ECT Seizure Duration: Measurement-Related Differences

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Abstract

Three alternative methods for assessing seizure duration during electroconvulsive therapy were studied. The visual and tactile estimation of motor convulsions and tachycardia as well as the mean integrated amplitude of the electroencephalogram were compared with the reference single channel unprocessed electroencephalogram in 78 treatment sessions given to 17 patients. The measure of seizure duration differed significantly ($P<0.001$, 0.01). Larger, but clinically unimportant, discrepancies were obtained with the visual and tactile observational measures. The difference between mean of integrated electroencephalogram amplitude and that of EEG were small and seen as an artifact of the device sampling procedure. The clinical and research values of the results were discussed.

Introduction Psychiatrists attempting to treat certain severe mental disorders have recently shown renewed interest in electroconvulsive therapy (ECT).A number of technical innovations have made ECT safer and more effective including, titrated stimulus dosing determination of seizure adequacy and electroencephalographic (EEG) monitoring (Weiner & Krystal 1994).

Seizure duration (SZD) guidelines have evolved, based on concerns about decreased effectiveness of very short seizures and neurotoxicity of overly long seizures (Lambert & Petty, 1994), which need further work to verify the assumptions they are based on.

The importance of monitoring of seizure activity during ECT is well established, and verified (Fink,1979). Optimal assessment of SZD includes both indices of electrocortical seizure generalization and signs of convulsive activity (Fink & Johnson,1982). Among the prevalent techniques used to document ECT-induced seizures are single- and multiple channel unprocessed and processed EEG (Fink & Johnson, 1982), observation of autonomic response (Kitamura & Page, 1984), and cardiovascular monitoring (Larson et al.1984). Unprocessed EEG monitoring is the preferred method, and the one with which others are most often compared (Fink & Johnson, 1982). Immediately following the transcranial electrical stimulus for ECT, five characteristics patterns of EEG activity are seen, three occuring during the ictus (Weiner, 1982). There is a brief preictal (recruiting) phase of relative EEG suppression as low-voltage, high frequency activity, which is often undetected due to amplifier saturation by stimulus. Next the tonic phase of rhythmic beta-frequency activity (14-22 Hz) appears within 15 seconds and lasts up to 20 seconds. Corresponding to the clonic phase of the seizure, high amplitude activity progresses from desynchronized, high frequency polyphasic wave forms to rhythmic delta 2-4 Hz activity (fourth form). Muscular jerking occurring during the clonic period is synchronous with high amplitude epileptiform bursts, corresponds to individual muscle contractions and may last 20-60 seconds

(Weiner,1983) A near flat line of postictal suppression (fifth pattern)signifies the physiological end of a cortical seizure that coincides with the cessation of muscular jerking. Abrams & Swartz(1989) noted that about 10% of seizures will blend so gradually into the postictal phase that a precise end point cannot be identified by inspection.

SZD can be measured by microprocessor-based EEG analyzer that relies on numeric descriptors. High correlation between records of unprocessed and processed EEG cortical SDZs have been reported (Swartz & Abrams,1986). An alternative approach relies on auditory modulation of EEG frequency and amplitude (Abrams & Swartz, 1989). Weiner et al. (1986) confirmed that judgment of seizure end point using this audible EEG monitor has been determined to be reliable and valid.

The external nervous application of electrical current during ECT stimulates an automatic nervous system discharge (Gravenstein, et al. 1965, Jones & Knight, 1981 and Weiner, 1982). Observed signs (tachycardia, hypertension, and increased electrodermal conductance) have been used as evidence of a generalized convlusion. There is a close agrement between clinically beneficial SZD measured by tachycardia and EEG (Larson, et al.,1984). The point of greatest heart rate deceleration represents the end of convulsion; however, autonomic signs lack specificity so that decisions based solely on these parameters are ill-advised (Kitamura & Page, 1984).

The primary goal of the current study is to compare the simplest, and cheapest method of visual and tactile clinical observation of gross motor activity and other autonomic responses as measures of ECT-SZD on one hand with the objective techniques assessed by unprocessed EEG and computer processed EEG amplitude in a group of psychiatric patients. Putting in mind that in a busy general hospital, in a developing country with tightness of availability of time, equipment and personnel, a simple practical method needing no equipment and only one or two personnel is badly needed if it is correlated with small differences and proved to verify seizure induction and document its favourite duration.

