Desideratum of Electroconvulsive Therapy for the Ministration of Distinct Psychological Infirmity: A Review

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ABSTRACT

Purpose: *"Electroconvulsive therapy" (E.C.T.) is used to treat a variety of mental illnesses. E.C.T. continues to be the most stigmatized psychiatric treatment that is currently available. This is because of fear among the general public due to a lack of knowledge. The main aim of this study was to collect information about an existing topic on electro-convulsion therapy. This may be useful for patient and caregiver education on E.C.T.*

Objective: *The objective of this particular research paper was to offer a good and thorough explanation of the idea of electroconvulsive therapy and its applications in the treatment of various psychological disorders. Another important goal of this work was to seek to remove the social stigma and taboo associated with electroconvulsive treatment by presenting credible sources of scientific clinical data and evidence. The collection of trustworthy data addressing the use of electroconvulsive therapy as a first-line treatment, second-line treatment, and as a last resort treatment modality was another major goal of this article.*

Design/Methodology/Approach: *The results of this clinical study were prepared using information from several credible authoritative reports and journal articles. Secondary source of data has been used.*

Findings/Result: *E.C.T. is a non-drug physiological treatment that has been proven in multiple clinical tests to be a tremendously successful therapeutic modality, especially for depressive episodes but also for psychosis. This is true for both initial and maintenance therapy; in the latter, E.C.T. may be used to prevent relapses. In particular, the safety and acceptability of electroshock treatment have been enhanced through the use of modified activation techniques and improvements in modern anesthetics. Medical conditions that were originally categorical contraindications to E.C.T. have evolved over time to become comparative contraindications. Sadly, although recent research has advanced our knowledge of potential ECT modes of operation and assisted us in creating a safe, well-tolerated therapy, scientists still lack a complete grasp of these underlying mechanisms. Yet, this incredibly potent clinical option shouldn't be withheld, especially from mentally ill individuals who haven't responded to standard treatments. E.C.T. is still an option for the therapy of "treatment-resistant depression".*

Originality and Value: *A new initiative has been launched to enlighten laypeople about electroconvulsive treatment (ECT)/electroshock therapy. All of the clinical information was gathered from reputable, scientific sources, and it was all organized logically in this paper without the use of many medical terms so that readers from all different academic backgrounds could understand the fundamental ideas behind electroconvulsive therapy and how it can be used to treat a variety of psychological issues. To comprehend certain fundamentals of this treatment, theoretical features and the mechanism of action (MoA) of electroconvulsive therapy are also discussed.*

Paper Type: *Clinical analysis and healthcare*

Keywords: Electroconvulsive therapy (ECT), Mechanism of action (MoA), Psychological problems, Theories of electroconvulsive therapy, Adverse effects

1. INTRODUCTION :

Since the sixteenth century, the idea of causing seizures, primarily chemically, to advance psychological health has been around. Italian researchers used electrically produced prophylactic convulsions for the very initial time in 1938. In order to cause a generalized seizure, "electroconvulsive therapy" (E.C.T.) applies electrical stimulation for a limited period of time [1]. Applying relatively short activation methods while being under anesthesia and with muscular immobilization, electroshock treatment (E.C.T.) is indeed the effective production of a succession of generalized epileptic episodes for treatment goals. The person's or the competent judicial observer's complete permission is required. E.C.T. can also be delivered following judicial authorization and the parental agreement of the client's judicial guardians in situations of life-threatening conditions when there exists no chance of gaining given permission because of the nature of the psychological condition [2]. Individuals in conditions such as coma, manic excitation, dazed incoordination, as well as extreme paranoia might not generally be willing to give formal assent, hence alternate permission procedures that fluctuate by province should be followed. Consideration of E.C.T. as a key criterion as well as familiarity with every one of the avenues for appropriate permission for therapy throughout their purview is helpful for clinicians who have the charge of dealing with highly urgent as well as chronically sick psychological patients [3]. Undoubtedly one of the highly successful biological therapeutic methods for a variety of chronic, medicine-resistant, or procedure-resistant mental diseases, including major depressive-disorder (M.D.D.) in European regions and psychosis in Asian nations, is electroconvulsive therapy (E.C.T.). For the past twenty years, ambulatory E.C.T. as a continuing therapy or an isolated urgent program has gained popularity. Despite contemporary advancements in the medication of mood symptoms, there are still significant issues that need to be addressed, such as the time it takes before therapeutic progress may be seen as well as the high percentage of nonadherence and no remission [4]. The nonpharmacological biological intervention known as "electroconvulsive therapy" (E.C.T.) has been shown to be a very successful therapeutic approach, mostly for the management of depressive episodes in addition to the management of psychotic and certain other conditions [5].

2. RELATED WORKS :

Even though being applied to manage a number of mental diseases, "electroconvulsive therapy" (E.C.T.) continues to be the highest successful treatment for all those conditions as well as is still more usually administered to relieve extreme spells of depression [6]. Notwithstanding this, E.C.T. is still the most stigmatized psychological/psychiatric treatment currently accessible, which limits the availability of beneficial care that might save a patient's existence. The clinical depression disorder problem, which is becoming a big worldwide medical issue, may become worse as a result of such stigmatization, and there may also be catastrophic repercussions for specific individuals who might not be provided or may reject a particularly helpful therapy [7]. The elderly people may use E.C.T. at a percentage much larger for a variety of reasons. Furthermore, numerous research has shown that therapy for late-life depressive episodes is not any more helpful than a sham, especially in depressed individuals with cerebral small-vessel disease. Due to age-related physicochemical variations as well as greater susceptibility to psychiatric pharmaceuticals, including antimuscarinic as well as especially over a long-time hypotensive health consequence, older people have a decreased threshold to therapy [8]. The latest anesthetic advancements and the application of improved activation methods have improved the security as well as the acceptability of electroshock therapy. As a result, individuals with increased psychosomatic difficulties can now receive a secure intervention. E.C.T. remains a viable choice, particularly for treating mental diseases that are unresponsive to drug therapies [9]. Early E.C.T. consideration may lower the prevalence of persistent as well as challenging-to-treat mental diseases. E.C.T. may have a lower incidence of problems in older people than in medication. Finally, older individuals with depression frequently respond to E.C.T. treatment greater than younger individuals. compared to younger persons, older individuals experience more neuropsychological abnormalities [10]. Neurobehavioral diseases including cataplexy as well as parkinsonism may benefit from E.C.T. treatment. From the early articles on the topic, several evaluations, as well as conceptual analyses, have revealed the outstanding curative success of this approach in the management of depressive episodes as well as different mental diseases [11]. Nonetheless, despite significant

advancements in the E.C.T. method as well as its use over the past few years, research is presently ongoing to determine the critical neurobiological underpinnings behind the treatment's effectiveness for various mental diseases [12].

3. OBJECTIVES :

This particular paper has been built by keeping the point as a fundamental objective to provide a good and comprehensive understanding of the concept of electroconvulsive therapy and its uses in treating various psychological disorders. The following are the objectives.

(1) To search the mechanism of action of electroconvulsive therapy.
(2) To investigate the impact of electroconvulsive therapy on psychological disorders.
(3) To synthesize information regarding the modern techniques of administering electroconvulsive therapy and its electrode placements.
(4) To report some common side effects of electroconvulsive therapy.

4. METHODOLOGY :

This clinical research output was created using information from several credible, authoritative reports and journal articles. The source of data is secondary. In order to gather information for this paper, data from unreliable, unscientific sources that lacked these qualities were omitted or eliminated.

5. PRESUMPTIVE MECHANISM OF ACTION (MoA) OF ELECTROCONVULSIVE THERAPY (ECT) :

The underpinning fundamental processes that lead to E.C.T.'s better curative results are constantly being researched, despite years of study as well as clinical knowledge improving E.C.T. procedure and practice. The majority of studies examining the neurological consequences of E.C.T. concentrated on the procedure's effectiveness as an antidepressant as well as made that discovery [13]. Neurotransmission pathways that may be implicated throughout the pathophysiology of depressive episodes are notably affected by E.C.T. Many investigations suggested that in line with the "monoamine deficit theory" for depression. Serotonin as well as nor-adrenergic signaling is reduced by E.C.T. Nevertheless, research on rats has shown contradictory conclusions, including such increased "presynaptic hippocampal serotonin (5-HT)1A receptor" sensitivity but also reduced hippocampus 5-HT1A channel sensitivity following electroconvulsive shocks (E.C.S.) in mice. Nonetheless, E.C.T. has been demonstrated to raise plasma concentrations of tryptophan in individuals with significant depression indicating that a greater supply of such serotonergic progenitor may help explain some of the medicinal benefits of E.C.T. [14].

In contrast, researchers have proposed that one potential process of E.C.T. involves a balancing enhancement in G.A.B.A synaptic transport. Research using "proton magnetic resonance spectroscopy" revealed that depressive individuals receiving E.C.T. have higher occipital brain G.A.B.A levels, which is consistent with both the anti-convulsant impacts of E.C.T. as well as the G.A.B.A-deficit theory of depressive episodes. In addition, research using "single-photon emission computer tomography" (S.P.E.C.T) revealed that E.C.T may work through increasing G.A.B. Aergic synaptic transport [15]. This is additionally supported by contemporary research suggesting E.C.T. increases the functioning of inhibition circuitry in the mammalian motor cortex. E.C.T. significantly alters G.A.B. Aergic communication between neurons.

The "hypothalamic-pituitary-adrenal" (H.P.A) axis has been linked to the medicinal benefits of E.C.T. alongside its impact on neural circuits. Among the popular sustained physiological research results in clinical depression ailments is a metabolic derangement of the H.P.A-axis, which also includes heightened concentrations of corticotropin-releasing hormone (C.R.H), adreno-corticotropic hormone (A.C.T.H), as well as corticosterone all through bouts of depression and returns to baseline after complete recovery [16]. During E.C.T., transient increases in plasma values of A.C.T.H. and cortisol have also been noticed, which may be regarded as a biological distress reaction. Yet, it has been discovered that plasma concentrations of A.C.T.H. and cortisol drop after E.C.T., indicating that decreased expression of the H.P.A- axis may be an E.C.T. therapeutic benefit for severe depressive episodes [17].

Several pieces of information have recently come to light suggesting that neuroactive chemicals, particularly affect synaptic responsiveness through nongenomic pathways, may play a part in the underlying causes of depressive episodes and may support the restorative benefits of pharmaceuticals.

Increased plasma rates of "dehydroepiandrosterone sulfate" (D.H.E.A.S), a powerful and effective deleterious regulator of the G.A.B.A -A transcription factor, have now been reported in psychopathically depressed clients populations as well as they were connected to nonadherence to E.C.T. in these patient populations, despite no changes of beneficial G.A.B. Aergic 3-oc- reduced neuromodulators stimulants having been discovered in depressed patient populations after intervention with E.C.T [18]. As a result, researchers have proposed that D.H.E.A.S. plasma concentrations could act as a signal for E.C.T. non-responsiveness. A medicinally mediated drop in D.H.E.A.S concentrations may act as a possible approach to reestablish the therapeutic outcome in depressed individuals who are susceptible to E.C.T. since D.H.E.A.S pre-treatment reduced the therapeutic benefit of E.C.S. in a hereditary rat clinical model of depressive disorder [19].

