis near elbow.
Dorsal Interossei (DI)	Writing & Flexion / Extension of fingers.	Dorsal Interossei.	Posterior surface of Forearm.
Gastrocnemius (GAS)	Planter Flexion & Dorsi flexion of foot.	Proximal end of GAS belly.	Distal end of GAS belly.

from obtained records in their respective units for amplitudes and duration. The parameters measured for the quantification of SEMG signal are Number of Peaks (NOPs) in a single record, Maximum Peak Amplitude (MPA), Peak to Peak Amplitude (PPA), Maximum Peak Duration (MPD) and Duration of response (DOR) as given in Fig.1.

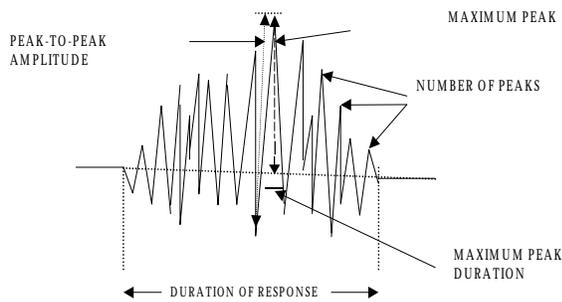


Fig. 1. Method for the measurements of SEMG Signal parameters

After calculating the average and standard error, the data was analyzed statistically for

comparison using student 't' test among the control and affected values at confidence limit of 0.05. The computation was done on Microsoft Excel version 97.

**RESULTS**

**1. TRAPEZIUS (TPZ):**

The average values of number of peaks (NOPs), duration of response (DOR) and the maximum peak duration (MPD) demonstrated non-significant difference (Table-II) between normal and affected muscle on first and second visit, except that DOR was found significantly ( $P < 0.025$ ) reduced in the affected muscle on second visit. While, on third visit, NOP was found significantly reduced ( $P < 0.0005$ ) and MPD, significantly ( $P < 0.025$ ) high in the affected muscle than it's normal one. Both the amplitudes, i.e., peak to peak amplitude (PPA) and maximum peak amplitude (MPA) were found significantly ( $P < 0.0005$  or both PPA and

**Table-II:** Comparison of various EMG parameters between normal and affected Trapezius muscles obtained on first, second and third visit. The patient was suffering from paralysis after cerebral stroke. All values are given as MEAN ± S.E.

EMG PARAMETERS	FIRST VISIT		SECOND VISIT		THIRD VISIT	
	NORMAL	AFFECTED	NORMAL	AFFECTED	NORMAL	AFFECTED
Number of Peaks	30 ± 2.94 (4)	37.4 ± 6.93 (5)	29.33 ± 3.58 (6)	24.83 ± 2.54 (6)	28.83 ± 1.66 (6)	16.67 ± 1.31 (6)
		P>0.05		P>0.05		P<0.0005
Duration of Response	1835 ± 181.9 (4)	2024 ± 344.93 (5)	2373.3 ± 190.1 (6)	1713.3 ± 182.55 (6)	1893.3 ± 106.67 (6)	1803.3 ± 84.28 (6)
		P>0.05		P<0.025		P>0.05
Peak to Peak Amplitude	249 ± 28.8 (4)	756 ± 68.85 (5)	533.33 ± 63.83 (6)	270.67 ± 20.28 (6)	787.7 ± 80.1 (6)	493.18 ± 74.2 (6)
		P<0.0005		P<0.005		P<0.025
Maximum Peak Amplitude	119.4 ± 12.94 (4)	405.4 ± 36.76 (5)	310 ± 59.3 (6)	161.33 ± 9.56 (6)	429.22 ± 62.04 (6)	246.56 ± 26.47 (6)
		P<0.0005		P<0.025		P<0.01
Maximum Peak Duration	25 ± 5 (4)	27.2 ± 6.25 (5)	19.33 ± 0.42 (6)	21 ± 1.84 (6)	19.33 ± 2.67 (6)	32.67 ± 5.33 (6)
		P>0.05		P>0.05		P<0.01

All durations presented in mS and all amplitudes in uV

**Table-III:** Comparison of various EMG parameters between normal and affected Biceps Brachii muscles obtained on first, second and third visit. The patient was suffering from paralysis after cerebral stroke. All values are given as MEAN ± S.E.