Subjects & Methods: A randomized study of SZD in 27 adult inpatients (8 females, 9 males), 20-70years old (mean=34.6±017.3) undergoing ECT for drug-resistant severe depressive (n=15) and schizophrenic disorder (n=2) was fulfilled. Each patient underwent a minimum of 4 treatments and received the induction drugs, described below, equally. A total of 78 treatment sessions were evaluated, in which all the three measurement techniques were applied.

Anesthetic management included pretreatment with atropine sulfate (0.01 mg/kg), thiopentone (5 mg/kg), and succinylcholine (1 mg/kg). Immediately prior to administration of the muscle relaxant, occlusion of blood flow to one limb by reinflating the applied blood pressure cuff to 10 mm Hg above systolic pressure was performed to permit observing the unmodified motor seizure in muscles distal to the cuff. Ventilation was assisted using a mask while administering 100% oxygen. This was followed by the assessment of sufficient muscle relaxation by the nerve stimulator. Routine monitoring included ECG, arterial blood pressure, and O2 saturation by pulse oxymeter.

All patients received bilateral ECT using the Mecta model-SR1 device. The stimulus parameters of the 800 mA current were individualized with the aim of inducing a cortical seizure of 30-60 sec-

Table (1) Stimulus parameters of the studied group (n=17):

	Range	Mean ± SD
Duration (s)	0.72-2.00	1.28 0.35
Frequency (Hz)	50-90	70.5 21.9
Pulse width (ms)	1.2-1.4	1.34 0.39

onds as measured by the conventional EEG (Table.1)

Visual and tactile assessment of motor convulsions in the muscles of the arm distal to the inflated cuff was done by the anesthesiologist, the trained nurse anesthetist and the psychiatric resident. The initial response of passage of electric stimulus is a sustained, tonic contraction of musculature (lasting 10-20 sec.) and characterized by flexion and a very fast, fine tremor. This tonic phase is gradually succeeded by repetitively clonic, jerking movements, gradually slowing from a frequency of 10-12 per sec. to 3-4 per sec. at seizure termination, when all movement abruptly ceases. At this point the blood pressure cuff is deflated. Reliability indices between rates were high ranging between (+0.84-0.91) when placing a mark on the EEG strip chart as well as using a stop-watch to register the duration of motor convulsion and tachycardia was also noted on the EEG strip of the monitor.

Unprocessed single-channel EEG recordings(25 mm/s 0.5 or 1.0 mV/cm) were obtained from the Mecta machine. The attending psychiatrist timed SZD from stimulus offset until onset of postictal suppression. In ten cases, an experienced ECT practitioner and a neurologist who were unfamiliar with the purpose of the record review, assessed SZD, with high interrater reliability(=0.97). Discrepancy of greater than 1s was noted in only one of the ten records.

The mean integrated EEG amplitude (MIA) was determined from the Anesthesia and Brain Activity Monitor

Table (2) Comparison between measurement techniques for seizure duration(s)

	Range	Mean±SD	Median	Skewness	Kurtosis	P-value
Simple	0-126	46±19.2	45	0.91	6.32b	0.001
EEG	33-234	771±35.0	60	2.04	8.52b	0.01
MIA	30-190	77±3.35	70	1..33	4.68b	

(AMB, Datex/Puritan-Bennett, Boston, Mass). The MIA was measured by selective filtering of biopotentials obtained from the adhesive electrodes placed on mid-forehead (active), over mastoid process(reference) and over temporal bone (ground). MIA ranged from 0-100MV rms in the 1.5-2.0 Hz band width. Digitized values were stored on a floppy disk. The sharp return of the ECT-induced MIA elevations towards pre-seizure baselines determined the end of ictal activity.

Since the purpose of the existing work was to compare measurement techniques rather than ECT effects, sessions for each patient were considered as independent events. The null hypothesis stated that indices of ECT-SZD did not differ. Since the duration were not normally distributed, the null hypothesis was tested using non-parametric analysis of variance. Individual pairwise comparisons were made using Wilcoxon matched-pairs single-ranks test (table2).

Results: The distribution of values of each measure of SZD is shown in Table (2). A Freidman two way analysis of variance by ranks indicated that the measures were significantly different ($P<0.001$). Absence of convulsive activity during a documented electrocortical seizure occurred once, and it seems that seizure did not generalize in that case. Though the duration of ictal activity, as defined by MIA technique differed from that directly related to EEG, the mean MIA-SZD was only 6 seconds longer than unprocessed EEG estimate. The variability of measures was related to their absolute magnitude. A more detailed examination of EEG-SZDs. indicated that a two-fold increase in duration was associated with three-fold increase in the standard duration, that is, longer electrocortical seizures were more variable in duration than shorter ones. The mean duration of the motor ictal component was consistently shorter than the other components (15-25 seconds less than processed MIA and unprocessed EEG respectively).