In the pathogenesis as well as therapeutic interventions of clinical depression diseases, there is mounting scientific proof that downstream signal transcriptional processes, of that kind as the "cyclo-adenosine monophosphate" (cA.M.P) "cAMP responsive element binding protein" (C.R.E.B) cluster, play a significant function. These channels also have an impact on neuroprotective elements like "brain-derived neurotrophic factor" (B.D.N.F) [20]. Throughout this regard, in vivo as well as mouse research showed that the hypothesized effects of E.C.S. on proliferation as well as neuroplasticity could be responsible for its antidepressant activities. Researchers have demonstrated that a solitary E.C.S increases B.D.N.F as well as "tyrosine kinase B" (Tr.k.B), a B.D.N.F responder, and m-RNA levels.

Additionally, as contrasted to the results of pharmaceutical antidepressant therapy, B.D.N.F m-RNA, as well as Tr-kB m-RNA is consistently raised following an E.C.S session. Besides that, some investigations have found that E.C.S. improves synaptic connection. Frequent E.C.S. promotes the formation of scattered fibers in the hippocampus as well as adjacent cerebral structures that include the amygdala as well as frontal sections. Moreover, E.C.S. causes an elevation in neuronal production in the hippocampus, an outcome that was initially apparent following a solitary E.C.S. but was significantly highly apparent following a sequence of "PX1S", indicating a dose-dependent function of E.C.T. on neurodevelopment [21]. Moreover, following E.C.S., higher concentrations of C.R.E.B. and improved production facilitated by C.R.E.B were seen in the hippocampal region of experimental laboratory mice.

In frontal regions, there was a temporary reduction in the flow of cerebral blood (C.B.F.) following E.C.T., according to S.P.E.C.T investigations in depressed individuals. C.B.F. has been demonstrated to rise and thereafter normalize in depressive individuals following a session of E.C.T., in counter to these initial impacts [22]. On receiving E.C.T., depressive individuals' frontal as well as parietal brains, as well as the "posterior and anterior cingulate gyrus", showed reduced local cerebral glycemic consumption. Aiders showed lower frontal cerebral sugar uptake than non - responders, indicating that the lower glucose consumption may help explain some of the medicinal benefits of E.C.T. Despite our growing understanding of the impact on the central nervous structure, we still don't fully understand the individual or combination of processes that are essential for E.C.T.'s treatment success in treating a variety of mental diseases [23].

6. IMPORTANT THEORIES OF ELECTROCONVULSIVE THERAPY (ECT) :

In an effort to comprehend how E.C.T. works therapeutically, several neuropsychological, psycho-analytical, as well as neurobiological explanations have been proposed. According to the impact of E.C.S. on several elements (operational, compositional, as well as morphological) of the cortex as well as underpinning neural and glial structures, physiological hypotheses offered can indeed be roughly categorized into neurobiological, neuro-biochemical, and neurodevelopmental theories [24]. This part sought to comprehend the changes in physiological theories describing E.C.T.'s modes of operation.

6.1 Neurophysiological Hypotheses :

By changing their intracellular electrochemical environment as well as ionic level, the electric power activity from E.C.T. electrodes stimulates cortex cells called neurons by passing across an intermediate tissue. The idea has been proposed that convulsions, which are produced when several depolarized cells activate at once and cause a jolt, can treat a variety of neuropsychological illnesses. The specifics of the convulsions, the location of the electrodes, and client characteristics are what determine which regions of the cortex are engaged. Important cerebral regions affected by generalized seizures include the "limbic system, thalamus, basal ganglia, cortex, and sub-cortex".Yet specific parts of the cortex are

visibly significantly affected than others. Notwithstanding these variations, the "Postictal Suppression Index" (P.S.I.) as well as "Burst Suppression (B.S.) index" have also been found to be reliable indicators of how well E.C.T. would reduce convulsions. The B.S. index was described by experts as a sign similar to P.S.I. The B.S. index decreases with several E.C.T. sessions, reflecting E.C.T.'s anti-convulsant impact. alterations in localized metabolic as well as blood circulation in the brain Employing radiological methods such as "positron emission tomography" (P.E.T.), "single photon emission computed tomography" (S.P.E.C.T.), as well as "functional magnetic resonance imaging" (f-M.R.I), several investigations have demonstrated that E.C.T. alters cortical blood circulation as well as glucose metabolism activity of the brain. Following the convulsion generalization pattern, "regional cerebral blood flow" (r-C.B.F.) has a tendency to spike rapidly in the regions of the cortex experiencing convulsions events. Recurrent convulsions cause larger fluctuations in r-C.B.F. than skipped ones, and the differences in r-C.B.F. differ depending on the kind of episode. Immediately following E.C.T-induced convulsions, r-C.B.F. starts to drop. The idea that E.C.T. alters r-C.B.F, as well as localized metabolic activity, is supported by a vast body of evidence. Patients with depressive episodes have been shown to have post-E.C.T. reductions in r-C.B.F. and glycogen utilization in the brain, especially in the "superior frontal regions, temporal cortex, as well as the dorsolateral and medial prefrontal cortex", as well as a boost in flows all across "amygdala, para-hippocampal gyri, pons, and limbic or para-limbic structural features" [25]. Yet, several researchers present conflicting results in this area. Moreover, no discernible variation in blood circulation was detected. On the other hand, completing an E.C.T. course, elevated r-C.B.F. has also been noted. Right temporal as well as bilateral parietal cortices with S.P.E.C.T. revealed higher combustion. The majority of the aforementioned data point to significant alterations in r-C.B.F. after E.C.T. in individuals with severe mental illnesses. The combustion in various cerebral regions is linked to these changes in r-C.B.F, which are associated with treatment results.

6.1.1 Alterations in the Blood-Brain Barrier (BBB) :

When elevated blood pressure takes place throughout the ictal period of ECT-induced convulsions, the "blood-brain barrier" (B.B.B) might momentarily disintegrate around this period. Specific neurochemicals could be delivered from the bloodstream to the brain parenchyma throughout this activity, causing particular alterations in the microclimate of the nervous system, including elevated amounts of the "brain-derived neurotrophic factor" (B.D.N.F), revascularization, as well as proliferation. Only following several applications of E.C.S. does the B.B.B. become compromised [26]. Likewise, investigations on animal experiments show that the shift in B.B.B permeability may not be connected to the alterations in the cortex that occur after a single sitting of electroshock therapy stimulation.

6.1.2 Changes in electroencephalogram (EEG):

The cerebral cortex's operational stability can be seen in electroencephalography. E.E.G. alterations in particular cerebral regions throughout as well as afterward E.C.T. may be used to forecast therapeutic outcomes and to better comprehend the physiological processes that underlie the procedure. There aren't many reports linking E.C.T.'s curative effects to alterations in the E.E.G. The most frequent E.E.G. observation has been a weakening of the pattern, which may be related to the medical result. Independent of the E.C.T. modality, recent research points to post-E.C.T. delta activation in the prefrontal cortex as a determinant of therapeutic responsiveness. The rate and degree of E.E.G. wave attenuation determine when the treatment reaction starts to occur.

The ictal E.E.G. alterations on bilateral E.C.T. are observed to be highly coordinated, proportional, and homogeneous with strong amplitude, as well as have strong post-ictal reduction (in contrast to single E.C.T.). Many varieties of depressive episodes have varied E.E.G. patterns. After the use of E.C.T., these variations in the E.E.G. features disappear. In the fronto-temporal region, there was less alpha activation after a sequence of E.C.T. treatments, according to contemporary research. In some brain regions, there was also a documented rise in theta (4 to 7 Hertz) activation. " Sub-genual anterior cingulate cortex" (Sg-A.C.C), as revealed by low-resolution electro-magnetic tomography studies, was the location of brainwaves. In individuals with psychotic depressive symptoms, increased theta production has been demonstrated to be correlated with a decrease in psychotic characteristics [27]. Moreover, the antipsychotic effect of E.C.T. was favorably correlated with the degree of reduced excitability in the S.g-A.C.C. before therapy.

6.2 Neurobiochemical Hypothesis:

The neurotransmission cascade is modulated by E.C.T., which also affects the production and discharge of a wide range of neurotransmitters in the cortex, comprising transcriptional regulators, various neurotransmitters, neuroprotective factors, as well as enzymes. It affects the movement of nearly each of the brain's key chemicals, including "serotonin, dopamine, glutamate, innate opioids, adrenaline, nor-epinephrine", and others [28]. It functions at several different stages throughout the biochemical neuronal activity, involving neurotransmitter production, secretion, attachment to sites, and recapture.

6.2.1 Changes in genetic structures:

Several target genes' expression was shown to be changed in mouse studies after receiving both single and repeated electroconvulsive impulses. These genes may represent different regulatory elements, architectural proteins, and brain-specific neuromodulators. For example, post-E.C.S. gene expression changes in the mouse brain for the genes "c-Fos", "Egr-1", "Neuritin-1", "BDNF", "Snap-29", "Synaptotagmin-III", "Synapsin I", "Psd-95", and "Npy". Parallel to this, experts found that individuals with dazed psychosis who had recurrent E.C.T. had considerably higher levels of the specific gene as "transcription factor 7" (TCF-7), as shown by sequencing screening of m-RNA taken from the peripheral circulation. It has additionally been proposed that E.C.T.'s treatment benefits can be explained by the chromosomal changes it causes [29]. The "gene Gadd-45b (growth-arrest and DNA-damage-inducible protein-45 betas)", which plays a responsibility in demethylating the control areas of the genes for "fibroblast growth factor 1" (F.G.F-1) as well as BDNF, two components implicated in the control of regeneration, has been shown to be activated by electroshock stimulation. In developing "hippocampal dentate gyrus neurons", Gadd-45b facilitates E.C.T.-induced dendrite growth. The hippocampus's synaptic connection is enhanced by electroshock therapy seizures. " Extracellular signal-regulated kinase 1/2" (E.R.K- 1/2) as well as "striatal enriched protein tyrosine phosphatase" (S.T.E.P-61), that further dephosphorylates tyrosine from the "N-methyl-D-aspartate" (N.M.D.A.) binding site and causes the effectors to internalize, are two examples of particular enzymes whose effect is expanded in response to a solitary electroshock stimulation. The rat hippocampal region has undergone this biological alteration. E.C.T. results in chromosomal remodeling in a number of ways. The "down-regulation of c-fos" and the "up-regulation of B.D.N.F" may act as a mediator for the restorative results [30]. The participation of the "histone deacetylase enzyme", which results in post-transcriptional alterations that downregulate N.M.D.A. "receptor signaling pathways" and cause the results of E.C.T., is explained by some other regulatory process.

6.3 Neuroplasticity and Electroconvulsive Therapy:

Changes in the density of cerebral regions have frequently been linked to mental diseases. Moreover, research has shown that E.C.T. causes modifications in the thickness of the entire cortex and its constituent parts, including the grey-matter, and white-matter, along with other brain regions. In regions with stronger connections to the prefrontal-cortex as well as other limbic structures implicated in emotional processing, such alterations are much more apparent. E.C.T. causes neurobiological adaptations in "glial cells" including their operations, as well as the "synapse", "neurons", "dendrites", and "vasculature", including their mechanisms ("synaptogenesis, neurogenesis, dendrogenesis, gliogenesis, angiogenesis, " respectively). As soon as one electroshock stimulation has occurred, synaptic plasticity alterations have been seen. Within minutes of administration, E.C.S. increases glial stimulation indicators in rats. The neurogenesis impact of E.C.T. in people with M.D.D. has been the subject of much investigation. Besides that, E.C.T. has been shown to significantly alter the morphology of some brain regions, including the hippocampal region, amygdala, posterior cingulate gyrus, as well as mid and superior temporal cortex [31]. According to Magnetic resonance research, individuals with Depressive disorders whom had E.C.T. experienced an increase in hippocampus thickness (normalization) following the treatment. Investigators found that, as opposed to a modification in hippocampus structure, the effects of ECT as well as the alleviation of symptoms are related to an enhancement in hippocampal operational connectivity/ functional connectivity (F.C).

