Sri Lakshmi Narayana Institute of Medical Sciences

Date: 02.2.2019

From
DR.V.R Sridhar
Professor and Head,
Department of Psychiatry,
Sri Lakshmi Narayana Institute Of Medical Sciences,
Bharath Institute of Higher Education and Research,
Chennai.

To
The Dean,
Sri Lakshmi Narayana Institute Of Medical Sciences,
Bharath Institute of Higher Education and Research,
Chennai.

Sub: Brain stimulation techniques – A detailed review

Dear Sir,

With reference to the subject mentioned above, the department proposes to conduct a value-added course titled: **Brain stimulation techniques – A detailed review** on 01/04/2020. We solicit your kind permission for the same.

Kind Regards

Dr.V.R. Sridhar

FOR THE USE OF DEANS OFFICE

Names of Committee members for evaluating the course:

The Dean: **Dr. Balagurunathan. K**

The HOD: **Dr. V.R Sridhar**

The Expert: **Dr. Arun Seetharaman**

The committee has discussed about the course and is approved.

Dean	Subject Expert	HOD
(Sign & Seal)	(Sign & Seal)	(Sign & Seal)

DEAN
Prof.K.BALAGURUNATHAN M.S
(General surgeon)
SRI LAKSHMI NARAYANA
INSTITUTE OF MEDICAL SCIENCES
OSUDU PONDICHERRY

Dr. ARUN SEETHARAMAN, MD.,
Reg. No: 91440
Associate Professor, Psychiatry
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Kudapakkam, Puducherry-605 502.

Dr. V. R. SRIDHAR, MD.,D.P.M.,
Reg. No: 30995
Professor & HOD, Psychiatry
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Kudapakkam, Puducherry-605 502.

OFFICE OF THE DEAN

Sri Lakshmi Narayana Institute of Medical Sciences

OSUDU, AGARAM VILLAGE, VILLIANUR COMMUNE, KUDAPAKKAM POST,
PUDUCHERRY - 605 502.
[Recognised by Medical Council of India, Ministry of Health letter No. U/12012/249/2005-ME (P -II) dt. 11/07/2011]
[Affliated to Bharath University, Chennai - TN]

Circular

14.02.2020

Sub: Brain stimulation techniques – A detailed review

With reference to the above mentioned subject, it is to bring to your notice that Sri Lakshmi Narayana Institute of Medical Sciences, **Bharath Institute of Higher Education and Research** is organizing **"Brain stimulation techniques – A detailed review"**. The course content and registration form is enclosed below."

The application must reach the institution along with all the necessary documents as mentioned. The hard copy of the application should be sent to the institution by registered/ speed post only so as to reach on or before 31, feb 2020. Applications received after the mentioned date shall not be entertained under any circumstances.

Dean

DEAN
Prof.K.BALAGURUNATHAN M.S
(General surgeon)
SRI LAKSHMI NARAYANA
INSTITUTE OF MEDICAL SCIENCES
OSUDU PONDICHERRY

Encl: Copy of Course content

Course Proposal

Course Title: **Brain stimulation techniques – A detailed review**

Course Objective:

Electroconvulsive therapy
ECT – Indication
Pre-treatment evaluation for ECT
Trans-cranial Magnetic Stimulation
TMS – Indication
TMS – S/E, interactions with medications & other risks
Patient selection
Trans-cranial Direct Current Stimulation
TDCS – S/E
TDCS – Mechanism of Action
TDCS – clinical studies
Cranial Electric Stimulation
Magnetic seizure Therapy
Vagus nerve stimulation
Deep brain Stimulation

Course Outcome:

Course Audience: CRRI STUDENTS of 2020
Course Coordinator: Dr.V.R. Sridhar
Course Faculties with Qualification and Designation:
1.Dr.V.R.SHIDHAR, Professor & HOD
2.Dr.Arun, Associate Professor
3.Dr. Agila, Assistant Professor
Course Curriculum/Topics with schedule (Min of 30 hours)

SlNo	Date	Topic	Resource person	Time	Hours
1.	01.04.2020	INTRODUCTION TO BST Introduction	Dr.Arun	4-5p.m	1
2.	03.04.2020	ELECTRO-CONVULSIVE THERAPY	Dr.Arun	2-3p.m	1
3.	06.04.2020	ECT – Indication	Dr.Agila	4-6p.m	2
4.	08.04.2020	Pre-treatment evaluation for ECT	Dr.Arun	4-6p.m	2
5.	10.04.2020	Trans-cranial Magnetic Stimulation	Dr.Arun	4-6p.m	2
6.	13.04.2020	• TMS – Indication • TMS – S/E, interactions with medications & other risks • Patient selection	Dr.Arun	4-5p.m	2

7.	15.04.2020	• Trans-cranial Direct Current Stimulation • TDCS – S/E • TDCS – Mechanism of Action	Dr.Agila	4-5P.M	1
8.	17.04.2020	TDCS – clinical studies	Dr.Arun	4-5p.m	1
9.	20.04.2020	Cranial Electric Stimulation	Dr. Agila	4-6p.m	1
10.	22.04.2020	Magnetic seizure Therapy	Dr.Agila	4-6p.m	2
11.	24.04.2020	Vagus nerve stimulation	Dr.Agila	4-6p.m	1
12.	27.04.2020	Deep brain Stimulation	Dr.Agila	4-6p.m	2
13.	29.04.2020	Pre course and Post Course evaluation, Feedback analysis from Likert scale	Dr.Arun	2-5p.m	3
		Practical Class I	Dr. Shridhar		
13.	02.05.2020	ECT MACHINE DEMO	Dr. Shridhar	2-3 PM	1
14.	04.05.2020	ECT ON PATIENT DEMO	Dr. Shridhar	2-3 PM	1
15.	06.05.2020	CASE PRESENTATION	Dr. Shridhar	2-4 PM	2
16.	08.05.2020	DIRECT CURRENT DEMO	Dr. Shridhar	2-4 PM	2
17.	11.05.2020	CASE PRESENTATION	Dr. Shridhar	2-4p.m	2
		Total			30 hrs

REFERENCE BOOKS:

- **Comprehensive textbook of psychiatry – Kaplan & Saddock**
- **Oxford Textbook Of Psychiatry**
- **Synopsis - Kaplan & Saddock**
- **Stahl's essential psychopharmacology**

VALUE ADDED COURSE

1. Name of the programme & Code

Brain stimulation techniques – A detailed review

2. Duration & Period

April – June 2020

3. Information Brochure and Course Content of Value Added Courses

Enclosed as Annexure- I

4. List of students enrolled

Enclosed as Annexure- II

5. Assessment procedures:

Assessment Evolution by MCQ method - *Enclosed as Annexure- III*

6. Certificate model

Enclosed as Annexure- IV

7. No. of times offered during the same year:

1 TIME -April – June 2020

8. Year of discontinuation: 2020

9. Summary report of each program year-wise

Value Added Course: April – June 2020					
Sl. No	**Course Code**	**Course Name**	**Resource Persons**	**Target Students**	**Strength & Year**
1	**PSYC12**	Brain stimulation techniques – A detailed review	Dr. V.R. Sridhar Dr.Agila.c	CRRI Interns	10 students APRIL- JUNE 2020