EMG PARAMETERS	FIRST VISIT		SECOND VISIT		THIRD VISIT	
	NORMAL	AFFECTED	NORMAL	AFFECTED	NORMAL	AFFECTED
Number of Peaks	17.83 ± 3.11 (6)	25.20 ± 4.73 (5)	23 ± 1.24 (6)	13.67 ± 1.15 (6)	23.6 ± 2.34 (5)	17.5 ± 1.18 (6)
		P>0.05		P<0.0005		P<0.025
Duration of Response	1727 ± 228 (6)	2056 ± 248.71 (5)	1793.3 ± 107 (6)	1560 ± 106.83 (6)	1488 ± 170.83 (5)	1200 ± 77.29 (6)
		P>0.05		P>0.025		P>0.05
Peak to Peak Amplitude	429.3 ± 44.81 (6)	862.80 ± 134.31 (5)	470 ± 75.79 (6)	288.75 ± 50.57 (6)	380 ± 32.1 (5)	379.48 ± 36.76 (6)
		P<0.01		P<0.05		P>0.05
Maximum Peak Amplitude	251.2 ± 31.19 (6)	497.20 ± 82.10 (5)	223.75 ± 35.83 (6)	155.00 ± 24.70 (6)	244.6 ± 15.46 (5)	219.22 ± 21.18 (6)
		P<0.025		P>0.05		P>0.05
Maximum Peak Duration	26 ± 1.37 (6)	45.60 ± 9.45 (5)	27.67 ± 4.01 (6)	35.00 ± 5.63 (6)	20 ± 1.26 (5)	18.67 ± 1.33 (6)
		P<0.05		P>0.05		P>0.05

All durations presented in mS and all amplitudes in uV

MPA) higher on first visit and significantly lower on second ( $P < 0.005$  for PPA and  $P < 0.025$  for MPA) as well as on third visit ( $P < 0.025$  for both PPA and MPA) in the affected muscle.

**2. BICEPS BRACHII (BB):**

The average values of NOPs (Table-III) have demonstrated non-significant difference between normal and affected BB on first visit, but

**Table-IV:** Comparison of various EMG parameters between normal and affected Flexor Digitorum Profundus (FDP) muscles obtained on first, second and third visit. The patient was suffering from paralysis after cerebral stroke. All values are given as MEAN  $\pm$  S.E.

EMG PARAMETERS	FIRST VISIT		SECOND VISIT		THIRD VISIT	
	NORMAL	AFFECTED	NORMAL	AFFECTED	NORMAL	AFFECTED
Number of Peaks	40 $\pm$ 1.15 (3)	27.75 $\pm$ 4.73 (4)	18.17 $\pm$ 0.7 (6)	18.5 $\pm$ 2.17 (6)	35 $\pm$ 3.31 (6)	23.17 $\pm$ 0.54 (6)
	P<0.05		P>0.05		P<0.005	
Duration of Response	2533 $\pm$ 70.55 (3)	2070 $\pm$ 281.13 (4)	1133.3 $\pm$ 52.32 (6)	1613.3 $\pm$ 170.85 (6)	2293.3 $\pm$ 234.3 (6)	2200 $\pm$ 213.4 (6)
	P>0.05		P<0.025		P>0.05	
Peak to Peak Amplitude	1575 $\pm$ 56.37 (3)	1159 $\pm$ 251.81 (4)	421.25 $\pm$ 81 (6)	652.5 $\pm$ 79.23 (6)	773.85 $\pm$ 67.76 (6)	504.32 $\pm$ 24.55 (6)
	P>0.05		P<0.05		P<0.005	
Maximum Peak Amplitude	844.7 $\pm$ 19.92 (3)	672.25 $\pm$ 130.82 (4)	231.25 $\pm$ 39.33 (6)	400 $\pm$ 44.94 (6)	544.57 $\pm$ 61.58 (6)	324.71 $\pm$ 25.84 (6)
	P>0.05		P<0.01		P<0.005	
Maximum Peak Duration	56.67 $\pm$ 6.67 (3)	49.5 $\pm$ 5.5 (4)	21 $\pm$ 1 (6)	28.33 $\pm$ 3.1 (6)	19 $\pm$ 2.52 (6)	17.67 $\pm$ 1.31 (6)
	P>0.05		P<0.025		P>0.05	

All durations presented in mS and all amplitudes in uV

**Table-V:** Comparison of various EMG parameters between normal and affected Flexor Digitorum Profundus muscles obtained on first, second and third visit by using hand dynamometer. The patient was suffering from paralysis after cerebral stroke. All values are given as MEAN  $\pm$  S.E.