7. MODERN ELECTROCONVULSIVE THERAPY (ECT)SYSTEM :

Professionals have implemented several adjustments to E.C.T. since it was initially administered in the 1930s to increase its usefulness as well as security [32]. Frequency specifications may be adjusted,

the dose can be tailored to each patient's convulsions threshold, as well as electrode location can be modified.

7.1 Pulse Width:

Often the E.C.T. equipment employed nowadays contains a constant current output modulator that permits uninterrupted current control. The standard unit of measurement for the overall charge is "millicou-lombs" (m.C). One waveform element that is frequently changed in E.C.T. administration is "pulse width". The majority of studies favor using many "brief or ultra-brief pulses" (0.5-2 milliseconds) as opposed to typical sine waveform E.C.T. dosage since it results in higher charge effectiveness as well as reduced negative consequences. Since it concentrates the impulse on parts of the brain that govern emotion while minimizing stimulation of brain structures engaged in mental function, adopting a shorter or "ultra-brief pulse width" promotes therapeutic effectiveness as well as reduces adverse consequences [33]. After three days of therapy, a client's intellectual function may restore to standard ranges with brief-pulse stimulation. A growing body of research shows that expanding the number of strokes delivered with small pulse duration as well as intensity improves the anti-depressant benefits of E.C.T. while minimizing its negative neurodevelopmental adverse reactions.

7.2 Understanding the Dosage and Duration:

The degree by which an electronic stimulation surpasses a person's seizure cutoff point minimal electric power discharge required to cause a widespread CNS seizure- is greater significant for therapeutic effectiveness than just the stimulus's actual amplitude. The positioning of the electrodes determines how much the stimulation must surpass the convulsions cutoff [34]. Individuals in brief psychotherapy generally undergo two to three sessions per week, for a total of twelve to eighteen sessions. The proportion of treatment response to the intensity of intellectual adverse reactions determines the ideal range of treatments to be given.

8. PLACEMENTS OF ELECTRODES :

Familiarity with a wide range of psycho-pharmaceuticals, involving multiple drugs from every category, is necessary for competency in treating depressive episodes. Similarly to this, E.C.T. proficiency requires familiarity with all 4 of the current electrode locations. E.C.T. electrode positioning options encompass bifrontal as well as "left anterior right temporal" (L.A.R.T.) deployments in addition to conventional bilateral as well as "right unilateral placements". Experimental studies have found few statistically significant distinctions across antidepressant medications, as well as use variances are far further uncommon. Similar to this, a few other variations in E.C.T. electrode positioning have also been demonstrated, and the majority of documented variations may be explained by significant variations in electrical stimulation dose [35].

8.1 Right unilateral electrode placement:

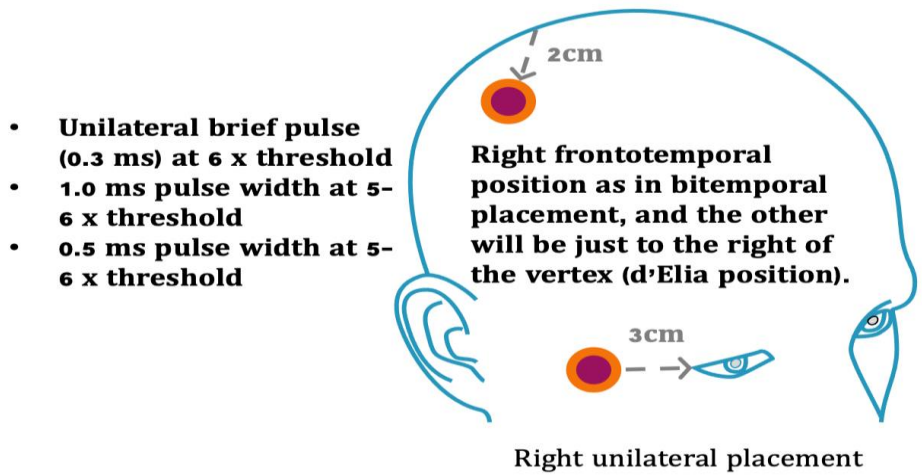


Fig 1: Shows the positioning of a right unilateral (RUL) electrode placement [36].

Right unilateral" (R.U.L.) electrode insertion serves as the greatest common first therapeutic option, with the exception of the sickest individuals. Relying on observations that decreased stimulation amplitude results in poorer rebuttal or remission levels, R.U.L. E.C.T. is now delivered at many variations of the "seizure threshold" (S.T). Several individuals may undergo a solitary minimal R.U.L. E.C.T. treatment as well as additional procedures with stimulation at a double of S.T. as a portion of an introductory dosage calibration to identify S.T. [37].

8.2. Bitemporal electrodplacement :

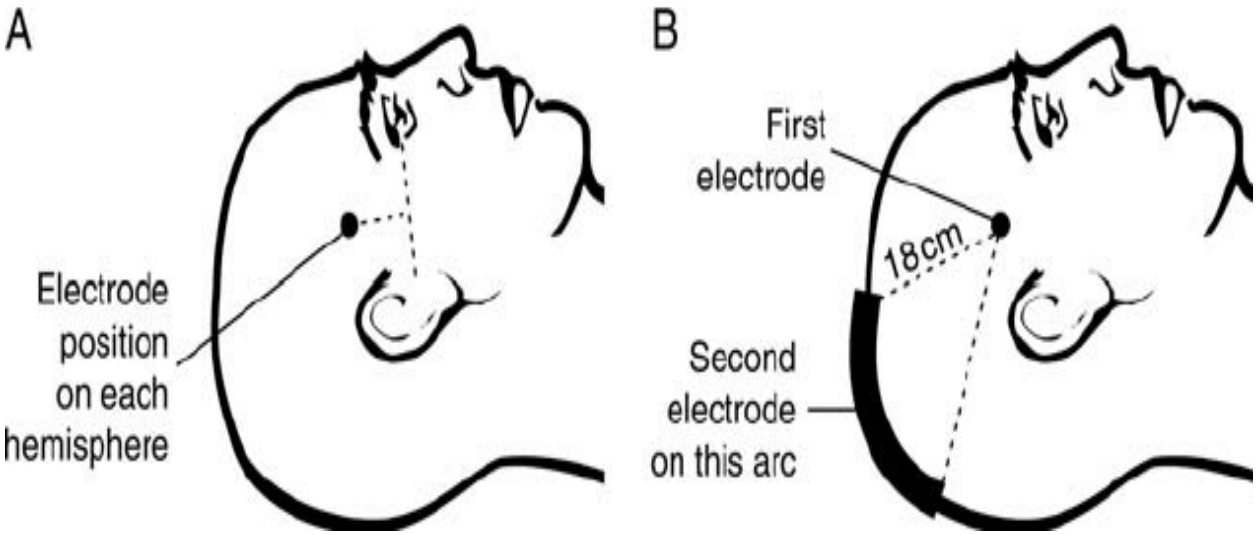
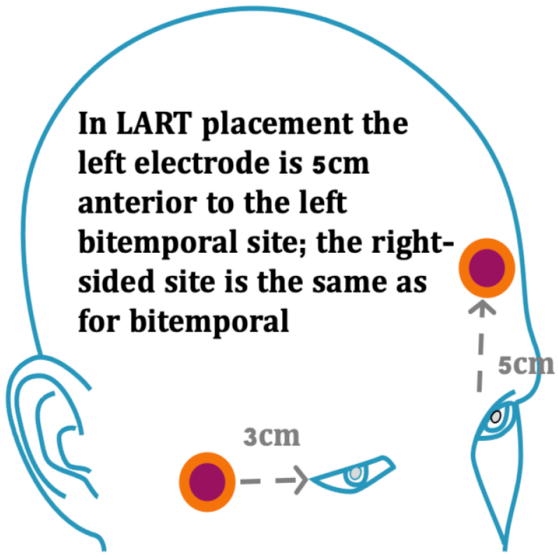


Fig 2: Shows a bitemporal (BT) electrode placement position [38].

8.3 LART eletrod placement:



Left anterior right temporal (LART) placement

Fig 3: Shows a left anterior right temporal ECT electrode placement location [36].

8.4. Bifrontal electrode placement:

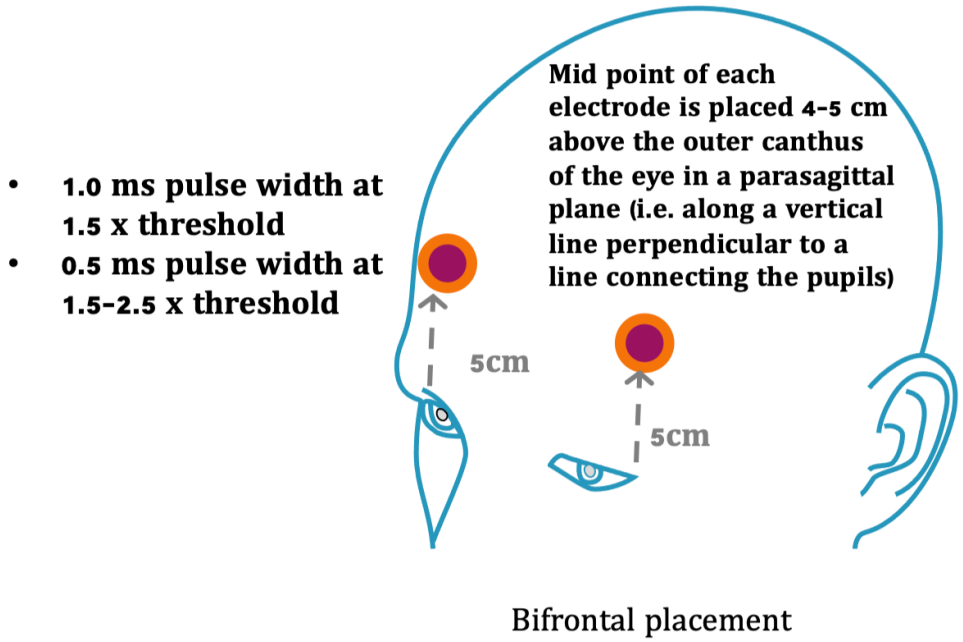


Fig 4: Shows a bifrontal placement of ECT electrodes [36].

The greatest outcomes in E.C.T. research clusters have been described as maximal therapeutic progress as well as a complete treatment in an average of six to eight rounds, cessation in at minimum 80 % of individuals, and sustained responsiveness such that recurrence between four to six weeks appears in less than 20% of disbursing clients. Taking into account indications of concomitant illnesses with depressive problems, such as anxious diseases, personality abnormalities, somatic illnesses, and abnormalities related to alcoholism and other drug misuses, the degree of recovery should really be substantially full [39]. Return that takes place after six weeks following E.C.T. appears to be connected to post-E.C.T. prophylaxis as opposed to initial E.C.T. treatment.