10. Course Feed Back

Enclosed as Annexure- V

RESOURCE PERSON

1. **Dr.V.R. Sridhar**
2. **Dr. Arun Seetharaman**

COORDINATOR

Dr. .V.R. Sridhar

BRAIN STIMULATION THERAPIES

PARTICIPANT HAND BOOK

BIHER SLIMS

COURSE DETAILS

Particulars	**Description**
Course Title	Brain stimulation therapies
Course Code	PSYC12
Objective	1. Introduction **2.** Electroconvulsive therapy **3.** ECT – Indication 4. Pre-treatment evaluation for ECT 5. Trans-cranial Magnetic Stimulation 6. TMS – Indication 7. TMS – S/E, interactions with medications & other risks 8. Patient selection 9. Trans-cranial Direct Current Stimulation 10. TDCS – S/E 11. TDCS – Mechanism of Action 12. TDCS – clinical studies 13. Cranial Electric Stimulation 14. Magnetic seizure Therapy 15. Vagus nerve stimulation 16. Deep brain Stimulation
Further learning opportunities	Brain stimulation therapies & its application
Key Competencies	On successful completion of the course the students will have skill in handling and knows the application of brain stimulation therapies.
Target Student	CRRI
Duration	30hrs Every APRIL 2020– JUNE 2020
Theory Session	20hrs
Practical Session	10hrs
Assessment Procedure	Multiple choice questions

Introduction: Brain stimulation uses electrical currents or magnetic fields to alter neuronal firing. There is a growing list of tools capable of eliciting such neuromodulation, each with a different spectrum of action (trans-cranially or surgical implantation of electrodes). Trans-cranial techniques include cranial electrical stimulation (CES), electroconvulsive therapy (ECT), trans-cranial direct current stimulation (tDCS, also called direct current polarization), trans-cranial magnetic stimulation (TMS), and magnetic seizure therapy (MST). The surgical techniques include cortical brain stimulation (CBS), deep brain stimulation (DBS), and vagus nerve stimulation (VNS).

I. Electroconvulsive Therapy:

In 1938, the first electroconvulsive treatment (ECT) course was administered to a delusional and incoherent patient, who improved with 1 treatment and remitted after 11 treatments and in 1940, the first use of ECT occurred in the United States. There is a dose–response relationship with right unilateral ECT and that bilateral ECT is likely to be ineffective with ultra-brief pulse widths. ECT remains the most effective treatment for major depression and a rapidly effective treatment for life-threatening psychiatric conditions. The induction of a bilateral generalized seizure is necessary for both the beneficial and the adverse effects of ECT. ECT affects the cellular mechanisms of memory and mood regulation and raises the seizure threshold. The latter effect may be blocked by the opiate antagonist naloxone (Narcan). Electroconvulsive therapy

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From Wikipedia, the free encyclopedia
Jump to navigationJump to search
"Electroshock" redirects here. For other uses, see Electroshock (disambiguation).

Electroconvulsive therapy

MECTA spECTrum 5000Q with electroencephalography (EEG) in a modern ECT suite

Other names	Electroshock therapy
ICD-10-PCS	GZB
ICD-9-CM	94.27
MeSH	D004565
OPS-301 code	8-630
MedlinePlus	007474

[edit on Wikidata]

Electroconvulsive therapy (**ECT**), formerly known as **electroshock therapy**, is a psychiatric treatment in which seizures in the brain (without muscular convulsions) are electrically induced in patients to provide relief from mental disorders.[1] Typically, 70 to 120 volts are applied externally to the patient's head resulting in approximately 800 milliamperes of direct current passed through the brain, for 100 milliseconds to 6 seconds duration, either from temple to temple (bilateral ECT) or from front to back of one side of the head (unilateral ECT).

The ECT procedure was first conducted in 1938 by Italian psychiatrist Ugo Cerletti[2] and rapidly replaced less safe and effective forms of biological treatments in use at the time. ECT is often used with informed consent[3] as a

safe and effective intervention for major depressive disorder, mania, and catatonia.[4] ECT machines were originally placed in the Class III category by the United States Food and Drug Administration (FDA) in 1976.[5] They were re-classified as Class II devices, for treatment of catatonia, major depressive disorder, and bipolar disorder, in 2018.[6]

ECT is the most effective treatment that currently exists for depression. A course of ECT is effective for about 80–90% of people with treatment-resistant unipolar or bipolar depression.[7][8][9] Aside from effects on the brain, the general physical risks of ECT are similar to those of brief general anesthesia.[10]:259 Immediately following treatment, the most common adverse effects are confusion and transient memory loss.[4][11] Among treatments for severely depressed pregnant women, ECT is one of the least harmful to the fetus.[12]

A usual course of ECT involves multiple administrations, typically given two or three times per week until the patient is no longer suffering symptoms. ECT is administered under anesthesia with a muscle relaxant.[13] ECT can differ in its application in three ways: electrode placement, treatment frequency, and the electrical waveform of the stimulus. These treatment parameters can pose significant differences in both adverse side effects and symptom remission in the treated patient.

Placement can be bilateral, where the electric current is passed from one side of the brain to the other, or unilateral, in which the current is solely passed across one hemisphere of the brain. High-dose unilateral ECT has some cognitive advantages compared to moderate-dose bitlateral ECT while showing no difference in antidepressant efficacy.[14]

ECT appears to work in the short term via an anticonvulsant effect primarily in the frontal lobes and longer term via neurotrophic effects primarily in the medial temporal lobe.[15]

Contents

Medical use[edit]

ECT is used with informed consent[3] in treatment-resistant major depressive disorder, treatment-resistant catatonia, prolonged or severe mania, and in conditions where "there is a need for rapid, definitive response because of the severity of a psychiatric or medical condition (e.g., when illness is characterized by stupor, marked psychomotor retardation, depressive delusions or hallucinations, or life-threatening physical exhaustion associated with mania)."[4][16] It has also been used to treat autism in adults with an intellectual disability, yet findings from a systematic review found this an unestablished intervention.[17]

Major depressive disorder[edit]

For major depressive disorder, ECT is generally used only when other treatments have failed, or in emergencies, such as imminent

suicide.[4][18][19][20] ECT has also been used in selected cases of depression occurring in the setting of multiple sclerosis, Parkinson's disease, Huntington's chorea, developmental delay, brain arteriovenous malformations, and hydrocephalus.[21]

Efficacy[edit]

A meta-analysis on the effectiveness of ECT in unipolar and bipolar depression was conducted in 2012. Results indicated that although patients with unipolar depression and bipolar depression responded to other medical treatments very differently, both groups responded equally well to ECT. Overall remission rate for patients given a round of ECT treatment was 50.9% for those with unipolar depression and 53.2% for those with bipolar depression. The severity of each patient's depression was assessed at the same baseline in each group.[7]

There is little agreement on the most appropriate follow-up to ECT for people with major depressive disorder.[22] When ECT is followed by treatment with antidepressants, about 50% of people relapsed by 12 months following successful initial treatment with ECT, with about 37% relapsing within the first 6 months. About twice as many relapsed with no antidepressants. Most of the evidence for continuation therapy is with tricyclic antidepressants; evidence for relapse prevention with newer antidepressants is lacking.[22]

In 2004, a meta-analytic review paper found in terms of efficacy, "a significant superiority of ECT in all comparisons: ECT versus simulated ECT, ECT versus placebo, ECT versus antidepressants in general, ECT versus tricyclics and ECT versus monoamine oxidase inhibitors."[23]

In 2003, The UK ECT Review Group published a systematic review and meta-analysis comparing ECT to placebo and antidepressant drugs. This meta-analysis demonstrated a large effect size (high efficacy relative to the mean in terms of the standard deviation) for ECT versus placebo, and versus antidepressant drugs.[24]

Compared with repetitive transcranial magnetic stimulation (rTMS) for people with treatment-resistant major depressive disorder, ECT relieves depression as shown by reducing the score on the Hamilton Rating Scale for Depression by about 15 points, while rTMS reduced it by 9 points.[25]