FORCE PARAMETERS	FIRST VISIT		SECOND VISIT		THIRD VISIT	
	NORMAL	AFFECTED	NORMAL	AFFECTED	NORMAL	AFFECTED
Maximum Voluntary Force	41.33 $\pm$ 0.67 (3)	24.67 $\pm$ 0.33 (3)	24 $\pm$ 0.37 (6)	16.83 $\pm$ 1.01 (6)	39 $\pm$ 1.18 (5)	27.17 $\pm$ 0.7 (6)
	P<0.0005		P<0.0005		P<0.0005	
Force duration	2867 $\pm$ 170.2 (3)	2620 $\pm$ 185.83 (3)	840 $\pm$ 27.33 (6)	1306.7 $\pm$ 139.18 (6)	1990 $\pm$ 159.69 (5)	1693.3 $\pm$ 144.5 (6)
	P>0.05		P<0.005		P>0.05	
Rate of Rise in Force	36.46 $\pm$ 3.64 (3)	23.56 $\pm$ 1.6 (3)	41.94 $\pm$ 3.92 (6)	29.59 $\pm$ 4.55 (6)	27.31 $\pm$ 3.32 (5)	33.34 $\pm$ 3.42 (6)
	P<0.025		P<0.05		P>0.05	

All force durations in mS; voluntary force in Kg and rate of rise in Kg/sec

**Table -VI:** Comparison of various EMG parameters between normal and affected Dorsal Interossei (DI) obtained on second & third visit. The patient was suffering from paralysis after cerebral stroke. All values are given as MEAN  $\pm$  S.E.

EMG PARAMETERS	SECOND VISIT		THIRD VISIT	
	NORMAL	AFFECTED	NORMAL	AFFECTED
Number of Peaks	9.67 $\pm$ 0.99 (6)	5 $\pm$ 0.77 (6)	16 $\pm$ 1.67 (5)	19.8 $\pm$ 3.26 (5)
	P<0.005		P>0.05	
Duration of Response	1653 $\pm$ 84.33 (6)	1826.7 $\pm$ 139.2 (6)	1328 $\pm$ 157.2 (5)	1712.3 $\pm$ 78.38 (5)
	P>0.05		P<0.05	
Peak to Peak Amplitude	69.69 $\pm$ 12.42 (6)	58.48 $\pm$ 14.91 (6)	200.88 $\pm$ 46.26 (5)	259.48 $\pm$ 29.52 (5)
	P>0.05		P>0.05	
Maximum Peak Amplitude	38.1 $\pm$ 10.58 (6)	28.01 $\pm$ 10.07 (6)	113.78 $\pm$ 14.29 (5)	174.12 $\pm$ 21.25 (5)
	P>0.05		P<0.025	
Maximum Peak Duration	32.6 $\pm$ 6.46 (6)	23.33 $\pm$ 2.11 (6)	22.8 $\pm$ 4.88 (5)	28 $\pm$ 3.74 (5)
	P>0.05		P>0.05	

All durations presented in mS and all amplitudes in uV

was found to be significantly less on second (P<0.0005) and third visit (P<0.025) in the affected muscle. However, results for the DOR demonstrated non-significant difference between normal and affected muscle during all the three visits. The average values of PPA showed significant difference on both visits, being higher on first visit (P<0.01) and lower on second visit (P<0.05) in the affected muscle. While, this parameter demonstrated non-significant difference when compared with its normal on third visit. In addition, the MPA and MPD both showed significantly higher values (P<0.025 for MPA and P<0.05 for MPD) in the affected BB on first visit. Similar comparison on second and third visit has demonstrated non-significant difference when compared with normal muscle.

**3. FLEXOR DIGITORUM PROFUNDUS (FDP):**

Results obtained from affected flexor digitorum profundus (FDP) showed (Table-IV) that the average values of NOPs have significantly lower values than normal on first (P<0.05) and third visit (P<0.005) but non-significantly higher on second visit. However, DOR and MPD demonstrated significantly (P<0.025) higher values on second visit, while, non-significantly lesser values on first and third visit in the affected muscle. In addition, the PPA and MPA were found to be non signifi-

cantly different on first visit but demonstrated significantly different values between normal and affected muscle on second (P<0.05 for PPA, P<0.01 for MPA) and third visit (P<0.005 for both PPA and MPA). These differences being higher on second and lower on third visit in the affected muscle.