The main cause of missing these effectiveness objectives is electromagnetic under-dosing. Every electrode location might be under-dosed, resulting in mediocre efficacy. With typical bilateral deployments, also known as bi-front-temporal or bi-temporal, right-unilateral, as well as bi-frontal positions, poor efficacy due to under-dosing has now been documented. An investigation that under-doses a deployment doesn't always reflect proper therapeutic practice, which means that as a result, it is unable to adequately evaluate the placement's usage in therapeutic settings. Several investigation findings indicated poor efficacy for one or even multiple therapeutic techniques however failed not to take into consideration how the minimal performance might be explained by the weak shock dosages employed [40]. The frequency of ECT treatments required to achieve remission rises with milder or infrequent under-dosing, and stringent under-dosing further lowers the clearance ratio.

On the one extreme, irrespective of electrode location, reduced stimulation dose results in a less intellectual impact. On the contrary, abusing too much might make any placement's benefits to cognition irrelevant. There is a way to alter the dosage. Higher electric dose biologically results in increased neuron depolarization or epileptic foci. This must result in more generalization of the convulsions across the cortex, which is superior E.C.T.The intensity of epileptic foci among the 2 stimulation electrodes increases as well as the minimum power necessary to cause an episode decreases when the 2 wires are spaced farther apart [41]. The baseline convulsion-inducing current is 40 to 50% greater and indeed the poles are spaced farther away in bi-temporal E.C.T. than in right-unilateral E.C.T. In contrast, right-unilateral E.C.T. is simpler to under-dose versus bi-temporal E.C.T. since seizures may be brought on with less electrical stimulation.

9. ADMINISTRATION OF ANESTHESIA AND PREPARATION OF PATIENT :

The basic criteria of temporary amnesia and muscular relaxation are also included in the anesthetic requirements for E.C.T., together with the regulation of these hemodynamic alterations and associated consequences. The dose of anesthesia should not be so high that it unduly suppresses the convulsion activity that the medication is intended to prevent, despite the fact that they are necessary [42]. The doctor has to have a solid understanding of how to maintain anesthesia for E.C.T.

9.1 Pre medications:

In order to counteract the early parasympathetic release, anticholinergic medications like "glycopyrrolate & atropine" are frequently used. " Glycopyrrolate" is preferable and is given intravenously soon preceding administering the induction drug, or intra-muscularly at least three minutes beforehand the planned treatment. Because of the "vagal effects" of E.C.T., this could lessen the incidence as well as the intensity of bradycardia or cardiac asystole as well as the danger of suction. Nonetheless, regular antispasmodic treatment has now been deemed unneeded.

These could be especially beneficial for individuals on beta-blockers or other sympathetic-blocking medications, or for those for whom a convulsion threshold has never generally been determined. To reduce the sympathetic reactivity to an increase in systolic bp as well as pulse rate, beta-blockers like esmolol or labetalol can indeed be administered. But only after a thorough assessment of each client's cardiovascular vulnerability should they be taken into account. Esmolol and labetalol have different effects on the length of seizures. Alpha 2 agonists as well as calcium channel blockers may additionally be employed. Hypertension can be controlled without influencing seizure length by taking clonidine as well as dexmedetomidine ten minutes prior to induction of anesthesia [43]. Drugs like lithium that worsen morbidity must be evaluated, as drugs like benzodiazepines/ anticonvulsants reduce the effectiveness of E.C.T.

9.2 Oxygenation of the patient :

By using unaltered E.C.T., several individuals developed hypoxia, cyanosis, as well as disruption of sphincter function. Less oxygen is needed when improved E.C.T. is used together with muscular relaxation. Yet, throughout the convulsion, brain oxygen demand rises by about two hundred percent. Thus, it is often advised to supply the lungs with 100percentage oxygen at a frequency of fifteen to twenty inhalation per minute starting about one minute well before inducement and then continuing until the restoration of voluntary respiration [44]. The convulsions may last longer if hyperventilate.

9.3 Induction agents:

Memory loss for the short electrical excitation as well as the muscle-relaxing agent's activity is given by induction medications. According to the person's physiological features, a number of induction medications may very well be utilized. An excellent induction medication would not compromise with convulsions amplitude or frequency and would possess a short wavelength with quick commencement and recuperation. And since its accessibility at the period, "sodium pentothal" was the initially available induction agent utilized then. " Methohexital", a much more recent barbiturate, gained popularity following its creation. It is regarded as the "gold standard" and has been the foremost often prescribed general anesthetic during E.C.T. for several years [45]. Due to their absence of antiepileptic qualities, "ketamine" as well as "etomidate" may seem superior to certain other medications; nevertheless, other factors such as drug tolerability must be taken into account. Substitute induction agents that confront the drawbacks of various agents include the "ketofol-dex" combination and the "ketofol-propofol" combo.

10. BASIC EQUIPMENT AND INSTRUMENTS NEEDED TO ADMINISTER ELECTROCONVULSIVE THERAPY (ECT) :

The usual devices recommended by the "American Society of Anesthesiologists" (A.S.A) must be present in the E.C.T. treatment as well as recovery rooms. A good quality stethoscope, electrocardiography (E.K.G) device, BP gauge, pulse-oximeter, suction device, and oxygen distribution equipment have to be available. In addition to ventilatory including CPR equipment, proper anesthetic induction systems, CPR devices, and medicine should really be supplied. A nozzle face mask, nerve stimulant to check for the neuromuscular blockade, an electro-myograph

(E.M.G), electro-encephalography (E.E.G) electrodes, several blood pressure devices, as well as a nasal tube or gas masks should indeed be supplied [46].

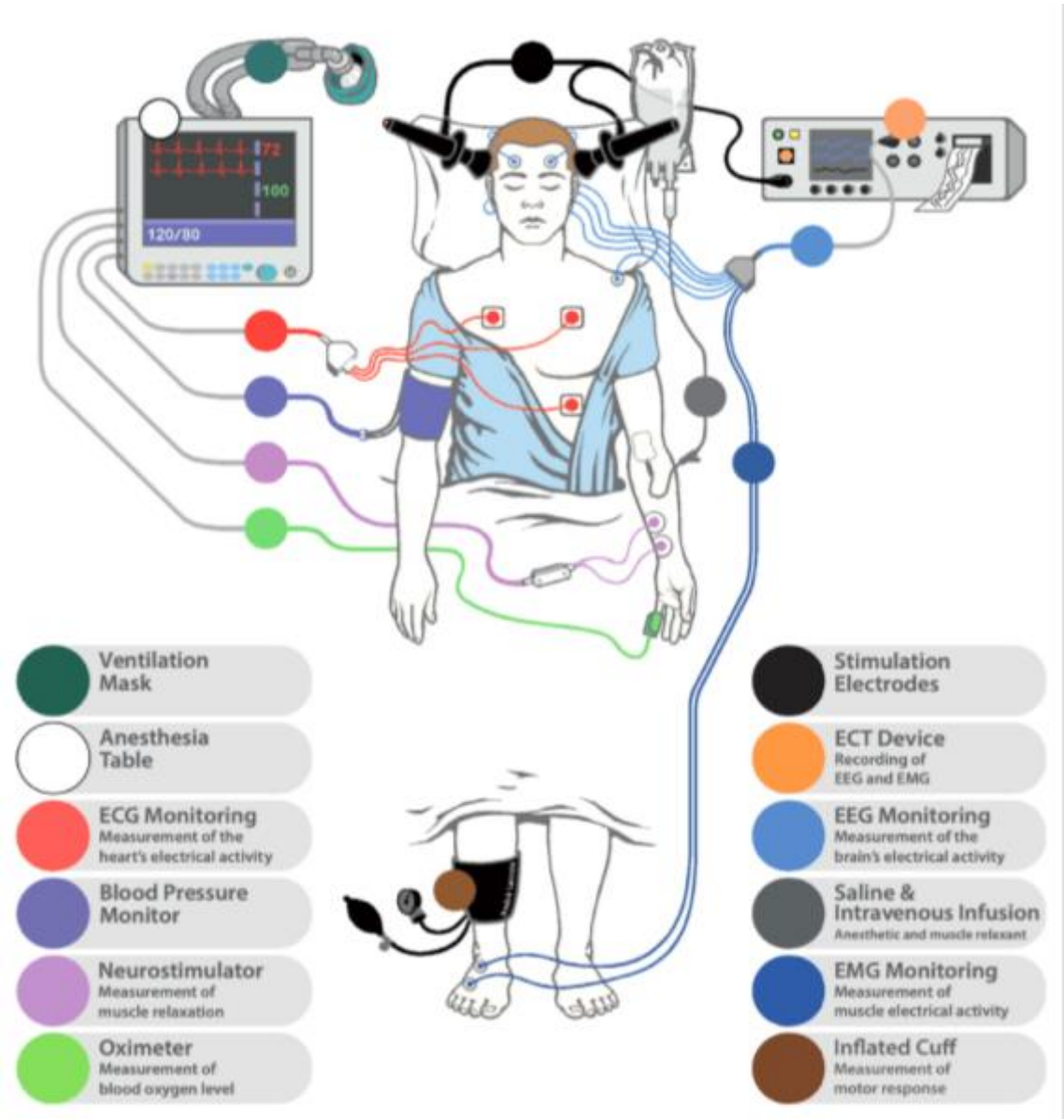


Fig 5: Showing a picture of the ECT preparation [47].

11. CLINICAL INDICATIONS :

E.C.T. comprises repeatedly delivering electrical impulses to the brain with the goal of reducing the signs of a particular psychological condition. Almost 90 percent of individuals who seek E.C.T. therapy have a severe depressive disorder indication. The initial ailment for which E.C.T. was being administered, schizophrenia, along with catatonia & severe mania, are still treated with it, albeit rarely often. Although it must only be taken into consideration in patients who do not react to other, relatively conventional therapies, it is also advised for the management of a few clinical disorders. These might comprise "neuroleptic malignant syndrome", uncontrollable epileptic abnormalities, and recalcitrant Parkinson's condition, notably with "on-off" phenomenon, such as significant, erratic motor oscillations. E.C.T. is regarded as a first-line intervention when health care or psychological variables demand a quick as well as effective clinical reactivity when E.C.T. involves very little hazard to a person than medicines, such as in pregnant or geriatric patient populations if there is a proven record of prescription drug impedance or a history of a favorable reactivity to E.C.T., or in cases where the person prefers E.C.T. to pharmacological intervention [48].

12. ELECTROCONVULSIVE THERAPY (ECT) AND MAJOR DEPRESSIVE DISORDER (MDD) :

ECT has a proven track record of success in treating severe depressive episodes. Results from comparison studies revealed that ECT has stronger antidepressant benefits than just about any other pharmaceutical medication, comprising "monoamine oxidase inhibitors" (MAOIs), "tricyclics", as well as "serotonin reuptake inhibitors" (SSRIs). A substantial collection of research suggests that ECT, even now in extreme old age (>85 years of age), is a successful as well as secure therapy approach for geriatric individuals with serious depression. When opposed to younger individuals, the effectiveness of ECT is noticeably higher in elderly people [49]. A significant follow-up survey's longevity evaluation revealed, from the standpoint of long-term management, that elderly people with serious depressive episodes who got ECT survived longer as well as experienced more therapeutic recovery than those who only underwent medication.