Discussion: The use of EEG monitoring in convulsive therapy has gained widespread acceptance in the developed countries where devices with this capability are readily available. In other countries clinical monitoring alone is more common. Discrepancies between EEG and clinical observation have been known to exist. As Lambert & Petty (1994) noted, currently available duration guideline do not indicate how to manage disparate results.

The traditional view showed that the production of generalized seizure of adequate duration is both necessary and sufficient for therapeutic response to ECT. An effective seizure was defined as a bilateralized seizure with minimum duration of 25 seconds (American Psychiatric Association Task Force Report,1984). Abrams & Swartz (1989) estimated that less than 20 seconds duration would result in abortive, missed partial or focal seizures that leads therapists to increase the dosing strategy to induce a generalized seizure of satisfactory duration for the effect. But Miller et al. (1985) reported insufficient data to indicate the relationship between seizure length and ECT efficacy, to specify a minimum duration of seizures, individually or cumulatively.

That traditional view was challenged by Mc Gravey et al. (1993) and Krystal et al. (1993) who commented that ECT efficacy not simply depends upon surpassing a minimum SZD as has been assumed, and Nobler et al.(1993) indicated that SZD is not a useful marker of therapeutic efficacy, and more useful markers are provided by ECT stimulus intensity

electrode placement and the ictal and immediate postictal EEG. Scott et al., (1994) assessed short term effects of a single bilateral ECT on uptake of 99 mTc exametazine into brains of patients with unipolar major depression using split-dose SPECT, and found that the changes were not correlated with stimulus intensity or EEG-monitored SZD, but were correlated with severity of depression and more weakly with ECT produced memory impairments.

Nevertheless reliable monitoring of electricity induced seizure is an important component of ECT. Present monitoring techniques rely on observations that generalized electrocortical SZD by EEG equals or exceeds that of the accompanying motor and autonomic activities. The current work, despite its methodological unification of most variables, demonstrates marked measurements- related differences between estimates of SZDby the use of different techniques.

The obtained differences either inter- or intra-individual can be interpreted to result from 1) higher seizure thresholds as reported by Sackeim et al. (1987-a) and Abrams & Swartz(1989). 2) Subsequent seizures (Sackeim et al.1987-b) despite that di-Michele, et al. (1992) showed only slight statistically insignificant reduction in ECT-induced SZD across a course of ECT, 3) anesthetic dose (Michell,et al., 1991 and Martensson, et al.,1994) but di-Michele & Rossi(1993) noted from a clinical point of view that SZD and threshold are not related to that dose, 4) stimulus energy and impedance (American Psychiatric Association Report, 1982), 5) stimulus train or number of pulses (Weaver, et al. ,1977 and 1982), 6) use of psychotropic drugs as shown in the work of Lanes & Ravaris (1993) who reported prolonged SZD in a 70-y-old female patient on trazodone that resolved upon its discontinuation, 7) dose of muscle relaxant as Miller et, al.(1985) recorded inverse relation between SZD and muscle relaxant, 8) use of cerebral stimulants like caffeine that can lengthen SZD by lowering convulsive threshold as declared by Hinkle, et al. (1987) and Lurie & Coffey (1990). Other factors that might be responsible for such variations in SZD that was abolished by the methodological awareness may be a) electrode placement as all cases were treated with bilateral than unilateral ECT,b) type of anesthetic drugs as they were all injected with thiopental and c) patients diagnosis as 88.2% of the studied group were depressed subjects. relatively small mean of number of ECT-treatment given may limit the indicated cumulative effect of SZD.

It seems that SZD estimation based on heart rate should be regarded with caution due to the modifications by the anesthetic agent itself, the atropinic agent, and succinylcholine. A valsalva effect consequent to forced expiration against a closed glottis triggered by the direct action of the electric current may cause a 25% increase in heart rate as detected by Kitamura & Page (1984). Another falacy was indicated by Lane et al. (1988) that bilateral ECT peak heart rate remains higher throughout the early postictal phase than with right- unilateral ECT, indicating greater circulating catecholamine levels. But, Larson et al.(1984) stated that duration of ECT-induced tachycardia which is easily determined as noted by high interrater reliability index in the existing study is correlated highly with both motor (cuffed arm) and ECT seizure estimates. As dose heart rate Abrams & Swartz (1989) showed that blood pressure fluctuated throughout the ictal and postictal phases especially in patients with arteriosclerosis and hypertension.