As shown in Table-V, force parameters obtained during Dynamometry involving FDP muscle demonstrated significantly lesser values (P<0.0005) in force generation (MVF) of the affected limb during all the three visits.

However, the force duration (MVC) was found to be non significantly different between normal and affected muscle on first and third visit, which was significantly high (P<0.005) on second visit, in the affected limb. A similar comparison, in the average values of the rate of rise in force (ROR), was found to be significantly lower on first (P<0.025) and second visit (P<0.05) in the affected limb. Nevertheless, this comparison on third visit demonstrated non-significant difference between normal and affected FDP.

**4. DORSAL INTEROSSEI (DI):**

Results obtained from DI showed significantly lower (P<0.005) values of NOPs in affected muscle on first visit (Table-VI). While, this parameter demonstrated non-significant difference on second visit. On the other hand, DOR was found non significantly different on

*Table-VII: Comparison of various EMG parameters between normal and affected Gastrocnemius obtained on first, second and third visit. The patient was suffering from paralysis after cerebral stroke. All values are given as MEAN ± S.E.*

EMG PARAMETERS	FIRST VISIT		SECOND VISIT		THIRD VISIT	
	NORMAL	AFFECTED	NORMAL	AFFECTED	NORMAL	AFFECTED
Number of Peaks	18.5 ± 3.46 (6)	15.2 ± 2.4 (5)	12.5 ± 1.1 (6)	11.83 ± 0.91 (6)	31.67 ± 3.47 (6)	28.17 ± 1.82 (6)
	P>0.05		P>0.05		P>0.05	
Duration of Response	1827 ± 391.1 (6)	1576 ± 310.25 (5)	2000 ± 210.9 (6)	2300 ± 81.158 (6)	2493.3 ± 239.48 (6)	2184.5 ± 154.5 (6)
	P>0.05		P>0.05		P>0.05	
Peak to Peak Amplitude	343.5 ± 37.44 (6)	81.46 ± 11.8 (5)	120 ± 20.66 (6)	92 ± 12 (6)	633.33 ± 129.94 (6)	353.65 ± 24.78 (6)
	P<0.0005		P>0.05		P<0.05	
Maximum Peak Amplitude	206 ± 25.8 (6)	40.8 ± 3.8 (5)	71.33 ± 11.24 (6)	43.38 ± 6.52 (6)	302.83 ± 70.65 (6)	214.43 ± 13.58 (6)
	P<0.0005		P<0.05		P>0.05	
Maximum Peak Duration	56.67 ± 4.33 (6)	36 ± 4 (5)	33.33 ± 2.11 (6)	43.33 ± 4.22 (6)	17.67 ± 1.96 (6)	24 ± 2 (6)
	P<0.005		P<0.05		P<0.025	

All durations presented in mS and all amplitudes in uV

first visit, which was significantly ( $P < 0.05$ ) higher in the affected muscle on second visit. In addition, the PPA, MPA and MPD were found non significantly different on both the first and second visit, except that MPA was significantly high ( $P < 0.025$ ) in the affected muscle on second visit.

##### 5. GASTROCNEMIUS:

Results obtained from GAS muscle demonstrated non-significant difference between normal and affected limb for the two parameters i.e., NOPs and DOR during all the three visits (Table-VII). The average values of PPA were found significantly lower on first ( $P < 0.0005$ ) and third visit ( $P < 0.05$ ). However, this comparison on second visit, demonstrated non-significant difference between normal and affected muscle. Further, the MPA was found to be significantly reduced in affected limb on first ( $P < 0.0005$ ) and second visit ( $P < 0.05$ ). While, this parameter demonstrated non-significant difference between normal and affected muscle on third visit. However, the average values of MPD showed significant difference between normal and affected muscle on all the three visits, being lower on first visit ( $P < 0.005$ ) and higher on second ( $P < 0.05$ ) and third visit ( $P < 0.025$ ) in the affected muscle.