12.1 Treatment-resistant depression (TRD):

A rise in the quantity of TRD individuals receiving ECT referrals might have an impact on how well the procedure works in terms including either short-term responsiveness as well as recurrence percentages. Whereas past research claimed ECT clearance ratios of 80% to 95%, the population cohorts currently receiving ECT treatment increasingly comprise individuals who did not react to sufficient antidepressant treatments (i.e., patients with TRD). Although there is some disagreement over what constitutes "treatment resistance," there is growing evidence and thus growing focus that the failure of depressed individuals to react to a sufficient volume of antidepressant drug regimens might promote therapy resistance as well as comorbidities [50]. Given the possible decline in individuals' psychological adjustment, the rising rate of treatment opposition creates substantial therapeutic concerns.

12.2 Melancholic depression:

"Melancholic depression" is a severe kind of major depressive disorder that is associated with a significant likelihood of hospitalization as well as the lack of ability to experience enjoyment from pleasant stimulation. With late-onset serious depressive episodes (>60 years old), it is frequently observed. Investigators have looked into whether melancholic sadness reacts to ECT uniquely from those various affective illnesses because of its distinctive medical characteristics. Scientific papers as well as series revealed that melancholy characteristics might forecast a successful ECT result [51]. However boosting stimulus strength may result in a significantly quicker commencement of responsiveness in ECT-naive individuals, ECT had no effect on the amount of change in melancholy symptoms or the quantity of ECT needed to obtain a treatment reaction in a comprehensive randomized study.

13. ELECTROCONVULSIVE THERAPY (ECT) AND MANIC DISORDER :

ECT was the preferred method of therapy for manic conditions prior to the advent and greater accessibility of pharmaceutical medications, notably lithium. In the therapy of both episodic and "treatment-refractory mania", ECT has consistently demonstrated effectiveness. It has been more generously mentioned that "electroconvulsive treatment is considered as the most efficient and effective therapeutic approach for mania, commonly elected (as well as anecdotal experience found to be particularly effective) when other methodologies have started to fail, it must be regarded in sufferers admitting this intervention and who have not reacted to preceding prescription medications" [52]. The prospective studies' results and comparative studies mentioned were indeed analyzed along with the previous literature for the A.P.A recommendations, which affirmed that E.C.T. has typically been used as a last resort for sufferers who have failed to respond to drugs due to the accessibility as well as simplicity of psychopharmacologic and anti-epileptic substances. Researchers also highlighted that, which is also the situation with various depressive illnesses, individuals who are drug resistant react to E.C.T. at a lesser frequency than any of those who require it as the very first therapy. In conclusion, E.C.T. as well as psychiatric drugs carry the potential in boosting the pace of recuperation as well as sustaining well-being over a lengthy period, despite the lack of thorough studies in this field, similar to schizophrenia [53]. More investigation is necessary, especially into the therapeutic aspects of mania

that seem to respond best to E.C.T. as well as the specifics of E.C.T. that seem to work best for managing manic episodes.

14. SCHIZOPHRENIA AND THE USE OF ELECTROCONVULSIVE THERAPY (ECT) :

Despite severe depressive episodes being the indication over which E.C.T. is currently very commonly advised in India as well as numerous different western countries, investigations on the efficacy of E.C.T. for the management of people with schizophrenic disorder have consistently been undertaken. Among the most crippling mental conditions, schizophrenia affects 3% to 5% of the planet's demographic populations and roughly 1.8 million adults in India. The A.P.A journal discusses a few of the studies reviewed from the early findings on the effectiveness of E.C.T. in treating schizophrenia. Further subsequently, conflicting suggestions from a variety of diverse bodies have served as an embodiment of the debate about E.C.T.'s effectiveness in the management of schizophrenic disorder. The "American Psychiatric Association's" recommendations are among the best. According to them, "The development of potent antipsychotic drugs dramatically decreased the use of E.C.T. in individuals with schizophrenia. Nonetheless, E.C.T. is still a crucial treatment option, especially for schizophrenic individuals who do not benefit from drug therapy".Some rebuttals to these suggestions have challenged their findings as well as provided proof in favor of the employment of E.C.T. in conjunction with psychiatric drugs for the management of individuals with "treatment-resistant schizophrenia" [54]. Moreover, they have backed the application of E.C.T. (C-E.C.T. and M-E.C.T.) for stabilization as well as a continuation in this group. It has also been explicitly noted that E.C.T. should only be used in extreme situations of treatment-resistant schizophrenia, with the exclusion of catatonia, as no benefits have really been persistently shown when contrasted to pharmacological therapies. It is clear that using E.C.T. as a kind of intervention for schizophrenia has traditionally been fraught with technical inaccuracies that continue to be debatable. Even though there is still disagreement over whether E.C.T. is effective in handling this crippling condition, it is clear that new studies have produced encouraging results, especially in light of the notable medical advancements attained when E.C.T. is used to supplement an already prescribed prescription drug regimen [55]. Current data from experimental research as well as meta-analyses show why these arguments are still being debated but also suggest that E.C.T. may have a function in schizophrenia therapeutic plans.

15. USE OF ELECTROCONVULSIVE THERAPY (ECT) FOR THE TREATMENT OF BIPOLAR DISORDER :

“Lithium” is the earliest as well as the most reliable mood preservative for bipolar illness, therefore medication is the first-line management for the condition. Yet, contrasted to young people, older adults have a lower threshold for lithium. " Bipolar disorder's late-life onset" is strongly linked to neuropsychological disorders. Thus, prior to E.C.T., elderly people who come with "new-onset mania" must have a thorough clinical examination as well as a neuropsychological workup. There is no discernible distinction in the rate of progress between bipolar and unipolar disorder following E.C.T. treatment for bipolar disorder [56]. According to numerous research, bipolar illness requires lesser E.C.T. therapy compared to unipolar disorder to produce equivalent results. As a result, E.C.T. may serve as a wonderful substitute for medication for senior bipolar individuals who are unable to handle it. The effectiveness of E.C.T. in treating bipolar mania is debatable because of the different structural positions of the insertion of the electrodes. Others have argued that mania was unaffected by solitary E.C.T. while vehemently defending the benefits of B.L-E.C.T. Once lithium as well as certain various antipsychotic prescription medications were employed as the "first-line" management for manic episodes, subsequent studies turned their attention to the usefulness and appropriateness of E.C.T. for "medicine-resistant mania". 90 percent of medication-resistant manic individuals who had a short session of E.C.T. obtained resolution or, at the very minimum, showed a significant change in symptoms, according to the findings of a comprehensive research assessment conducted before 2020 [57].

16. CATATONIA AND THE APPLICATION OF ELECTROCONVULSIVE TREATMENT (ECT) :

The complicated as well as diverse phenomenon known as catatonia is characterized by motor irregularities that take place in conjunction with alterations in cognition, temperament, as well as alertness. Catatonia has a complicated underpinning etiology that includes mental disorders, physical

ailments, as well as neuropsychological problems. The much more extreme type of catatonia is called "malignant catatonia", which may be made worse by body part dysfunction, autonomic disturbance, and potentially fatal clinical problems like malnutrition, sepsis, hemorrhage, and deep vein thrombosis. " Malignant catatonia" is more likely to occur in elderly people. If ignored or incorrectly identified, it may be fatal. For elderly persons who had signs of mild or aggressive catatonia, E.C.T. may be very much useful. The very first therapy for fairly benign catatonia is prescription drugs like benzodiazepines. For older individuals who are catatonic, several writers have also recommended substitute drugs such as "midazolam, memantine, topiramate, and amantadine" [58]. Regarding catatonia that is unresponsive to medicine, E.C.T. could be quite successful. For N.M.S., aggressive catatonia, especially remaining or recalcitrant catatonia, a mixture of benzodiazepine drugs as well as E.C.T. has proven to be quite successful.

17. ELECTROCONVULSIVE TREATMENT (ECT) AND DEMENTIA :

Among the elderly community, dementia has become one of the leading factors of impairment. Following "vascular dementia" and "dementia with Lewy bodies" as the main causes of dementia come "Alzheimer's disease". Intellectual deficiencies might ameliorate in many, however not all, demented individuals with concomitant significant depressive episodes when underpinning emotional or psychotic manifestations are effectively managed with E.C.T. Additional research discovered that, despite the fact that E.C.T. was effective in treating depressive episodes, individuals with "vascular dementia" and medically non-demented individuals with Magnetic resonance image brain signal hyperintensity experienced mental impairment or brief deterioration following the procedure. Several randomized prospective studies have demonstrated the efficacy of E.C.T., which was employed as a last resort after all other possible therapeutic interventions had failed [59]. Nevertheless, the main focus of this research was the advantage of E.C.T. for relatively brief behavioral management. Nowadays, E.C.T. is thought to be the sole option left for treating restlessness and violence in people living with Alzheimer's/dementia. In certain demented individuals who got B.L-E.C.T., postictal extended disorientation, as well as impaired intellectual or recollection performance, may develop. Nevertheless, it was noted that most of these detrimental consequences were temporary as well as recoverable, lasting anywhere from just a few hours up to a couple of weeks. Aspects including advanced aging, post-intellectual disability, E.C.T. co-administration with some other medications, as well as clinical complications might potentially be an influence [60].In practice, E.C.T. is acceptable as well as successful for managing individuals with serious behavioral problems brought on by Alzheimer's disease.

18. THE USE OF ELECTROCONVULSIVE THERAPY (ECT) IN THE TREATMENT OF PARKINSON'S DISEASE (PD) :

Individuals suffering from Parkinson's disease could also experience mental deterioration, depressive symptoms, anxiousness, and/or sadness. L-dopa, as well as dopaminergic agonists may even have adverse consequences that can include "frank hallucinations", which are often optical, suspicion or fantasies, hyperactivity, as well as agitation. Drug rehabilitation is an option for symptomatic management. Individuals who experience "antiparkinsonian drug-induced psychosis", "antipsychotic drug-refractory psychosis", or persistent motor abnormalities caused by antipsychotics notwithstanding stopping the problematic drugs may benefit from receiving E.C.T. Parkinson's disease is more common in older adults who have used psychiatric medications [61]. Parkinsonism in older individuals may impair their ability to manage themselves, which might result in disobedience with medication therapy. Scientific papers including series showed that E.C.T. was a successful therapy for chronic Parkinson's disease, indicating that it could be a different choice of therapy if motion difficulties need to be resolved. Many individuals who really are resistant to antiparkinsonian medications may benefit from M-E.C.T. as an additional therapy. Temporary delirium, disorientation, forgetfulness, as well as intellectual disability are just the negative consequences of E.C.T. that people with Parkinson's disorder experience much more frequently [62]. To prevent post-E.C.T. delirium as well as dyskinesis, it could be necessary to lower the quantity of L-dopa or dopaminergic agonists. Generally, E.C.T. is well received, secure, as well as successful in treating senior parkinsonism individuals.

