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ESTIMATION OF CORTICAL SURFACE AREA IN SHOCK TREATED GOLGI COX STAINED SLIDES AS A MARKER OF NEUROPLASTICITY

Anatomy

Azra Jabeen* Tutor, Department of Anatomy, St. John's Medical College, Bangalore, India
*Correspondence Author

Roopa R Professor, Department of Anatomy, St. John's Medical College, Bangalore, India

Yogitha R Professor, Department of Anatomy, St. John's Medical College, Bangalore, India

Khaleel N Associate Professor, School of Medicine, Goba Referral Hospital, Madawalabu University, Ethiopia

ABSTRACT

Neuroplasticity is the ability of nervous system to undergo structural and functional re-organization in response to altered environmental input. Electroconvulsive seizures (ECS), is an animal model of electroconvulsive therapy (ECT) known to induce neuroplasticity in hippocampus, amygdala and subventricular zone of adult rats.

The aim of this study was to see if quantifiable changes in surface area (SA) can be detected in slides of ECS treated rats, which can be used as an indicator of neuroplasticity. ECS induced Golgi Cox stained slides of cortical region from 35 sections of high dose ECS treated and 25 sections of sham group were examined under the microscope, photographed and surface area calculated. The mean values for High dose and Sham groups were $6.02 \pm 0.92 mm^2$ and $5.82 \pm 1.35 mm^2$ respectively.

No statistical significant difference was noted in the surface area between the two groups.

KEYWORDS

Neuroplasticity, Electroconvulsive seizures.

INTRODUCTION:

Human brain has the ability to learn, remember, reorganize structurally and functionally through neuroplasticity to adapt and respond to stimuli.[1]

Neuroplasticity is a characteristic phenomenon in the developing brain in which a single neuron has the capacity to modify synaptic connections, change its morphology and functional re-organization.[2] Neuroplasticity is known to occur primarily in hippocampus and sub ventricular zone. The process is regulated by environment, physical activity, genetic, stress, administration of chemical antidepressants, ECT and the like.

ECT is the most effective treatment of choice for the management of major depression, mania, schizophrenia, and drug resistant depression which are associated with changes in the neuronal morphology, synapses and glia. ECT transiently enhances the cerebral blood flow and cerebral metabolism.[3]

ECS is an animal model of ECT. ECS increases the levels of neurotransmitters, neuropeptides, glial cell proliferation, neural sprouting, and synaptic modeling in hippocampus, amygdala, sub-ventricular zone, prefrontal and frontal cortex.[4]

Experimental studies have shown morphological similarities in general cell types of basolateral amygdala and cerebral cortex. Light microscopic Golgi Cox and electron microscopic studies have revealed remarkable similarity of principal projection neuron in the basolateral nuclei of amygdala to the pyramidal cell in the neocortex.[5,6]

The study was taken up to see if quantifiable change in SA of cortex can be detected in slides of ECS treated rats, which if present, could possibly be used to replace the tedious methods which are indicators of neuroplasticity. To the best of our knowledge, no reported studies have been done with regard to cortical SA changes in ECS treated rats. Hence the present study was undertaken.

AIM AND OBJECTIVES:

To observe quantifiable changes in SA of representative cortices in Golgi stained sham and high dose ECS treated rats as a marker of neuroplasticity.

To estimate and compare the SA of representative - primary, secondary, retrosplenial granular and dysgranular cerebral cortices in Golgi stained coronal sections of sham and high dose ECS treated rats.

MATERIAL AND METHODS:

Golgi Cox stained slides containing cortices were taken from the collection of previous work done in the department of Anatomy, St John's medical college Bangalore, in which neuroplasticity was observed in basolateral nucleus of amygdala. The cross sectioned slides were of 120µm thick sections of adult male Wistar rats, 2-3 months old; weighing 175-225gms of six Sham and six High dose (suprathreshold of 60mC, low pulse width of 0.75ms, high frequency of 150pps, stimulus duration of 2.14sec with amplitude of 250mA) ECS treated rats. The sections comprising the areas of cerebral cortex were primary, secondary, retrosplenial granular and retrosplenal dysgranular in the range of -2 to -3 bregma region of rat brain described by Paxinos and Watson C.[14]

INCLUSION CRITERIA:

Golgi Cox stained coronal sections of sham and high dose ECS treated rats

EXCLUSION CRITERIA:

Sections of cortices with irregular and incomplete areas

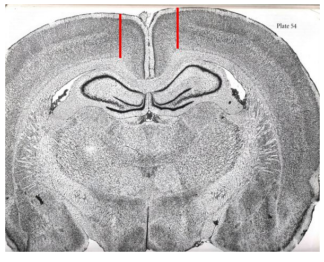


Figure 1: Representative cortices of rat brain at amygdalar complex described by Paxinos & Watson C