Catatonia[edit]

ECT is generally a second-line treatment for people with catatonia who do not respond to other treatments, but is a first-line treatment for severe or life-threatening catatonia.[4][26][27] There is a plethora of evidence for its efficacy, notwithstanding a lack of randomised controlled trials, such that "the excellent efficacy of ECT in catatonia is generally acknowledged".[26] For people with autism spectrum disorders who have catatonia, there is little published

evidence about the efficacy of ECT; as of 2014 there were twelve case reports.[28]

Mania[edit]

ECT is used to treat people who have severe or prolonged mania;[4] NICE recommends it only in life-threatening situations or when other treatments have failed[29] and as a second-line treatment for bipolar mania.[30][31]

Schizophrenia[edit]

ECT is widely used worldwide in the treatment of schizophrenia, but in North America and Western Europe it is invariably used only in treatment resistant schizophrenia when symptoms show little response to antipsychotics; there is comprehensive research evidence for such practice.[32] It is useful in the case of severe exacerbations of catatonic schizophrenia, whether excited or stuporous.[4][29] There are also case reports of ECT improving persistent psychotic symptoms associated with Stimulant-induced psychosis.[33][34]

Effects[edit]

Aside from effects in the brain, the general physical risks of ECT are similar to those of brief general anesthesia; the U.S. Surgeon General's report says that there are "no absolute health contraindications" to its use.[10]:259 Immediately following treatment, the most common adverse effects are confusion and memory loss. Some patients experience muscle soreness after ECT. The death rate during ECT is around 4 per 100,000 procedures.[35] There is evidence and rationale to support giving low doses of benzodiazepines or otherwise low doses of general anesthetics, which induce sedation but not anesthesia, to patients to reduce adverse effects of ECT.[36]

While there are no absolute contraindications for ECT, there is increased risk for patients who have unstable or severe cardiovascular conditions or aneurysms; who have recently had a stroke; who have increased intracranial pressure (for instance, due to a solid brain tumor), or who have severe pulmonary conditions, or who are generally at high risk for receiving anesthesia.[11]:30

In adolescents, ECT is highly efficient for several psychiatric disorders, with few and relatively benign adverse effects.[37][38][39]

Cognitive impairment[edit]

Cognitive impairment is sometimes noticed after ECT.[40][41][42][43] It has been claimed by some non-medical authors that retrograde amnesia occurs to some extent in almost all patients receiving ECT.[44] However, most experts consider this adverse effect relatively uncommon.[45] The American Psychiatric Association (APA) report in 2001 acknowledges: “In some patients the recovery

from retrograde amnesia will be incomplete, and evidence has shown that ECT can result in persistent or permanent memory loss".[11] After treatment, drug therapy is usually continued and some patients will continue to receive maintenance ECT treatments.[4] It is the purported effects of ECT on long-term memory that give rise to much of the concern surrounding its use.[46] However, the methods used to measure memory loss are generally poor, and their application to people with depression, who have cognitive deficits including problems with memory, have been problematic.[45]

The acute effects of ECT can include amnesia, both retrograde (for events occurring before the treatment) and anterograde (for events occurring after the treatment).[47] Memory loss and confusion are more pronounced with bilateral electrode placement rather than unilateral, and with outdated sine-wave rather than brief-pulse currents. The use of either constant or pulsing electrical impulses also varied the memory loss results in patients. Patients who received pulsing electrical impulses, as opposed to a steady flow, seemed to incur less memory loss. The vast majority of modern treatment uses brief pulse currents.[47]

Retrograde amnesia is most marked for events occurring in the weeks or months before treatment, with one study showing that although some people lose memories from years prior to treatment, recovery of such memories was "virtually complete" by seven months post-treatment, with the only enduring loss being memories in the weeks and months prior to the treatment.[48][49] Anterograde memory loss is usually limited to the time of treatment itself or shortly afterwards. In the weeks and months following ECT these memory problems gradually improve, but some people have persistent losses, especially with bilateral ECT.[1][47] One published review summarizing the results of questionnaires about subjective memory loss found that between 29% and 55% of respondents believed they experienced long-lasting or permanent memory changes.[50] In 2000, American psychiatrist Sarah Lisanby and colleagues found that bilateral ECT left patients with more persistently impaired memory of public events as compared to right unilateral ECT.[46]

Effects on brain structure[edit]

Considerable controversy exists over the effects of ECT on brain tissue, although a number of mental health associations—including the APA—have concluded that there is no evidence that ECT causes structural brain damage.[11][19] A 1999 report by the U.S. Surgeon General states: "The fears that ECT causes gross structural brain pathology have not been supported by decades of methodologically sound research in both humans and animals."[51]

Many expert proponents of ECT maintain that the procedure is safe and does not cause brain damage. Dr. Charles Kellner, a prominent ECT researcher and

former chief editor of the *Journal of ECT*, stated in a 2007 interview that, "There are a number of well-designed studies that show ECT does not cause brain damage and numerous reports of patients who have received a large number of treatments over their lifetime and have suffered no significant problems due to ECT."[52] Dr. Kellner cites a study purporting to show an absence of cognitive impairment in eight subjects after more than 100 lifetime ECT treatments.[53] Dr. Kellner stated "Rather than cause brain damage, there is evidence that ECT may reverse some of the damaging effects of serious psychiatric illness."

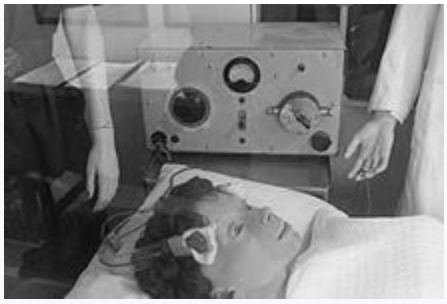
Effects in pregnancy[edit]

If steps are taken to decrease potential risks, ECT is generally accepted to be relatively safe during all trimesters of pregnancy, particularly when compared to pharmacological treatments.[12][54] Suggested preparation for ECT during pregnancy includes a pelvic examination, discontinuation of nonessential anticholinergic medication, uterine tocodynamometry, intravenous hydration, and administration of a nonparticulate antacid. During ECT, elevation of the pregnant woman's right hip, external fetal cardiac monitoring, intubation, and avoidance of excessive hyperventilation are recommended.[12] In many instances of active mood disorder during pregnancy, the risks of untreated symptoms may outweigh the risks of ECT. Potential complications of ECT during pregnancy can be minimized by modifications in technique. The use of ECT during pregnancy requires thorough evaluation of the patient's capacity for informed consent.[55]

Effects on the heart[edit]

ECT can cause a lack of blood flow and oxygen to the heart, heart arrhythmia, and "persistent asystole". Deaths, however, are very rare after ECT: 6 per 100,000 treatments. If they do occur, cardiovascular complications are considered as causal in about 30%.[56]

Technique[edit]



Electroconvulsive therapy machine on display at Glenside Museum in Bristol, England

ECT device produced by Siemens and used for example at the Asyl psychiatric hospital in Kristiansand, Norway from the 1960s to the 1980s.