19. UTILIZATION OF ELECTROCONVULSIVE THERAPY (ECT) IN THE TREATMENT OF POST-STROKE SYMPTOMS :

Among aged persons in the overall community, "post-stroke depression" was 64 percent more common than it was in the entire community. Many post-stroke depressive symptoms happen during the initial two years following a cerebro-vascular occurrence. The incidence, as well as intensity of post-stroke depressive episodes, are significantly influenced by a lesion's site, notably by how close it is to the "left frontal pole". Even though post-stroke psychosis is rare, it can have medically substantial effects in cases when injuries occur in the "right hemisphere". An analysis of Fifty senior individuals' records who already had undergone E.C.T. for post-stroke melancholy revealed that 98 percent of them recovered, while two percent experienced brief interictal disorientation or forgetfulness. In this research, there were no patients who had acute cognitive impairments that worsened [63]. These results imply that E.C.T. must not be postponed for senior individuals with strokes who are depressed since treatment is typically well received as well as beneficial.

20. ELECTROCONVULSIVE THERAPY (ECT) AS A FIRST-LINE THERAPEUTIC MODALITY :

Although further therapies are recommended, melancholic lethargy including divisibility, such as in gloomy, delirious, or psychotic despair, may constitute a first-line rationale requiring E.C.T. In cases of significant psychomotor impairment or rejection of meals as well as water, E.C.T. is crucial since it has been demonstrated to be related to a rapid, alleviation of manifestations. When there is a substantial danger of suicidal behavior including severe psychotic episodes, E.C.T. should always be explored before alternative treatment methods. With E.C.T., the recovery percentage for psychotic melancholy is close to 95%, and improvement is often seen within ten to fourteen days. E.C.T. quickly reduces the likelihood of suicidal behavior that characterizes serious mental diseases, but focusing on follow-up therapies is crucial to maintain the effect [64]. Moreover, the delivery of drugs is frequently not possible, making ECT a viable therapy alternative when despair, instability, as well as psychotic manifestations are prominent together with underlying disorders, prevalent throughout early gestation, or evident throughout the post-partum lactating periods. E.C.T. mono-therapy may serve as a secure initial line of therapeutic approach for serious as well as life-threatening drug side effects. This is particularly relevant for people who have serious physical illnesses that might get more serious as a result of using antidepressants or antipsychotic medications. Yet, the intense censure and sometimes perhaps regulatory prohibitions regarding its usage in certain countries make E.C.T.'s primary application difficult. Consideration of E.C.T. as a key criterion as well as familiarity with every one of the avenues for appropriate permission for therapy under their territory is beneficial for clinicians who have the charge of the most symptomatic as well as seriously sick mental patient populations [65].

21. ELECTROCONVULSIVE THERAPY (ECT) AS A SECOND-LINE THERAPEUTIC MODALITY :

Although individuals very seldom undergo E.C.T. right away after meeting the parameters for pharmacologic rejection, therapeutic setbacks comprise the most common reason for its use. The use of E.C.T. greatly improves treatment outcomes. Even when psychotropic medicines have been used to their fullest potential, this is particularly true in individuals who are experiencing psychotic melancholy [66]. A regimen of E.C.T. therapy may often be started due to unbearable negative effects of antipsychotic pharmaceuticals, physiological abnormalities that develop following pharmacological therapy, or increasing depression manifestations, especially serious suicidality.

22. ELECTROCONVULSIVE THERAPY (ECT) AS A LAST RESORT THERAPEUTIC MODALITY :

No solid information produced from randomized controlled trials proving the effectiveness of E.C.T. can indeed be discovered in the research domain for uncommon last-resort reasons. Yet, despite several therapeutic setbacks with pharmacologic as well as psychotherapy techniques, circumstantial medical studies, clinical studies, and historical evaluations point to the overall success of E.C.T. in treating "obsessive-compulsive disorder" [67]. Furthermore, "Tourettc's syndrome" patients reported experiencing quick and long-lasting symptomatic alleviation. Whether there is concurrent melancholy existing or missing, a treatment-resistant seizure can benefit from E.C.T. for quick symptomatic alleviation. After just a period of electroshock therapy, Parkinson's disease patients' poor neuromuscular performance and depressed moods both improved. Naturally, an individualized

advantages/risks calculation, comprising a thorough assessment of recent therapy difficulties, must be performed for each subject, especially in these kinds of uncommon situations with last-resort reasons.

23. SIDE EFFECTS OF ELECTROCONVULSIVE THERAPY (ECT) :

Many have been discouraged from seeking therapy because of the widespread dread of E.C.T., which is a result of inadequate therapy procedures as well as erroneous press depictions. With its proven effectiveness and tolerability, as well as the recorded effective management of famous writers, singers, actors, and statesmen, the taboo surrounding E.C.T. and the dread of significant clinical and mental side effects appear contradictory [68]. From this therapy's debut, the idea that it "fries the brain" or produces neural destruction has just been spread. Unfortunately, there is no evidence to back up this claim, and research actually disproves it. Researchers examined intellectual outcomes, neuroimaging, the autopsy of previous E.C.T. participants, clinical seizure investigations, and more than fifty mouse models in a comprehensive analysis that they released. They did not discover proof that E.C.T. causes any harm to the head on a morphological or molecular basis.

23.1 Common physical side effects of electroconvulsive therapy (ECT):

The majority of individuals would not claim that receiving E.C.T. is a distressing treatment. Nevertheless, specifically during the initial stage of therapy, few individuals may have somatic negative impacts from E.C.T. such as headache, vomiting, and muscular discomfort. These negative outcomes are not life-threatening and can be brought on by convulsions, the anesthetic, or a conjunction of both of them. Individuals may also suffer "anesthetic awareness," an uncommon but uncomfortable adverse impact of the anesthetic agent and muscular relaxing agent combo, in which they might awaken before the muscular relaxation wears off. Anesthetists may now create muscle relaxation separately from anesthesia, allowing them to regulate the two main aspects as necessary to make sure their clients are both securely asleep as well as adequately relaxed to receive therapy. As previously stated, "anesthetic awareness," which results from unexpected personal variances in anesthesia needs, can albeit rarely, happen. The person could be incapable to inhale or operate in this circumstance, yet he or she is powerless to notify carers of their condition. Despite not being harmful to the individual because oxygenation is being given, it is very upsetting to the person's perceptual experience and necessitates both a change in the anesthetic delivery as well as a strong psychodynamic psychotherapy approach. Unfortunately, before beginning therapy, individuals are unable to predict if they would have any harmful consequences. Enough that, it is critical to let individuals understand any unexpected but potential negative impacts as well as the fact that if needed, therapy would be given for any particular problems they may have [69].

Approximately forty percent of the total participants are said to suffer from "post-ictal headaches". This complaint is often moderate, affects people most commonly when they are younger, and includes a frontal pulsating feature. Despite the fact that its cause is unknown, it seems to possess a noticeable vascular feature and might be related to the mechanical components of the therapy [70]. Conventional painkillers may typically relieve post-ictal headaches, although few individuals develop more terrible headaches that call for preventive use of a "non-steroidal anti-inflammatory drug" or a targeted "anti-migraine" medication.

After E.C.T., individuals might feel vomiting; documented incidences of vomiting range from 1 percent to 3 percent. The feeling of vomiting may well be brought on by a headache, or its management or it could develop on its own as an anesthetic side effect. However vomiting may go away after the headache is treated, preventive therapy with medications like "ondansetron" can also be employed.

Muscular discomfort may accompany E.C.T., more typically just after initial treatment. This discomfort is perhaps most definitely the result of severe fasciculation brought on by the use of "depolarizing muscle relaxants" like succinylcholine. Because the E.C.T. stimulus directly stimulates the musculature close to the jaw, individuals could also feel temporomandibular joint discomfort. A bite frame should be tightly closed to prevent the onset of jaw pain, which may then be managed with aspirin tablets and otherwise N.S.A.I.D.s.Although depolarizing muscular flexing agents are often tolerable in subsequent sessions, a non-depolarizing drug like curare that prevents muscular fasciculations might well be utilized as a therapeutic adjustment in situations of more prolonged discomfort. Individuals with nerve and muscle ailments who could really encounter negative impacts from the na+ release as well as the muscle-depolarizing response of drugs like succinylcholine

can additionally be given [71]. Unfortunately, the majority of individuals would not feel enough muscular discomfort to call for the adoption of this adjustment.

23.2 Common cognitive side effects of electroconvulsive therapy (ECT) :

The much more troublesome side effect of E.C.T. is still memory difficulties. Those who have had E.C.T. since it was first introduced have noted intellectual adverse consequences. These outcomes continue to be of worry. The intellectual adverse responses of E.C.T. may be influenced by a variety of neurobiological, procedural, and patient-specific variables, and individuals may respond to E.C.T. in profoundly different ways, either objectively or subjectively.

It is recognized that E.C.T. has physiological impacts on the regions of the cerebrum that are thought to be crucial to both short- as well as long-declarative memory. For instance, E.C.T. has an impact on recollection functions connected to the "medial temporal lobe", which houses the hippocampal region and is involved in the retention of novel knowledge ("anterograde"). Long-term ("retrograde") cognitive problems brought on by E.C.T., nonetheless, may well be connected to physiological alterations in the "prefrontal cortex" brought on by the procedure. "Medial temporal lobe" modifications may well not account for these abnormalities. If these problems are resolved, it could influence how people think about the mechanical elements of therapy and lead to those changes. Mechanical therapeutic factors such as stimulant amplitude, electrode location, stimulation strength, dose in relation to a cutoff, and the quantity and timing of therapies, in total, can have a big impact on the kind as well as the severity of intellectual impairments after a session of E.C.T [72]. Client- related variables including gender, ethnicity, basic intellectual performance, and concurrent psychiatric medicines might also be important. The four main types of intellectual adverse reactions are listed below.

23.2.1 Stereotypical and transient postictal disorientation:

The whole initial sort of cognitive consequence is the "stereotypical and brief postictal disorientation" experienced by people just after receiving E.C.T., and this is the result of both the initial convulsions as well as the anesthetic used. This could also span from minor occurrences that go away in a matter of minutes or hrs to uncommon instances of serious organic disorders. Restoration times may vary depending on the delivery of E.C.T., involving electrode positioning as well as other therapies. Individuals, nevertheless, have quite varied emotional perceptions of this time. Many individuals regain consciousness quickly and thus are capable to carry on with their daily routines. Some might doze off for a while before being capable of eating and getting on with their day [73]. There's certain are certain individuals, nevertheless, who noticeably get disoriented, and all these consequences might lead to increased perceptual discomfort.

22.2.2 Anteriograde amnesia:

Anterograde amnesia", which similarly fluctuates in degree, is a separate kind of intellectual consequence. It is the incapacity to remember knowledge acquired throughout and immediately after a session of E.C.T. therapy. " Anterograde amnesia" can play a key role in a person's failure to remember crucial details regarding their condition as well as their therapy in particular. Individuals may believe that doctors are evading questions or just not answering them while in reality, they might have never understood what was said [74]. For all of these causes, keeping a personal diary is strongly advised.

23.2.3 Short-term retrograde amnesia:

"Short-term retrograde amnesia", which causes recollection lapses for incidents that took place within a couple of days/weeks or potentially months well preceding the session of E.C.T., is the next category of intellectual effects. " Retrograde amnesia" often gets better over the initial several months following the short E.C.T. cycle, while some individuals may not fully recuperate. This may distress individuals, especially if they put a high significance on their own recollections as well as activities [75].

23.2.4 More extensive retrograde amnesia:

The last form of intellectual disability, which is thankfully uncommon, includes extremely significant retrograde-memory decline, in that the individual has significant, ongoing memory problems that go back many weeks or perhaps years [75].