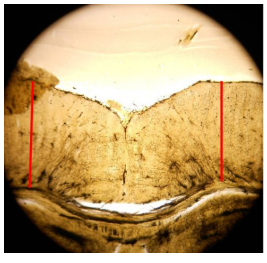


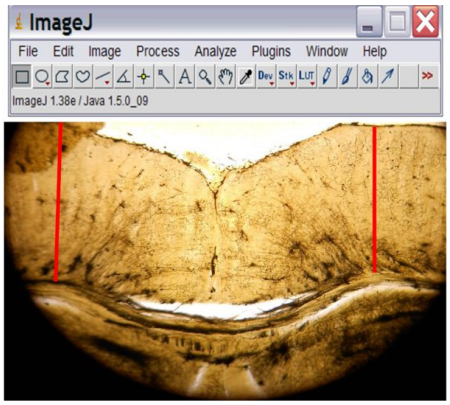
Figure 2: Selected representative cortical area under 4X magnification

MORPHOMETRIC ANALYSIS:

Cortical region from 35 sections of high dose ECS treated and 25 sections of sham group were examined under the microscope with 4X magnification; the cortical region that extends from the median fissures to that of highest elevation of corpus callosum on both sides were identified which comprised of primary, seceondary, retrosplenial granular and retrosplenial dysgranular cortices. A digital camera (16 megapixel Nikon coolpix S2700) was then placed, photographed and transferred to the computer. The area to be calculated were outlined, standardized and SA calculated using software Image J analyzer.

The data available was analyzed for comparison between the two groups.

Figure 3: Representative cortical area with Image J



STATISTICAL ANALYSIS:

Cross sectional analytical study was carried out. Data was analyzed using SPSS version16.02. Mean and standard deviation were calculated. The SA of both groups was compared using Independent sample 'T' test. 'P' value <0.005 was considered significant.

RESULTS:

Table: 1 Comparison of Surface Area between Sham and High Dose ECS treated rats

	Sham (n=25)	High Dose ECS (n=35)	p value
Surface Area (sq mm)	Mean/SD 5.82 ± 1.35	Mean/SD 6.02 ± 0.510	0.510

No significant difference was noted in Surface area between Sham and High dose ECS treated rats.

DISCUSSION:

Depression and pathological stress are associated with changes in neuronal morphology such as shrinkage of dendrites, loss of dendrite spines and synapses with increase or decrease in glia resulting in pathological behavior.[7] Antidepressants may reverse some of these behavior along with stress induced neurohistological changes.[8] ECT is a controversial, non chemical antidepressant and treatment of choice in mania, major depressive disorder and drug resistant patients with depressive disorder.

ECS is an animal model of ECT. ECS seizures induce neuroplasticity and about 88% of newly generated cells co labeled with Neu N were identified, within the hippocampal dentate gyrus compared to 83% in sham treated.[9] These new cells express markers of endothelial cells or oligodendrocytes but not neurons. Proliferation of these cells could reverse the loss of glial cell number and the reduced volume.[10] It is probable that the reversal of reduced volume or expected increase in volume in the cortical region following ECS treatment in our slides, when studied only as surface area, has not shown statistical significance.

Repeated ECS not only induces synaptic changes in the infra limbic prefrontal cortex, but also stimulates long lasting increase in glial proliferation by decreasing the SPRY2 expression[11]; this was another reason for us to expect a change in the measurable cortical surface area. Dose dependent ECS administration has shown neuroplasticity in CA1 region of hippocampus[12], we expected a similar result in the cortical regions. In contrast High dose ECS attenuated dendrite arborization in the basolateral nucleus of amygdala, was apparent even after one month of ECS[13]; since this causes functional improvement apparently, so neuroplasticity should manifest in the cortex also.

Our study did not show the expected statistical difference in values using Independent sample 'T' test, following which log transformation was done. This also is in tandem with the result obtained using Independent sample 'T' test. It is probable that the processing of tissue during Golgi Cox method might have caused shrinkage of the tissue in cortical region, cortex being in the periphery. In our study, the sections comprised were primary, secondary, retrosplenial granular and retrosplenal dysgranular areas of cerebral cortex in the range of -2 to -3 bregma region of rat brain. It is possible that only specific areas out of these many regions studied might have undergone notable change in SA. However in our study we have summed up the above regions for comparing the sham and treated rats. This may be another reason as to why our results have not shown statistical difference.

CONCLUSION:

No statistical change was seen in cortical surface area in ECS treated Golgi Cox stained slides.

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