ECT requires the informed consent of the patient.[1]:1880[3][4]

Whether psychiatric medications are terminated prior to treatment or maintained, varies.[1]:1885[57] However, drugs that are known to cause toxicity in combination with ECT, such as lithium, are discontinued, and benzodiazepines, which increase the seizure threshold,[58] are either discontinued, a benzodiazepine antagonist is administered at each ECT session, or the ECT treatment is adjusted accordingly.[1]:1879:1875

In the US, the medical team performing the procedure typically consists of a psychiatrist, an anesthetist, an ECT treatment nurse or qualified assistant, and one or more recovery nurses.[11]:109 Medical trainees may assist, but only under the direct supervision of credentialed attending physicians and staff.[11]:110 The placement of electrodes, as well as the dose and duration of the stimulation is determined on a per-patient basis.[1]:1881

In unilateral ECT, both electrodes are placed on the same side of the patient's head. Unilateral ECT may be used first to minimize side effects such as memory loss.

In bilateral ECT, the two electrodes are placed on opposite sides of the head. Usually bitemporal placement is used, whereby the electrodes are placed on the temples. Uncommonly bifrontal placement is used; this involves positioning the electrodes on the patient's forehead, roughly above each eye.

Unilateral ECT is thought to cause fewer cognitive effects than bilateral treatment, but is less effective unless administered at higher doses.[1]:1881 Most patients in the US[59] and almost all in the UK[60][61][62] receive bilateral ECT.

The electrodes deliver an electrical stimulus. The stimulus levels recommended for ECT are in excess of an individual's seizure threshold: about one and a half times seizure threshold for bilateral ECT and up to 12 times for unilateral ECT.[1]:1881 Below these levels treatment may not be effective in spite of a seizure, while doses massively above threshold level, especially with bilateral ECT, expose patients to the risk of more severe cognitive impairment without additional therapeutic gains.[63] Seizure threshold is determined by trial and

error ("dose titration"). Some psychiatrists use dose titration, some still use "fixed dose" (that is, all patients are given the same dose) and others compromise by roughly estimating a patient's threshold according to age and sex.[59] Older men tend to have higher thresholds than younger women, but it is not a hard and fast rule, and other factors, for example drugs, affect seizure threshold.

Immediately prior to treatment, a patient is given a short-acting anesthetic such as methohexital, etomidate, or thiopental,[1] a muscle relaxant such as suxamethonium (succinylcholine), and occasionally atropine to inhibit salivation.[1]:1882 In a minority of countries such as Japan,[64] India,[65] and Nigeria,[66] ECT may be used without anesthesia. The Union Health Ministry of India recommended a ban on ECT without anesthesia in India's Mental Health Care Bill of 2010 and the Mental Health Care Bill of 2013.[67][68] The practice was abolished in Turkey's largest psychiatric hospital in 2008.[69]

The patient's EEG, ECG, and blood oxygen levels are monitored during treatment.[1]:1882

ECT is usually administered three times a week, on alternate days, over a course of two to four weeks.[1]:1882–1883

An illustration depicting electroconvulsive therapy.

Devices[edit]


ECT machine from before 1960.

Most modern ECT devices deliver a brief-pulse current, which is thought to cause fewer cognitive effects than the sine-wave currents which were originally used in ECT.[1] A small minority of psychiatrists in the US still use sine-wave stimuli.[59] Sine-wave is no longer used in the UK or Ireland.[62] Typically, the electrical stimulus used in ECT is about 800 milliamps and has up to several hundred watts, and the current flows for between one and six seconds.[63]

In the US, ECT devices are manufactured by two companies, Somatics, which is owned by psychiatrists Richard Abrams and Conrad Swartz, and Mecta.[70] In the UK, the market for ECT devices was long monopolized by Ectron Ltd, which was set up by psychiatrist Robert Russell.[71]

Mechanism of action[edit]

Despite decades of research, the exact mechanism of action of ECT remains elusive. Neuroimaging studies in people who have had ECT, investigating differences between responders and nonresponders, and people who relapse, find that responders have anticonvulsant effects mostly in the frontal lobes, which corresponds to immediate responses, and neurotrophic effects primarily in the medial temporal lobe. The anticonvulsant effects are decreased blood flow and decreased metabolism, while the neurotrophic effects are opposite - increased perfusion and metabolism, as well as increased volume of the hippocampus.[15]

A recently proposed mechanism of action is that the seizures induced by ECT cause a profound change in sleep architecture; it is this change in the state of the organism that drives the therapeutic effects of ECT and not any simple change in the release of neurotransmitters, neurotrophic factors and/or hormones.[72]

Use[edit]

As of 2001, it was estimated that about one million people received ECT annually.[73]

There is wide variation in ECT use between different countries, different hospitals, and different psychiatrists.[1][73] International practice varies considerably from widespread use of the therapy in many Western countries to a small minority of countries that do not use ECT at all, such as Slovenia.[74]

About 70 percent of ECT patients are women.[1] This may be due to the fact that women are more likely to be diagnosed with depression.[1][75] Older and more affluent patients are also more likely to receive ECT. The use of ECT is not as common in ethnic minorities.[75][76]

Sarah Hall reports, "ECT has been dogged by conflict between psychiatrists who swear by it, and some patients and families of patients who say that their lives have been ruined by it. It is controversial in some European countries such as the Netherlands and Italy, where its use is severely restricted".[77]

United States[edit]

ECT became popular in the US in the 1940s. At the time, psychiatric hospitals were overrun with patients whom doctors were desperate to treat and cure. Whereas lobotomies would reduce a patient to a more manageable submissive state, ECT helped to improve mood in those with severe depression. A survey

of psychiatric practice in the late 1980s found that an estimated 100,000 people received ECT annually, with wide variation between metropolitan statistical areas.[78] Accurate statistics about the frequency, context and circumstances of ECT in the US are difficult to obtain because only a few states have reporting laws that require the treating facility to supply state authorities with this information.[79] In 13 of the 50 states, the practice of ECT is regulated by law.[80] In the mid-1990s in Texas, ECT was used in about one third of psychiatric facilities and given to about 1,650 people annually.[75] Usage of ECT has since declined slightly; in 2000–01 ECT was given to about 1500 people aged from 16 to 97 (in Texas it is illegal to give ECT to anyone under sixteen).[81] ECT is more commonly used in private psychiatric hospitals than in public hospitals, and minority patients are underrepresented in the ECT statistics.[1] In the United States, ECT is usually given three times a week; in the United Kingdom, it is usually given twice a week.[1] Occasionally it is given on a daily basis.[1] A course usually consists of 6–12 treatments, but may be more or fewer. Following a course of ECT some patients may be given continuation or maintenance ECT with further treatments at weekly, fortnightly or monthly intervals.[1] A few psychiatrists in the US use multiple-monitored ECT (MMECT), where patients receive more than one treatment per anesthetic.[1] Electroconvulsive therapy is not a required subject in US medical schools and not a required skill in psychiatric residency training. Privileging for ECT practice at institutions is a local option: no national certification standards are established, and no ECT-specific continuing training experiences are required of ECT practitioners.[82]

United Kingdom[edit]

In the UK in 1980, an estimated 50,000 people received ECT annually, with use declining steadily since then[83] to about 12,000 per annum in 2002.[84] It is still used in nearly all psychiatric hospitals, with a survey of ECT use from 2002 finding that 71 percent of patients were women and 46 percent were over 65 years of age. Eighty-one percent had a diagnosis of mood disorder; schizophrenia was the next most common diagnosis. Sixteen percent were treated without their consent.[84] In 2003, the National Institute for Health and Care Excellence, a government body which was set up to standardize treatment throughout the National Health Service in England and Wales, issued guidance on the use of ECT. Its use was recommended "only to achieve rapid and short-term improvement of severe symptoms after an adequate trial of treatment options has proven ineffective and/or when the condition is considered to be potentially life-threatening in individuals with severe depressive illness, catatonia or a prolonged manic episode".[85]