24. CONCLUSION :

E.C.T. is a non - medication physiological therapy that has been demonstrated in several clinical experiments to be a massively successful therapeutic modality, mostly for depressive episodes but also for psychosis. This is applicable to both initial and sustaining therapy; E.C.T. may be employed to avoid relapses during the latter. In particular, the use of altered activation methods and advancements in contemporary anesthetics have improved the security and acceptability of electroshock therapy. Clinical disorders that were once definite restrictions for E.C.T. have changed to comparative contraindications in current history [76]. Hence, individuals with greater physiological hazards can now also receive secure therapy. Nevertheless, the information describing the neurological processes behind E.C.T., erratic study results prevent making conclusive conclusions. This is due to gaps in the current knowledge, which include the dearth of comparison groups in many experiments and the absence of uniformity in the research design and respondents of the research project. However, it was not possible to demonstrate with exact confidence the cause-and-effect link between both the results as well as the curative potential of E.C.T., therefore it's hardly advisable to presume that a solitary technique can account for these outcomes. Presently, E.C.T. is the most extensively accessible nonpharmacologic therapeutic method for serious psychological disorders, while other brain stimulation treatments are now being explored. These much more recent possible therapeutic techniques range from less infiltrative techniques like "transcranial magnetic stimulation", "transcranial direct current stimulation", and "magnetic seizure therapy" (M.S.T.) to even more infiltrative techniques like "vagal nerve stimulation", "deep brain stimulation", and "epidural cortical stimulation" [77]. The U.S-F.D.A. has only authorized "transcranial magnetic stimulation" and "vagal nerve stimulation". One of the main factors restricting the application of E.C.T. is its negative intellectual consequences. Further efforts are being made to lessen the negative impacts on mental performance and to place electrodes close to certain anatomical regions of the cortex that are connected to personality as well as behavior while conserving regions involved in the acquisition, remembering, and comprehension. Current investigation has improved our understanding of potential ECT modes of operation as well as helped us develop a secure, well-tolerated therapy, however, regrettably, researchers still don't have a definitive understanding of these fundamental processes. But, this extremely powerful clinical choice shouldn't be withheld, particularly from mental patients who have failed to respond to conventional therapies. In the management of "treatment-resistant depression", E.C.T. is still a viable choice [78]. The stereotype and discrimination towards psychological diseases as well as particular treatments like E.C.T. might be reduced with comprehensive informational campaigns in institutions and the increased availability of accurate, factual reporting in the mainstream press.

REFERENCES :

[1] Baghai, T. C., & Möller, H. J. (2008). Electroconvulsive therapy and its different indications. *Dialogues in clinical neuroscience*, *10*(1), 105–117. Google Scholar↗

[2] Singh, A., & Kar, S. K. (2017). How Electroconvulsive Therapy Works?: Understanding the Neurobiological Mechanisms. *Clinical psychopharmacology and neuroscience: the official scientific journal of the Korean College of Neuropsychopharmacology*, *15*(3), 210–221. Google Scholar↗

[3] Tirmizi, O., Raza, A., Trevino, K., & Husain, M. M. (2012). Electroconvulsive therapy: How modern techniques improve patient outcomes: Refinements have decreased memory loss, other adverse effects while retaining efficacy: Refinements have decreased memory loss, other adverse effects while retaining efficacy. *Current psychiatry*, *11*(10), 24 - 46. Google Scholar↗

[4] Kerner, N., & Prudic, J. (2014). Current electroconvulsive therapy practice and research in the geriatric population. *Neuropsychiatry*, *4*(1), 33–54. Google Scholar↗

[5] Payne, N. A., & Prudic, J. (2009). Electroconvulsive therapy: Part I. A perspective on the evolution and current practice of ECT. *Journal of psychiatric practice*, *15*(5), 346–368. Google Scholar↗

[6] UK Ect Review Group. (2003). Efficacy and safety of electroconvulsive therapy in depressive disorders: a systematic review and meta-analysis. *The Lancet*, *361*(9), 799-808. Google Scholar↗

[7] Wahlund, B., & von Rosen, D. (2003). ECT of major depressed patients in relation to biological and clinical variables: a brief overview. *Neuropsychopharmacology*, *28*(1), 21-26. Google Scholar↗

[8] Newman, M. E., Gur, E., Shapira, B., & Lerer, B. (1998). Neurochemical mechanisms of action of ECS: evidence from in vivo studies. *The Journal of ECT*, *14*(3), 153-171. Google Scholar↗

[9] Gur, E., Dremencov, E., Garcia, F., Van de Kar, L. D., Lerer, B., & Newman, M. E. (2002). Functional effects of chronic electroconvulsive shock on serotonergic 5-HT1A and 5-HT1B receptor activity in rat hippocampus and hypothalamus. *Brain research*, *952*(1), 52-60. Google Scholar↗

[10] Hoekstra, R., van den Broek, W. W., Fekkes, D., Bruijn, J. A., Mulder, P. G., & Pepplinkhuizen, L. (2001). Effect of electroconvulsive therapy on biopterin and large neutral amino acids in severe, medication-resistant depression. *Psychiatry research*, *103*(3), 115-123. Google Scholar↗

[11] Palmio, J., Huuhka, M., Saransaari, P., Oja, S. S., Peltola, J., Leinonen, E., & Keränen, T. (2005). Changes in plasma amino acids after electroconvulsive therapy of depressed patients. *Psychiatry research*, *137*(3), 183-190. Google Scholar↗

[12] Sackeim, H. A. (1999). The anticonvulsant hypothesis of the mechanisms of action of ECT: current status. *The journal of ECT*, *15*(1), 5-26. Google Scholar↗

[13] Sundsted, K. K., Burton, M. C., Shah, R., & Lapid, M. I. (2014). Preanesthesia medical evaluation for electroconvulsive therapy: a review of the literature. *The journal of ECT*, *30*(1), 35-42. Google Scholar↗

[14] Hoirisch-Clapauch, S., Mezzasalma, M. A., & Nardi, A. E. (2014). Pivotal role of tissue plasminogen activator in the mechanism of action of electroconvulsive therapy. *Journal of Psychopharmacology*, *28*(2), 99-105. Google Scholar↗

[15] Medda, P., Toni, C., & Perugi, G. (2014). The mood-stabilizing effects of electroconvulsive therapy. *The journal of ECT*, *30*(4), 275-282. Google Scholar↗

[16] Swartz, C. M. (2014). A mechanism of seizure induction by electricity and its clinical implications. *The journal of ECT*, *30*(2), 94-97. Google Scholar↗

[17] Takano, H., Motohashi, N., Uema, T., Ogawa, K. I., Ohnishi, T., Nishikawa, M., & Matsuda, H. (2011). Differences in cerebral blood flow between missed and generalized seizures with electroconvulsive therapy: a positron emission tomographic study. *Epilepsy research*, *97*(2), 225-228. Google Scholar↗

[18] Deng, Z. D., Lisanby, S. H., & Peterchev, A. V. (2013). Controlling stimulation strength and focality in electroconvulsive therapy via current amplitude and electrode size and spacing: comparison with magnetic seizure therapy. *The journal of ECT*, *29*(4), 325-343. Google Scholar↗

[19] Enev, M., McNally, K. A., Varghese, G., Zubal, I. G., Ostroff, R. B., & Blumenfeld, H. (2007). Imaging onset and propagation of ECT-induced seizures. *Epilepsia*, *48*(2), 238-244. Google Scholar↗

[20] Kranaster, L., Plum, P., Hoyer, C., Sartorius, A., & Ullrich, H. (2013). Burst suppression: a more valid marker of postictal central inhibition?. *The Journal of ECT*, *29*(1), 25-28. Google Scholar↗

[21] Takano, H., Motohashi, N., Uema, T., Ogawa, K., Ohnishi, T., Nishikawa, M., & Matsuda, H. (2007). Changes in regional cerebral blood flow during acute electroconvulsive therapy in patients with depression: positron emission tomographic study. *The British Journal of Psychiatry*, *190*(1), 63-68. Google Scholar↗

[22] Greenberg, R. M., & Kellner, C. H. (2005). Electroconvulsive therapy: a selected review. *The American journal of geriatric psychiatry*, *13*(4), 268-281. Google Scholar↗

[23] Janicak, P. G., Dowd, S. M., Rado, J. T., & Welch, M. J. (2010). The re-emerging role of therapeutic neuromodulation: recent developments have revived interest in for difficult-to-treat patients. *Current Psychiatry*, *9*(1), 66-74. Google Scholar↗

[24] Kellner, C. H., Knapp, R. G., Petrides, G., Rummans, T. A., Husain, M. M., Rasmussen, K., & Fink, M. (2006). Continuation electroconvulsive therapy vs pharmacotherapy for relapse prevention in major depression: a multisite study from the Consortium for Research in

Electroconvulsive Therapy (CORE). *Archives of general psychiatry*, *63*(12), 1337-1344. Google Scholar↗

[25] Husain, M. M., Rush, A. J., Fink, M., Knapp, R., Petrides, G., Rummans, T., & Kellner, C. H. (2004). Speed of response and remission in major depressive disorder with acute electroconvulsive therapy (ECT): a Consortium for Research in ECT (CORE) report. *The Journal of clinical psychiatry*, *65*(4), 199 217. Google Scholar↗

[26] Petrides, G., Fink, M., Husain, M. M., Knapp, R. G., Rush, A. J., Mueller, M., & Kellner, C. H. (2001). ECT remission rates in psychotic versus nonpsychotic depressed patients: a report from CORE. *The journal of ECT*, *17*(4), 244-253. Google Scholar↗

[27] Semkovska, M., Keane, D., Babalola, O., & McLoughlin, D. M. (2011). Unilateral brief-pulse electroconvulsive therapy and cognition: effects of electrode placement, stimulus dosage and time. *Journal of Psychiatric Research*, *45*(6), 770-780. Google Scholar↗

[28] Peterchev, A. V., Rosa, M. A., Deng, Z. D., Prudic, J., & Lisanby, S. H. (2010). ECT stimulus parameters: rethinking dosage. *The journal of ECT*, *26*(3), 159-172. Google Scholar↗

[29] Weiner, R. D., Rogers, H. J., Davidson, J. R., & Squire, L. R. (1986). Effects of stimulus parameters on cognitive side effects. *Annals of the New York Academy of Sciences*, *462*(1), 315-325. Google Scholar↗

[30] Leiknes, K. A., Schweder, L. J. V., & Høie, B. (2012). Contemporary use and practice of electroconvulsive therapy worldwide. *Brain and behavior*, *2*(3), 283-344. Google Scholar↗

[31] Rosenbach, M. L., Hermann, R. C., & Dorwart, R. A. (1997). Use of electroconvulsive therapy in the Medicare population between 1987 and 1992. *Psychiatric services (Washington, DC)*, *48*(1), 1537-1542. Google Scholar↗

[32] Weiner, R. D., & Prudic, J. (2013). Electroconvulsive therapy in the United States: how often is it used?. *Biological psychiatry*, *73*(2), 105-106. Google Scholar↗

[33] Roose, S. P., Sackeim, H. A., Krishnan, K. R. R., Pollock, B. G., Alexopoulos, G., Lavretsky, H., & Old-Old Depression Study Group. (2004). Antidepressant pharmacotherapy in the treatment of depression in the very old: a randomized, placebo-controlled trial. *American Journal of Psychiatry*, *161*(1), 250-279. Google Scholar↗

[34] Schatzberg, A. F., & Kraemer, H. C. (2000). Use of placebo control groups in evaluating efficacy of treatment of unipolar major depression. *Biological psychiatry*, *47*(8), 736-744. Google Scholar↗

[35] Roose, S. P., & Sackeim, H. A. (2002). Clinical trials in late-life depression: revisited. *The American Journal of Geriatric Psychiatry*, *10*(5), 503-505. Google Scholar↗

[36] Retrived from Google https://images.app.goo.gl/sfyomxNwaVKFiqQD6 on 25th February 2023.