Another source of difference in SZD estimation may be the notation of Abrams & Volavka (1982) and Abrams & Swartz (1989) that EEG seizure activity may continue for 10-15 seconds longer than when one relies upon observing the motor manifestations of the cerebral activity in the arm distal to the inflated blood-pressure cuff. In the present results, EEG seizure activity continued when all movement seems abruptly to cease. The reason may be related to some characteristics of neuronal activities. Differences were greater with the first ECT of the given course in younger patients. These differences can be partially abolished by combining measures of magnitude and length of ECT-induced automatic response as tachycardia and hypertension to motor response, thus increasing the potential for more accurate clinical SZD evaluation compared to that with EEG. The same conclusion was given by Lippman et al. (1982) and Bergsholm, et al. (1993) recommending use of the above mentioned combination for more precise clinical assessment.

Although the difference between clinical and EEG determined SZD were statistically significant, they do not dispute the clinical value of the simple primitive measures, particularly if combined. This difference in data suggested that both the clinical and electrical measures of cortical activity express directly related but different neurophysiological processes.

The smaller but significant ($P<0.01$) differences in SZD between unprocessed and processed EEG (MIA) were anticipated. The closer agreement suggested that the MIA information reflects the same phenomenon as the unprocessed EEG. The observation of $\pm$ 6 seconds in the mean of SZD resolution of the processed graphic recording is sufficient to explain all differences, and illustrates the need for familiarity with the EEG processor logarithm. Sampling techniques of some devices, e.g. 4-seconds epochs at 1 minute intervals, preclude measurements of electrocortical SZD despite their attractive graphic displays (Swartz & Abrams,1986). The interrater reliability of measured SZD monitored by EEG was higher than that of the clinical observation technique (0.97). When it was examined by Lambert (1992) between five psychiatrists assessing 30 readout strips, he also commented that it was good, with no effect of the rate found on duration monitoring.

Regardless of the measurements techniques, the interpretation of EEG during ECT requires caution. The interval between anesthetic/relaxant administration and ECT application must be long enough to minimize unintended interactions that may complicate interpretation. Short acting barbiturates generally depress vigilance and muscular tone. However they occasionally increase EEG amplitude and induce myocolonus soon after injection (Lippman et al. 1993, Fredman et al. 1994). Similarly, the short acting neuromuscular blocker, as succinylcholine, may induce a brief period of muscular fasciculations. Out of the present study, we prefer to have an appropriate delay in the application of ECT to assure that increased EEG amplitude or muscular activity is not drug related.

The non-Gaussian skewed distribution of ECT-seizure lengths was expected because of the stimulation technique (Abrams, 1988). Stimulus parameters are typically adjusted to produce a seizure lasting at least 25 seconds. This limits the lower end of the distribution while prolonged seizures extend the upper limit. The use of parametric statistics with such skewed data can lead to unwarranted conclusions.

In conclusion, this work is hoped to cast light on some basic problems per-

taining to SZD assessment. It reflects at the electrocortical measures (EEG & MIA)agreed closely, but differed from the clinical observation convulsion and autonomic variables. Inspite of the significant difference detected in the SZDs measured by the visual and tactile method on one hand and those measured by the EEG methods on the other hand there was a direct relationship between them.. Thus the crude clinical method could be practically useful for every day routine clinical use especially from prognostic view point (i.e.) to monitor the progress of the psychiatric disorder, the effect of concurrent therapies, and to estimate the number of ECT sessions needed. A more precise method like EEG or MIA is required for accurate research measurements.

A variable change in the SZD was observed in relation to the original diagnosis as well as the progress of the disorder in the exiting work, for which a further study is recommended to quantify this change and to qualify its relation to these two variables.

References

Abrams, R. (1988) : Electroconulsive Therapy. Oxford Univ. Press, N.Y., pp. 101-3

Abrams, R. ,and Swartz, C. (1989) : ECT-Instruction Manual. 3rd Ed. Chicago Med. School, pp. 10-9,33-6

Abrams, R.,and Volavka, J. (1982) : Electroencephalagraphic effects of convulsive therapy. In : R. Abrams & W. Essaman, Electroconulsive Therapy : Biological Foundation and Clinical Application, Spectrum Publications, N.Y.,pp157-67.