The guidance received a mixed reception. It was welcomed by an editorial in the *British Medical Journal*[86] but the Royal College of Psychiatrists launched an unsuccessful appeal.[87] The NICE guidance, as the *British Medical*

Journal editorial points out, is only a policy statement and psychiatrists may deviate from it if they see fit. Adherence to standards has not been universal in the past. A survey of ECT use in 1980 found that more than half of ECT clinics failed to meet minimum standards set by the Royal College of Psychiatrists, with a later survey in 1998 finding that minimum standards were largely adhered to, but that two-thirds of clinics still fell short of current guidelines, particularly in the training and supervision of junior doctors involved in the procedure.[88] A voluntary accreditation scheme, ECTAS, was set up in 2004 by the Royal College, and as of 2017 the vast majority of ECT clinics in England, Wales, Northern Ireland and the Republic of Ireland have signed up.[89]

The Mental Health Act 2007 allows people to be treated against their will. This law has extra protections regarding ECT. A patient capable of making the decision can decline the treatment, and in that case treatment cannot be given unless it will save that patient's life or is immediately necessary to prevent deterioration of the patient's condition. A patient may not be capable of making the decision (they "lack capacity"), and in that situation ECT can be given if it is appropriate and also if there are no advance directives that prevent the use of ECT.[90]

China[edit]

ECT was introduced in China in the early 1950s and while it was originally practiced without anesthesia, as of 2012 almost all procedures were conducted with it. As of 2012, there are approximately 400 ECT machines in China, and 150,000 ECT treatments are performed each year.[91] Chinese national practice guidelines recommend ECT for the treatment of schizophrenia, depressive disorders, and bipolar disorder and in the Chinese literature, ECT is an effective treatment for schizophrenia and mood disorders.[91] Although the Chinese government stopped classifying homosexuality as an illness in 2001, electroconvulsive therapy is still used by some establishments as a form of "conversion therapy".[92][93]

History[edit]

A *Bergonic chair*, a device "for giving general electric treatment for psychological effect, in psycho-neurotic cases", according to original photo description. World War I era.

Further information: History of electroconvulsive therapy in the United Kingdom and History of electroconvulsive therapy in the United States

As early as the 16th century, agents to induce seizures were used to treat psychiatric conditions. In 1785, the therapeutic use of seizure induction was documented in the *London Medical and Surgical Journal*.[1][94][95] As to its earliest antecedents one doctor claims 1744 as the dawn of electricity's therapeutic use, as documented in the first issue of *Electricity and Medicine*. Treatment and cure of hysterical blindness was documented eleven years later. Benjamin Franklin wrote that an electrostatic machine cured "a woman of hysterical fits." In 1801, Giovanni Aldini used galvanism to treat patients suffering from various mental disorders.[96] G.B.C. Duchenne, the mid-19th century "Father of Electrotherapy", said its use was integral to a neurological practice.[97]

In the second half of the 19th century, such efforts were frequent enough in British asylums as to make it notable.[98]

Convulsive therapy was introduced in 1934 by Hungarian neuropsychiatrist Ladislas J. Meduna who, believing mistakenly that schizophrenia and epilepsy were antagonistic disorders, induced seizures first with camphor and then metrazol (cardiazol).[99][100] Meduna is thought to be the father of convulsive therapy.[101] In 1937, the first international meeting on schizophrenia and convulsive therapy was held in Switzerland by the Swiss psychiatrist Max Müller.[102] The proceedings were published in the *American Journal of Psychiatry* and, within three years, cardiazol convulsive therapy was being used worldwide.[100] Italian Professor of neuropsychiatry Ugo Cerletti, who had been using electric shocks to produce seizures in animal experiments, and his assistant Lucio Bini at Sapienza University of Rome developed the idea of using electricity as a substitute for metrazol in convulsive therapy and, in 1938, experimented for the first time on a person affected by delusions. It was believed early on that inducing convulsions aided in helping those with severe schizophrenia but later found to be most useful with affective disorders such as depression. Cerletti had noted a shock to the head produced convulsions in dogs. The idea to use electroshock on humans came to Cerletti when he saw how pigs were given an electric shock before being butchered to put them in an anesthetized state.[103] Cerletti and Bini practiced until they felt they had the right parameters needed to have a successful human trial. Once they started trials on patients, they found that after 10-20 treatments the results were significant. Patients had much improved. A positive side effect to the treatment was retrograde amnesia. It was because of this side effect that patients could not remember the treatments and had no ill feelings toward it.[103] ECT soon replaced metrazol therapy all over the world because it was cheaper, less frightening and more convenient.[104] Cerletti and Bini were nominated for a Nobel Prize but did not receive one. By 1940, the procedure was introduced to

both England and the US. In Germany and Austria, it was promoted by Friedrich Meggendorfer. Through the 1940s and 1950s, the use of ECT became widespread. At the time the ECT device was patented and commercialized abroad, the two Italian inventors had competitive tensions that damaged their relationship.[105] In the 1960s, despite a climate of condemnation, the original Cerletti-Bini ECT apparatus prototype was hotly contended by scientific museums between Italy and the USA[106] The ECT apparatus prototype is now owned and displayed by the Sapienza Museum of the History of Medicine in Rome.[107]

In the early 1940s, in an attempt to reduce the memory disturbance and confusion associated with treatment, two modifications were introduced: the use of unilateral electrode placement and the replacement of sinusoidal current with brief pulse. It took many years for brief-pulse equipment to be widely adopted.[108] In the 1940s and early 1950s ECT, was usually given in "unmodified" form, without muscle relaxants, and the seizure resulted in a full-scale convulsion. A rare but serious complication of unmodified ECT was fracture or dislocation of the long bones. In the 1940s, psychiatrists began to experiment with curare, the muscle-paralysing South American poison, in order to modify the convulsions. The introduction of suxamethonium (succinylcholine), a safer synthetic alternative to curare, in 1951 led to the more widespread use of "modified" ECT. A short-acting anesthetic was usually given in addition to the muscle relaxant in order to spare patients the terrifying feeling of suffocation that can be experienced with muscle relaxants.[108]

The steady growth of antidepressant use along with negative depictions of ECT in the mass media led to a marked decline in the use of ECT during the 1950s to the 1970s. The Surgeon General stated there were problems with electroshock therapy in the initial years before anesthesia was routinely given, and that "these now-antiquated practices contributed to the negative portrayal of ECT in the popular media."[109] *The New York Times* described the public's negative perception of ECT as being caused mainly by one movie: "For Big Nurse in *One Flew Over the Cuckoo's Nest,* it was a tool of terror, and, in the public mind, *shock therapy* has retained the tarnished image given it by Ken Kesey's novel: dangerous, inhumane and overused".[110]

In 1976, Dr. Blatchley demonstrated the effectiveness of his constant current, brief pulse device ECT. This device eventually largely replaced earlier devices because of the reduction in cognitive side effects, although as of 2012 some ECT clinics still were using sine-wave devices.[73] The 1970s saw the publication of the first American Psychiatric Association (APA) task force report on electroconvulsive therapy (to be followed by further reports in 1990 and 2001). The report endorsed the use of ECT in the treatment of depression. The decade also saw criticism of ECT.[111] Specifically, critics pointed to

shortcomings such as noted side effects, the procedure being used as a form of abuse, and uneven application of ECT. The use of ECT declined until the 1980s, "when use began to increase amid growing awareness of its benefits and cost-effectiveness for treating severe depression".[109] In 1985, the National Institute of Mental Health and National Institutes of Health convened a consensus development conference on ECT and concluded that, while ECT was the most controversial treatment in psychiatry and had significant side-effects, it had been shown to be effective for a narrow range of severe psychiatric disorders.[112]