## DISCUSSION

This patient suffered left cerebral ischemia resulting in right hemiparesis. EMG recordings from two of the proximal limb muscles, i.e., TPZ and BB showed that all the EMG parameters were higher in the affected limb on first visit. This enhanced response in the affected side was probably due to firing of greater number of motor units (MUs) in the affected limb. This suggests that even due to the damage in the left side of brain, activation of pre-existing uncrossed motor neural pathways, accessed or recruited for compensation after ischemic stroke<sup>6</sup>. Further, it is speculated that there might be collateral sprouting from the distal branches of the nerve fibers<sup>7</sup> occurring in the affected muscles that accounted for enhanced

response. However, on second and third visit all the EMG parameters except MPD (significantly higher in TPZ) showed a decline in the affected side. It is probably due to upper and lower motoneuronal degeneration in the cortex and also representing the lesser muscle involvement. Study<sup>8,9</sup> of a group of patients rendered hemiplegic by a cerebro-vascular accident found the evidence of MUs loss in the extensor digitorum brevis on the affected side. They considered it as evidence of wasting in hemiplegia due to the degeneration of anterior horn cell (AHCs) instead of disuse following deprivation of 'trophic' influences from cortical sources. Further, the higher values of MPD on second visit may be attributed to an increase in motor unit territory as demonstrated by Erminio et al.<sup>10</sup> that MU territory may be increased to 80-140 percent in patients with weakness due to degeneration of AHCs.

In distal muscles FDP, DI and GAS, all the EMG parameters were found lesser in the affected side on first visit, except DOR being high in DI. The lesser values of majority of the parameters in these muscles indicate muscular weakness. It has been suggested<sup>11</sup> that after large lesions of the primary motor cortex (MI), voluntary movements of affected body parts become weak and slow. In addition, the suggestion of less number of MUs activation in the affected limb also involves hyper-excited inhibitory neurons after cortical lesions. It has been proposed<sup>12</sup> earlier, that in stroke patients severe motor dysfunction may be caused by hyperactivity of cortical inhibitory inter-neurons rather than by direct lesions of descending motor tracts.

On second visit, DI demonstrated higher values of all the EMG parameters in the affected side. It is probably due to rapid motor recovery during patient's skilled learning that involved greater number of MUs during muscular performance in subsequent visits. It suggests reorganization of motor cortex following unilateral cerebral infarction<sup>13</sup>. It is also proposed<sup>14,15</sup> that a slowly evolving, long-term, experience-dependent reorganization of the adult MI lead to the acquisition and retention

of the motor skill. Accordingly, it is proposed that in the present study, recovery from cerebral stroke is evident from improved muscular performance since, patient had undergone different types of skilled movements and daily practices during recovery period.

The results obtained for FDP on second visit were similar to the above-discussed results for DI, with all the EMG parameters high in the affected muscle. Higher values of EMG parameters indicate involvement of greater number of MUs, that suggests a possible compensatory action mediated through other parts of brain. In addition, due to lesion on left side of the patient's brain, the contra lateral Somatomotor Association Area and Somatomotor<sup>3,11</sup> were highly activated and involved in the movements of body parts of right side to compensate for the lesion on left side.

In GAS, all the EMG parameters were lesser in the affected muscle except MPD on third visit. The lower amplitudes were due to weak contractions. In this connection, the finding that after large lesions of the MI, voluntary movements of the affected body part becomes weak and slow<sup>16</sup> is relevant. However, in the present study GAS response duration was rather shortened with fall of amplitude. Moreover, the higher values of MPD on third visit may be similar to the findings<sup>10</sup> that showed an increment in MU territory in patients with weakness due to degeneration of AHC.

Force parameters recorded from FDP muscle showed lesser values in the affected muscle on all the three visits, except that force duration was significantly high on second visit and ROR was non significantly high on third visit. Similarly, in a study Radhakrishnan et al.<sup>17</sup> found 1/8<sup>th</sup> grip strength in hemiplegic cases than normal ones.

In the light of the above discussion, it is clear that the rapid recovery in muscular performance observed in the present study is due to one or more of the following compensatory reasons:

1. Access of pre-existing uncrossed motor neural pathways.
2. Hyper-excitability<sup>18</sup> in the vicinity of dam-

aged portion of brain or in the contra lateral hemisphere with functional reorganization after stroke.

3. Skilled learning<sup>19</sup> accounted for enhanced response in skeletal muscles of the affected limb on third visit due to rebuilt neuronal circuits, e.g., by sprouting and/or regeneration.

## CONCLUSION

Surface Electromyography & Dynamometry are useful in correlating the muscular performance with corticospinal activity & changes observed in SEMG Dynamometry during recovery from hemiparesis following the reorganization of cerebral cortex for the revival of muscular performance.

However findings are based on study of just one case and there is a need for further studies including more cases especially elderly in which stroke is common.

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