American Psychiatric Association Task Force Report (1978) : on ECT Report 14, Am. Psyhchiat. Assoc., Washington, DC.

American Psychiatric Association Task Force Report (1982) : on ECT Device standards, Petition to Reclassify ECT Devices, Appendix G., Am. Psyhchiat. Assoc., Washington, DC.

Bergsholm, P., Bleie,H., Gran,L., and d'Elia G.,(1993) : Cardiovascular response and seizure duration as determined by electroencephalography during unilateral electroconvulsive therapy. Acts Pshychiat. Scand, 88(1),pp.25-8.

di-Michele, V., Giordano,L., de-Cataldo, S., Sabatini, M.,et al. (1992) : Electroencephalography seizure duration in electroconvulsive therapy. A clinical study. Conv. Therapy, vol.8(4), pp.258-61 {Eng. Abst.)

di-Michele, , and Rossi, A. (1993) : Seizure duration and seizure threhsold : Reply. Conv. Therapy, Vol. 9(2), p.131. [Eng. Abst.] .

Fink, M. (199) : Convulsive Therapy : Theroy and Practice. Raven Press, N.Y., pp137-41.

Fink, M. (1988) : Convulsive Therapy : A manual of practice. In : review of Psychiatry. A Frances & R. Hales (Eds.), Vol. 7, p482. Am. Psyhchiat. Assoc., Washington, DC.

Fink, M. , and Johnson, L. (1982) : Monitoring the duration of electroconvulsive therapy seizures. Am. J. Psychiat. 39 : 1189-91.

Fredman, B., d'Etienne, J., Smith,I., Husain, M., and White, P. (1994) : Anesthesia for electroconvulsive therapy : effects of propofol and methohexital on

seizure activity and recovery. Anesth. Anls. 79(1) : pp75-9.

Gravenstein, J., Anton,A. Weiner,S., and Tetlow,A. (1965) : Catecholamine and cardiovascular response to electroconvulsive therapy in man. Br. J. Anesth., 37: 833-9.

Hinkle, P., Coffey, C., Weiner, R.,Cress, M., christison, C. (1987) : Use of caffeine to lenthen seizures in ECT . Am. J. Psychiat 144(9), pp.1143-8.

Jones,R.,and Knight, P. (1981) : Cardiovascular and hormonal responses to electroconvulsive therapy : Modification of an exaggerated response in a hypertensive patients by beta- receptor blockade. Anesthesia 36 : 795-9.

Kitamura,J., and Page, A.(1984) : Electrocardioghraphic changes following a lectroconvulsive therapy. Eur. Arch. Psychiat. Neurol. Sci. 234 : 147-8.

Krystal, A., Weiner, R., Mc Call, W., Shelp, F., Arias, R., and Sm (1993) : The effect of ECT stimulus dose and electrode placement on the ictal electroencep halogram : an intraindividual crossover study. Bio Psychiat. , 34(1), pp.75.

Lambert, M (1992) : Interrater reliability of seizure duration in ECT. Biol. Psychiat., vol. 31(1) : 102-3.

Lambert, M ,and Petty, F. (1994) : EEG seizure duration monitoring of ECT. Biol. Psychiat., vol. 18(3) : 497-502.

Lane, R., Zeitlin, S., abrams, R., Swartz, C. (1988) :Differential effects of right unilateral and bilateral ECT on heart rate. Am. J. Psychiat 148 : 961.

Lanes, T., and Ravaris, C. (1993) : Prolongled ECT seizure duration in a patiant taking trazodone. Am. J. Psyciat. vol. 150(3) : p. 525.

Larson, G., Swartz, C., and Abrams, R. (1984) : Duration of ECT-induced tachycardia as a measre of seizure length . Am. j. Psyhiact. 141 : 1269.

Lippman, S., Hass,S., and Quast, G. (1993) : Procedural complication of electroconvulsive therapy : assessment and recommendations. South Med J. (USA), 86(10), pp1110-4.

Lurie, S. and Coffey, C. (1990) : Caffeine-modified electroconvulsive therapy in depressed patients with medical illness. J. Clin. Psychiat. 51(4) 154-7.

Martensson,B.,Bartfai, A.,Hallen, B., Hellstrom, C. Junthe, T., and Olander (1994) : Acomparison of propofol and methohexital as anesthetic agents for ECT: Effects on seizure duration, theapeutic outcome and memory. Biol. Psychiat., 35(3), pp. 179-89.