Because of the backlash noted previously, national institutions reviewed past practices and set new standards. In 1978, the American Psychiatric Association released its first task force report in which new standards for consent were introduced and the use of unilateral electrode placement was recommended. The 1985 NIMH Consensus Conference confirmed the therapeutic role of ECT in certain circumstances. The American Psychiatric Association released its second task force report in 1990 where specific details on the delivery, education, and training of ECT were documented. Finally, in 2001 the American Psychiatric Association released its latest task force report.[11] This report emphasizes the importance of informed consent, and the expanded role that the procedure has in modern medicine. By 2017, ECT was routinely covered by insurance companies for providing the "biggest bang for the buck" for otherwise intractable cases of severe mental illness, was receiving favorable media coverage, and was being provided in regional medical centers.[113]

Though ECT use declined with the advent of modern antidepressants, there has been a resurgence of ECT with new modern technologies and techniques.[114] Modern shock voltage is given for a shorter duration of 0.5 milliseconds where conventional brief pulse is 1.5 milliseconds.[115]

Society and culture[edit]

Controversy[edit]

Surveys of public opinion, the testimony of former patients, legal restrictions on the use of ECT and disputes as to the efficacy, ethics and adverse effects of ECT within the psychiatric and wider medical community indicate that the use of ECT remains controversial.[116][117][118][119][120][121][122] This is reflected in the January 2011 vote by the FDA's Neurological Devices Advisory Panel to recommend that FDA maintain ECT devices in the Class III device category for high risk devices, except for patients suffering from catatonia, major depressive disorder, and bipolar disorder.[6] This may result in the manufacturers of such devices having to do controlled trials on their safety and efficacy for the first time.[4][123][124] In justifying their position, panelists referred to the memory loss associated with ECT and the lack of long-term data.[125]

Legal status[edit]

Informed consent[edit]

The World Health Organization (2005) advises that ECT should be used only with the informed consent of the patient (or their guardian if their incapacity to consent has been established).[16]

In the US, this doctrine places a legal obligation on a doctor to make a patient aware of the reason for treatment, the risks and benefits of a proposed treatment, the risks and benefits of alternative treatment, and the risks and benefits of receiving no treatment. The patient is then given the opportunity to accept or reject the treatment. The form states how many treatments are recommended and also makes the patient aware that consent may be revoked and treatment discontinued at any time during a course of ECT.[10] The US Surgeon General's Report on Mental Health states that patients should be warned that the benefits of ECT are short-lived without active continuation treatment in the form of drugs or further ECT, and that there may be some risk of permanent, severe memory loss after ECT.[10] The report advises psychiatrists to involve patients in discussion, possibly with the aid of leaflets or videos, both before and during a course of ECT.

To demonstrate what he believes should be required to fully satisfy the legal obligation for informed consent, one psychiatrist, working for an anti-psychiatry organisation, has formulated his own consent form[126] using the consent form developed and enacted by the Texas Legislature[127] as a model.

According to the US Surgeon General, involuntary treatment is uncommon in the US and is typically used only in cases of great extremity, and only when all other treatment options have been exhausted. The use of ECT is believed to be a potentially life-saving treatment.[51]

In one of the few jurisdictions where recent statistics on ECT usage are available, a national audit of ECT by the Scottish ECT Accreditation Network indicated that 77% of patients who received the treatment in 2008 were capable of giving informed consent.[128]

In the UK, in order for consent to be valid it requires an explanation in "broad terms" of the nature of the procedure and its likely effects.[129] One review from 2005 found that only about half of patients felt they were given sufficient information about ECT and its adverse effects[130] and another survey found that about fifty percent of psychiatrists and nurses agreed with them.[131]

A 2005 study published in the *British Journal of Psychiatry* described patients' perspectives on the adequacy of informed consent before ECT.[130] The study found that "About half (45–55%) of patients reported they were given an adequate explanation of ECT, implying a similar percentage felt they were not." The authors also stated:

Approximately a third did not feel they had freely consented to ECT even when they had signed a consent form. The proportion who feel they did not freely choose the treatment has actually increased over time. The same themes arise whether the patient had received treatment a year ago or 30 years ago. Neither current nor proposed safeguards for patients are sufficient to ensure informed consent with respect to ECT, at least in England and Wales.[130]

Involuntary ECT[edit]

This section **needs expansion**. You can help by adding to it. *(June 2017)*

Procedures for involuntary ECT vary from country to country depending on local mental health laws.

United States[edit]

In most states in the US, a judicial order following a formal hearing is needed before a patient can be forced to undergo involuntary ECT.[10] However, ECT can also be involuntarily administered in situations with less immediate danger. Suicidal intent is a common justification for its involuntary use, especially when other treatments are ineffective.[10]

United Kingdom[edit]

Until 2007 in England and Wales, the Mental Health Act 1983 allowed the use of ECT on detained patients whether or not they had capacity to consent to it. However, following amendments which took effect in 2007, ECT may not generally be given to a patient who has capacity and refuses it, irrespective of his or her detention under the Act.[132] In fact, even if a patient is deemed to lack capacity, if they made a valid advance decision refusing ECT then they should not be given it; and even if they do not have an advance decision, the psychiatrist must obtain an independent second opinion (which is also the case if the patient is under age of consent).[133] However, there is an exception regardless of consent and capacity; under Section 62 of the Act, if the treating psychiatrist says the need for treatment is urgent they may start a course of ECT without authorization.[134] From 2003 to 2005, about 2,000 people a year in England and Wales were treated without their consent under the Mental Health Act.[135] Concerns have been raised by the official regulator that psychiatrists are too readily assuming that patients have the capacity to consent to their treatments, and that there is a worrying lack of independent advocacy.[136] In Scotland, the Mental Health (Care and Treatment) (Scotland) Act 2003 also gives patients with capacity the right to refuse ECT.[137]

Regulation[edit]

In the US, ECT devices came into existence prior to medical devices being regulated by the Food and Drug Administration. In 1976, the Medical Device Regulation Act required the FDA to retrospectively review already existing devices, classify them, and determine whether clinical trials were needed to prove efficacy and safety. The FDA initially classified the devices used to administer ECT as Class III medical devices. In 2014, the American Psychiatric Association petitioned the FDA to reclassify ECT devices from Class III (high-risk) to Class II (medium-risk), which would significantly improve access to an effective and potentially lifesaving treatment. A similar reclassification proposal in 2010 met significant resistance from anti-psychiatry groups and did not pass.[138] In 2018, the FDA re-classified ECT devices as Class II devices when used to treat catatonia or a severe major depressive episode associated with major depressive disorder or bipolar disorder.[6]

Public perception[edit]

A questionnaire survey of 379 members of the general public in Australia indicated that more than 60% of respondents had some knowledge about the main aspects of ECT. Participants were generally opposed to the use of ECT on depressed individuals with psychosocial issues, on children, and on involuntary patients. Public perceptions of ECT were found to be mainly negative.[122] A sample of the general public, medical students, and psychiatry trainees in the United Kingdom found that the psychiatry trainees were more knowledgeable and had more favorable opinions of ECT than did the other groups.[139] More members of the general public believed that ECT was used for control or punishment purposes than medical students or psychiatry trainees.[139]

Famous cases[edit]

Main article: List of people who have undergone electroconvulsive therapy

Ernest Hemingway, an American author, died by suicide shortly after ECT at the Mayo Clinic in 1961.[140] He is reported to have said to his biographer, "Well, what is the sense of ruining my head and erasing my memory, which is my capital, and putting me out of business? It was a brilliant cure but we lost the patient."[141] Robert Pirsig suffered a nervous breakdown and spent time in and out of psychiatric hospitals between 1961 and 1963.[142] He was diagnosed with paranoid schizophrenia and clinical depression as a result of an evaluation conducted by psychoanalysts, and was treated with electroconvulsive therapy on numerous occasions,[143] a treatment he discusses in his novel, *Zen and the Art of Motorcycle Maintenance*.[144]

Thomas Eagleton, United States Senator from Missouri, was dropped from the Democratic ticket in the 1972 United States Presidential Election as the party's Vice Presidential candidate after it was revealed that he had received electroshock treatment in the past for depression. Presidential nominee George

McGovern replaced him with Sargent Shriver, and later went on to lose by a landslide to Richard Nixon.