[37] Sneed, J. R., Culang-Reinlieb, M. E., Brickman, A. M., Gunning-Dixon, F. M., Johnert, L., Garcon, E., & Roose, S. P. (2011). MRI signal hyperintensities and failure to remit following antidepressant treatment. *Journal of affective disorders*, *135*(1), 315-320. Google Scholar↗

[38] Retrived from Google https://images.app.goo.gl/b11erQoBuyB67U8HA on 2nd March 2023.

[39] Kerr, R. A., McGrath, J. J., O'kearney, R. T., & Price, J. (1982). ECT: misconceptions and attitudes. *Australian and New Zealand Journal of Psychiatry*, *16*(1), 43-49. Google Scholar↗

[40] Roose, S. P., & Schatzberg, A. F. (2005). The efficacy of antidepressants in the treatment of late-life depression. *Journal of clinical psychopharmacology*, *25*(4), 1-7. Google Scholar↗

[41] Hirshbein, L., & Sarvananda, S. (2008). History, power, and electricity: American popular magazine accounts of electroconvulsive therapy, 1940–2005. *Journal of the History of the Behavioral Sciences*, *44*(1), 1-18. Google Scholar↗

[42] Hamilton, M. A. X. (1967). Development of a rating scale for primary depressive illness. *British journal of social and clinical psychology*, *6*(4), 278-296. Google Scholar↗

[43] Kapur, S. (1996). Psychopharmacology: the fourth generation of progress. *Journal of Psychosomatic Research*, *3*(1), 291-292. Google Scholar↗

[44] Sackeim, H. A. (1999). The anticonvulsant hypothesis of the mechanisms of action of ECT: current status. *The journal of ECT*, *15*(1), 5-26. Google Scholar↗

[45] Mervaala, E., Könönen, M., Föhr, J., Husso-Saastamoinen, M., Valkonen-Korhonen, M., Kuikka, J. T., & Lehtonen, J. (2001). SPECT and neuropsychological performance in severe depression treated with ECT. *Journal of affective disorders*, *66*(1), 47-58. Google Scholar↗

[46] Swartz, C. M., & Nelson, A. I. (2005). Rational electroconvulsive therapy electrode placement. *Psychiatry (Edgmont (Pa.: Township))*, *2*(7), 37–43. Google Scholar↗

[47] Retrieved from Google https://images.app.goo.gl/b6vpExNnEhUwbVBd7 on 4th March 2023.

[48] Kadiyala, P. K., & Kadiyala, L. D. (2017). Anesthesia for electroconvulsive therapy: An overview with an update on its role in potentiating electroconvulsive therapy. *Indian journal of anaesthesia*, *61*(5), 373–380. Google Scholar↗

[49] Holsboer, F. (2000). The corticosteroid receptor hypothesis of depression. *Neuropsychopharmacology*, *23*(5), 477-501. Google Scholar↗

[50] Szuba, M. P., O'Reardon, J. P., & Evans, D. L. (2000). Physiological effects of electroconvulsive therapy and transcranial magnetic stimulation in major depression. *Depression and anxiety*, *12*(3), 170-177. Google Scholar↗

[51] Grunhaus, L., Zelnik, T., Albala, A. A., Rabin, D., Haskett, R. F., Zis, A. P., & Greden, J. F. (1987). Serial dexamethasone suppression tests in depressed patients treated only with electroconvulsive therapy. *Journal of affective disorders*, *13*(3), 233-240. Google Scholar↗

[52] Rupprecht, R. (1997). The neuropsychopharmacological potential of neuroactive steroids. *Journal of psychiatric research*, *31*(3), 297-314. Google Scholar↗

[53] Baghai, T. C., di Michele, F., Schüle, C., Eser, D., Zwanzger, P., Pasini, A., & Rupprecht, R. (2005). Plasma concentrations of neuroactive steroids before and after electroconvulsive therapy in major depression. *Neuropsychopharmacology*, *30*(6), 1181-1186. Google Scholar↗

[54] Allen, P., Chaddock, C. A., Egerton, A., Howes, O. D., Bonoldi, I., Zelaya, F., & McGuire, P. (2016). Resting hyperperfusion of the hippocampus, midbrain, and basal ganglia in people at high risk for psychosis. *American Journal of Psychiatry*, *173*(4), 392-399. Google Scholar↗

[55] Hua, J., Brandt, A. S., Lee, S., Blair, N. I., Wu, Y., Lui, S., & Margolis, R. L. (2017). Abnormal grey matter arteriolar cerebral blood volume in schizophrenia measured with 3D inflow-based vascular-space-occupancy MRI at 7T. *Schizophrenia bulletin*, *43*(3), 620-632. Google Scholar↗

[56] Berggren, Å., Gustafson, L., Höglund, P., & Johanson, A. (2014). A long-term follow-up of clinical response and regional cerebral blood flow changes in depressed patients treated with ECT. *Journal of affective disorders*, *16(*7), 235-243. Google Scholar↗

[57] Hosokawa, T., Momose, T., & Kasai, K. (2009). Brain glucose metabolism difference between bipolar and unipolar mood disorders in depressed and euthymic states. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *33*(2), 243-250. Google Scholar↗

[58] Suwa, T., Namiki, C., Takaya, S., Oshita, A., Ishizu, K., Fukuyama, H., & Murai, T. (2012). Corticolimbic balance shift of regional glucose metabolism in depressed patients treated with ECT. *Journal of affective disorders*, *136*(3), 1039-1046. Google Scholar↗

[59] Nobler, M. S., Oquendo, M. A., Kegeles, L. S., Malone, K. M., Campbell, C., Sackeim, H. A., & Mann, J. J. (2001). Decreased regional brain metabolism after ECT. *American Journal of Psychiatry*, *158*(2), 305-308. Google Scholar↗

[60] Yatham, L. N., Clark, C. C., & Zis, A. P. (2000). A preliminary study of the effects of electroconvulsive therapy on regional brain glucose metabolism in patients with major depression. *The journal of ECT*, *16*(2), 171-176. Google Scholar↗

[61] Bonne, O., Krausz, Y., Shapira, B., Bocher, M., Karger, H., Gorfine, M., & Lerer, B. (1996). Increased cerebral blood flow in depressed patients responding to electroconvulsive therapy. *Journal of Nuclear Medicine*, *37*(7), 1075-1080. Google Scholar↗

[62] Andrade, C., & Bolwig, T. G. (2014). Electroconvulsive therapy, hypertensive surge, blood-brain barrier breach, and amnesia: exploring the evidence for a connection. *The journal of ECT*, *30*(2), 160-164. Google Scholar↗

[63] Trevino, K., McClintock, S. M., & Husain, M. M. (2010). A review of continuation electroconvulsive therapy: application, safety, and efficacy. *The journal of ECT*, *26*(3), 186-195. Google Scholar↗

[64] Merkl, A., Heuser, I., & Bajbouj, M. (2009). Antidepressant electroconvulsive therapy: mechanism of action, recent advances and limitations. *Experimental neurology*, *219*(1), 20-26. Google Scholar↗

[65] Ding, Z., & White, P. F. (2002). Anesthesia for electroconvulsive therapy. *Anesthesia & Analgesia*, *94*(5), 1351-1364. Google Scholar↗

[66] Kalinowsky, L. B. (1986). History of Convulsive Therapy a. *Annals of the New York Academy of Sciences*, *462*(1), 1-4. Google Scholar↗

[67] Ghaziuddin, N., Dumas, S., & Hodges, E. (2011). Use of continuation or maintenance electroconvulsive therapy in adolescents with severe treatment-resistant depression. *The journal of ECT*, *27*(2), 168-174. Google Scholar↗

[68] Nuttall, G. A., Bowersox, M. R., Douglass, S. B., McDonald, J., Rasmussen, L. J., Decker, P. A., & Rasmussen, K. G. (2004). Morbidity and mortality in the use of electroconvulsive therapy. *The journal of ECT*, *20*(4), 237-241. Google Scholar↗

[69] Hihn, H., Baune, B. T., Michael, N., Markowitsch, H., Arolt, V., & Pfleiderer, B. (2006). Memory performance in severely depressed patients treated by electroconvulsive therapy. *The Journal of ECT*, *22*(3), 189-195. Google Scholar↗

[70] Prudic, J., Peyser, S., & Sackeim, H. A. (2000). Subjective memory complaints: a review of patient self-assessment of memory after electroconvulsive therapy. *The journal of ECT*, *16*(2), 121-132. Google Scholar↗

[71] Alexopoulos, G. S., Murphy, C. F., Gunning-Dixon, F. M., Latoussakis, V., Kanellopoulos, D., Klimstra, S., ... & Hoptman, M. J. (2008). Microstructural white matter abnormalities and remission of geriatric depression. *American Journal of Psychiatry*, *165*(2), 238-244. Google Scholar↗

[72] Navarro, V., Gastó, C., Lomeña, F., Torres, X., Mateos, J. J., Portella, M., ... & Marcos, T. (2004). Prognostic value of frontal functional neuroimaging in late-onset severe major depression. *The British Journal of Psychiatry*, *184*(4), 306-311. Google Scholar↗

[73] Taylor, W. D., Steffens, D. C., MacFall, J. R., McQuoid, D. R., Payne, M. E., Provenzale, J. M., & Krishnan, K. R. R. (2003). White matter hyperintensity progression and late-life depression outcomes. *Archives of general psychiatry*, *60*(11), 1090-1096. Google Scholar↗

[74] Perera, T. D., Luber, B., Nobler, M. S., Prudic, J., Anderson, C., & Sackeim, H. A. (2004). Seizure expression during electroconvulsive therapy: relationships with clinical outcome and cognitive side effects. *Neuropsychopharmacology*, *29*(4), 813-825. Google Scholar↗

[75] Altman, J., & Das, G. D. (1965). Autoradiographic and histological evidence of postnatal hippocampal neurogenesis in rats. *Journal of Comparative Neurology*, *124*(3), 319-335. Google Scholar↗

[76] Perera, T. D., Coplan, J. D., Lisanby, S. H., Lipira, C. M., Arif, M., Carpio, C., & Dwork, A. J. (2007). Antidepressant-induced neurogenesis in the hippocampus of adult nonhuman primates. *Journal of Neuroscience*, *27*(18), 894-901. Google Scholar↗

[77] Madsen, T. M., Treschow, A., Bengzon, J., Bolwig, T. G., Lindvall, O., & Tingström, A. (2000). Increased neurogenesis in a model of electroconvulsive therapy. *Biological psychiatry*, *47*(12), 1043-1049. Google Scholar↗

[78] Sheline, Y. I., Gado, M. H., & Kraemer, H. C. (2003). Untreated depression and hippocampal volume loss. *American journal of psychiatry*, *16*(8), 1516-1518. Google Scholar↗

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