Mc Gavey, K, Zis, A., Brown, E., Nomikos, G., and Fibiger, H (1993) : Ecsinduced dopamine release : effects on electrode placement, anticonvlsive treatment, and stimulus itensity. Biol. Psychiat., 34(3), pp.152-7.

Miller, A., Faber, R., Hatch, J., and Alexander, H. (1985) : Factors affecting amnesia, seizure duration, and efficacy in ECT. Am. J. Psychiat 142(6)- pp.692-6.

Miller , P., torda, T., Hiokie, I., and Burke, c., (1991) : Propofol as anesthetic agent for ECT : Effect on outcome and length of course. Australian-and- Newzealand J. of Psychiat. vol. 25(2), pp. 255-61, [Abst.].

Nobler, M., Sackeim, M., Solomou,M., Luber,B., Devanand,D., and Pru-

dic,J.(1993) : EEG manifestations during ECT : effects of electrode placement and stilmulus intensity. Biol. Psychiat.,34(5), pp.321-30.

Sackein, H., Decina, P., Malitz, S., and Resor,S., (1993) : Anticonvulsant and antidpressant properties of electroconvulsive therapy : A proposed mechanism of action. Biol. Phychiat. 18 :1301-10.

Sackein, H., Decina, P.,, Protnoy, S., Neeley, P., Maltize, S., (1987-a): Studied of dossage, seizure threshold and seizure duration in ECT. Biol. Psychiat. 22(3), pp249-68.

Sackein, H., Decina, P.,,Prohovink,I., and Maltiz,S. (1987-b) : Seizure threshold in electroconvulsive therapy : Effects of age, sex, electrode placement and number of treatments. Arch. Gen. Psychiat. 44:355-60.

Swatrz, C. and Abrams, R. (1986) : An auditory representation of ECT-induced seizure. Convul. Ther. 2:125-8.

Weaver, L., Ives, J., Williams,R., and Nies,A.(1977) : A comparison of standard alternating current and low-energy breif puls electro therapy. biol. Psychiat. 12:525-43.

Weaver, L., Ives, J. andWilliams,R.,(1982) : Studies in brief pulse electroconvulsive therapy : the voltage threshold, interpulse interval, and pulse polarity parametrers. Biol. Psychiat.17 : 1131-43.

Weiner, R.(1983) : The rate of stimulus waveform in therapeutic and adverse effecs of ECT. Psychopharm. Bull. 18(2) : 71-2.

Weiner, R. (1983) : EEG related to electroconvulsive therapy. In EEG and Evoked potentials in psychiatry. J. Hughes & W. Wilson (Eds.) ,p101. Butter worth, Boston.

Weiner, R.(1989) : Electroconvulsive therapy. Compreh. Textbook of Psychiat. vol 11, ch. 31(7), pp.1670-8. 5th ed. H. Kaplan & B. Sadock (Eds.) Williams & Wilkins, Baltimore, USA.

Weiner, R., Roger,H., Davidson, J., and Squire,L.(1986) : Effects of stimulus pararmeters on cognitive side effects. In : Electroconvulsive therapy : Clinical and Basic Research Issues. S. Malitz & H. Sackeim (Eds.) Vol.462,p.135, Ann. of NY Acad. of Sci, NY.

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مدة نوبة العلاج الكهربى التشنجى :الفروق فى القياس

تمت دراسة ثلاث طرق لتقدير مدة النوبة أثناء العلاج الكهربى التشنجى . وقد قورنت القياسات البصرية و اللمسية للتشنجات الحركية و القلبية بالاضافة إلى متوسط أتساع تخطيط الدماغ الكهربى بالتخطيط الكهربى غير المعامل أحادى القناه فى سبع و ثمانون جلسة أعطيت لسبعة عشر مريضا. و اختلفت قياسات مدة النوبة بدلالة احصائيه (<٠,٠١ ، <٠,٠٠١). وقد وجدت اختلافات أخرى أكبر ولكنها ليست ذات أهمية اكلينيكية بالنسبة للقياسات البصرية و اللمسية. وكانت الفروق بين متوسطات أتساع تخطيط الدماغ الكهربى المتكامل و تخطيط الدماغ الكهربى صغير وبدت ناتجة عن عملية تشغيل الجهاز. و فى النهاية نوقشت دلالات و مغزى النتائج.