American surgeon and award-winning author Sherwin B. Nuland is another notable person who has undergone ECT.[145] In his 40s, this successful surgeon's depression became so severe that he had to be institutionalized. After exhausting all treatment options, a young resident assigned to his case suggested ECT, which ended up being successful.[146] Author David Foster Wallace also received ECT for many years, beginning as a teenager, before his suicide at age 46.[147]

New Zealand author Janet Frame experienced both insulin coma therapy and ECT (but without the use of anesthesia or muscle relaxants).[148] She wrote about this in her autobiography, *An Angel at My Table* (1984),[148] which was later adapted into a film (1990).[149]

American actor Carrie Fisher wrote about her experience with memory loss after ECT treatments in her memoir *Wishful Drinking*.[150]

Fictional examples[edit]

Electroconvulsive therapy has been depicted in fiction, including fictional works partly based on true experiences. These include Sylvia Plath's autobiographical novel, *The Bell Jar*, Ken Loach's film Family Life, and Ken Kesey's novel *One Flew Over the Cuckoo's Nest*; Kesey's novel is a direct product of his time working the graveyard shift as an orderly at a mental health facility in Menlo Park, California.[151][152]

In the 2000 film *Requiem for a Dream*, Sarah Goldfarb receives "unmodified" electroconvulsive therapy after experiencing severe amphetamine psychosis following prolonged stimulant abuse. Unlike typical ECT treatment, she is given no anesthetic or medication before.

In the 2014 TV series *Constantine*, the protagonist John Constantine is institutionalized and specifically requests electroconvulsive therapy as an attempt to alleviate or resolve his mental problems.

The musical *Next to Normal* revolves around the family of a woman who undergoes the procedure.

In the HBO series *Six Feet Under* season 5, George undergoes an ECT treatment to deal with his increasing paranoia. The depiction is shown realistically, with an actual ECT machine.

In the WB/CW TV series *Smallville*, Lionel Luthor condemns his son Lex Luthor to electroshock therapy to remove Lex's short-term memory of a murder he discovered Lionel committed.

In the Netflix series *Stranger Things*, Eleven's mother is given electroshock therapy to silence her.

Electroshock therapy is used on various characters throughout season 2 of American Horror Story.

Special populations[edit]

Sex difference[edit]

Throughout the history of ECT, women have received it two to three times as often as men.[153] Currently, about 70 percent of ECT patients are women.[1] This may be due to the fact that women are more likely to be diagnosed with depression.[1][75] A 1974 study of ECT in Massachusetts reported that women made up 69 percent of those given ECT.[154] The Ministry of Health in Canada reported that from 1999 until 2000 in the province of Ontario, women were 71 percent of those given ECT in provincial psychiatric institutions, and 75 percent of the total ECT given was given to women.[155

II. Trans-cranial Magnetic Stimulation:

TMS induces electrical fields in the brain without an electrode through the application of alternating magnetic fields via a coil held on the scalp. It is a non-invasive stimulation of focal regions of the brain without the need for anaesthesia.

A. Indications. It is approved by the FDA for the treatment of MDD in adult patients who have failed to achieve satisfactory improvement from one prior antidepressant medication at or above the minimal effective dose and duration in the current episode.

B. Side effects, interactions with medications, and other risks. Administration of TMS is a non-invasive, relatively benign procedure but it is not entirely without risk, the most serious being an unintended seizure.

C. Patient selection. Patients who have failed a trial of one or more antidepressant medications or have untoward side effects to medications may be good candidates for TMS. However, given the lower effect size of TMS, for urgent or severely refractory cases, ECT would remain the ultimate gold standard treatment.

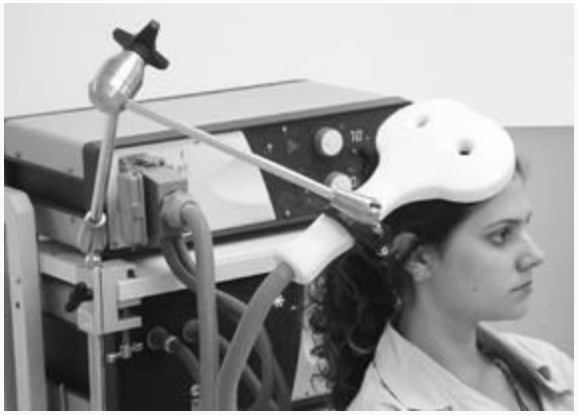
Transcranial magnetic stimulation (**TMS**) is a noninvasive form of brain stimulation in which a changing magnetic field is used to cause electric current at a specific area of the brain through electromagnetic induction. An electric pulse generator, or stimulator, is connected to a magnetic coil, which in turn is connected to the scalp. The stimulator generates a changing electric

current within the coil which induces a magnetic field; this field then causes a second inductance of inverted electric charge within the brain itself.[1]:3[2]

TMS has shown diagnostic and therapeutic potential in the central nervous system with a wide variety of disease states in neurology and mental health, with research still evolving.[3][4][5][6][7][8][9][10]

Adverse effects of TMS are rare, and include fainting and seizure.[11] Other potential issues include discomfort, pain, hypomania, cognitive change, hearing loss, and inadvertent current induction in implanted devices such as pacemakers or defibrillators.[11]

Contents



A magnetic coil is positioned at the head of the person[12]

TMS is non-invasive, and does not require surgery or electrode implantation. Its use can be divided into diagnostic and therapeutic applications. Effects vary based on frequency and intensity of the magnetic pulses as well as the length of the train, which affects the total number of pulses given.[*citation needed*] TMS treatments are now approved by the FDA in the USA and by NICE in the UK for the treatment of depression and are predominantly provided by private clinics.

Diagnosis[edit]

TMS can be used clinically to measure activity and function of specific brain circuits in humans, most commonly with single or paired magnetic pulses.[3] The most widely accepted use is in measuring the connection between the primary motor cortex of the central nervous system and the peripheral nervous system to evaluate damage related to past or progressive neurologic insult.[3][13][14][15]

Treatment[edit]

Repetitive high frequency TMS (rTMS) has shown diagnostic and therapeutic potential with the central nervous system in a variety of disease states, particularly in the fields of neurology and mental health.[3][4][5][7][8][9][10]

Adverse effects[edit]

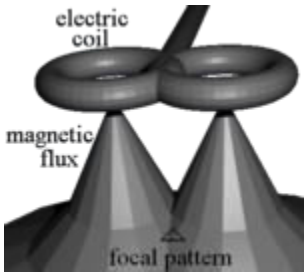
Although TMS is generally regarded as safe, risks are increased for therapeutic rTMS compared to single or paired diagnostic TMS.[16] Adverse effects generally increase with higher frequency stimulation.[11]

The greatest immediate risk from TMS is fainting, though this is uncommon. Seizures have been reported, but are rare.[11][17][18] Other adverse effects include short term discomfort, pain, brief episodes of hypomania, cognitive change, hearing loss, impaired working memory, and the induction of electrical currents in implanted devices such as cardiac pacemakers.[11]

Procedure[edit]

During the procedure, a magnetic coil is positioned at the head of the person receiving the treatment using anatomical landmarks on the skull, in particular the inion and nasion.[12] The coil is then connected to a pulse generator, or stimulator, that delivers electric current to the coil.[2]

Physics[edit]



TMS – Butterfly Coils

TMS uses electromagnetic induction to generate an electric current across the scalp and skull.[19][20] A plastic-enclosed coil of wire is held next to the skull and when activated, produces a magnetic field oriented orthogonal to the plane of the coil. The magnetic field can then be directed to induce an inverted electric current in the brain that activates nearby nerve cells in a manner similar to a current applied superficially at the cortical surface.[21]

The magnetic field is about the same strength as an MRI, and the pulse generally reaches no more than 5 centimeters into the brain, unless using a modified coil and technique for deeper stimulation.[20]

From the Biot–Savart law,

it has been shown that a current through a wire generates a magnetic field around that wire. Transcranial magnetic stimulation is achieved by quickly discharging current from a large capacitor into a coil to produce pulsed magnetic fields between 2 and 3 Tesla in strength.[22] Directing the magnetic field pulse at a targeted area in the brain causes a localized electrical current which can then either depolarize or hyperpolarize neurons at that site. The magnetic flux generated by the current causes its own electric field, as explained by the Maxwell-Faraday equation,

.

This electric field causes a change in transmembrane currents resulting in depolarization or hyperpolarization of neurons, causing them to be more or less excitable, respectively.[22]

Deep TMS can reach up to 6 cm into the brain to stimulate deeper layers of the motor cortex, such as that which controls leg motion. The path of this current can be difficult to model because the brain is irregularly shaped with variable internal density and water content, leading to a nonuniform magnetic field strength and conduction throughout its tissues.[23]

Frequency and duration[edit]

The effects of TMS can be divided based on frequency, duration and intensity (amplitude) of stimulation:[24]

- Single or paired pulse TMS causes neurons in the neocortex under the site of stimulation to depolarize and discharge an action potential. If used in the primary motor cortex, it produces muscle activity referred to as a motor evoked potential (MEP) which can be recorded on electromyography. If used on the occipital cortex, 'phosphenes' (flashes of light) might be perceived by the subject. In most other areas of the cortex, there is no conscious effect, but behaviour may be altered (e.g., slower reaction time on a cognitive task), or changes in brain activity may be detected using diagnostic equipment.[25]
- Repetitive TMS produces longer-lasting effects which persist past the period of stimulation. rTMS can increase or decrease the excitability of the corticospinal tract depending on the intensity of stimulation, coil orientation, and frequency. Low frequency rTMS with a stimulus frequency less than 1 Hz is believed to inhibit cortical firing while a stimulus frequency greater than 1 Hz, or high frequency, is believed to provoke it.[26] Though its mechanism is not clear, it has been suggested as being due to a change in synaptic efficacy related to long-term potentiation (LTP) and long-term depression (LTD).[27]

Coil types[edit]

Most devices use a coil shaped like a figure-eight to deliver a shallow magnetic field that affects more superficial neurons in the brain.[28] Differences in magnetic coil design should be considered when comparing results, with important elements including the type of material, geometry and specific characteristics of the associated magnetic pulse.

The core material may be either a magnetically inert substrate ('air core'), or a solid, ferromagnetically active material ('solid core'). Solid cores result in more efficient transfer of electrical energy to a magnetic field and reduce energy loss to heat, and so can be operated with the higher volume of therapy protocols without interruption due to overheating. Varying the geometric shape of the coil

itself can cause variations in focality, shape, and depth of penetration. Differences in coil material and its power supply also affect magnetic pulse width and duration.[29]

A number of different types of coils exist, each of which produce different magnetic fields. The round coil is the original used in TMS. Later, the figure-eight (butterfly) coil was developed to provide a more focal pattern of activation in the brain, and the four-leaf coil for focal stimulation of peripheral nerves. The double-cone coil conforms more to the shape of the head.[30] The Hesed (H-core), circular crown and double cone coils allow more widespread activation and a deeper magnetic penetration. They are supposed to impact deeper areas in the motor cortex and cerebellum controlling the legs and pelvic floor, for example, though the increased depth comes at the cost of a less focused magnetic pulse.[11]

History[edit]

Luigi Galvani (1737-1798) undertook research on the effects of electricity on the body in the late-eighteenth century and laid the foundations for the field of electrophysiology.[31] In the 1830s Michael Faraday (1791-1867) discovered that an electrical current had a corresponding magnetic field, and that changing one could induce its counterpart.[32]

Work to directly stimulate the human brain with electricity started in the late 1800s, and by the 1930s the Italian physicians Cerletti and Bini had developed electroconvulsive therapy (ECT).[31] ECT became widely used to treat mental illness, and ultimately overused, as it began to be seen as a panacea. This led to a backlash in the 1970s.[31]

In 1980 Merton and Morton successfully used transcranial electrical stimulation (TES) to stimulate the motor cortex. However, this process was very uncomfortable, and subsequently Anthony T. Barker began to search for an alternative to TES.[33] He began exploring the use of magnetic fields to alter electrical signaling within the brain, and the first stable TMS devices were developed in 1985.[31][32] They were originally intended[*by whom?*] as diagnostic and research devices, with evaluation of their therapeutic potential being a later development.[31][32] The United States' FDA first approved TMS devices in October 2008.[31]

Research[edit]

TMS has shown potential with neurologic conditions such as Alzheimer's disease,[4] amyotrophic lateral sclerosis,[4][34] persistent vegetative states,[4] epilepsy,[4][35] stroke related disability,[4][11][14][15][36][37] tinnitus,[4][38] multiple sclerosis,[4] schizophrenia,[4][10] and traumatic brain injury.[39]

With Parkinson's disease, early results suggest that low frequency stimulation may have an effect on medication associated dyskinesia, and that high frequency stimulation improves motor function.[40] The most effective treatment protocols appear to involve high frequency stimulation of the motor cortex, particularly on the dominant side,[41] but with more variable results for treatment of the dorsolateral prefrontal cortex.[42] It is less effective than electroconvulsive therapy for motor symptoms, though both appear to have utility.[43][44][45] Cerebellar stimulation has also shown potential for the treatment of levodopa associated dyskinesia.[46]

In psychiatry, it has shown potential with anxiety disorders, including panic disorder[47] and obsessive-compulsive disorder (OCD).[4] The most promising areas to target for OCD appear to be the orbitofrontal cortex and the supplementary motor area.[48] Older protocols that targeted the prefrontal dorsal cortex were less successful.[49] It has also been studied with autism,[50] substance abuse,[4] addiction,[4][51] and posttraumatic stress disorder (PTSD).[4] For treatment-resistant major depressive disorder, high-frequency (HF) rTMS of the left dorsolateral prefrontal cortex (DLPFC) appears effective and low-frequency (LF) rTMS of the right DLPFC has probable efficacy.[4][5][7][8][9]

TMS can also be used to map functional connectivity between the cerebellum and other areas of the brain.[52]

Study blinding[edit]

Mimicking the physical discomfort of rTMS with placebo to discern its true effect is a challenging issue in research.[4][11][53][54] It is difficult to establish a convincing placebo for TMS during controlled trials in conscious individuals due to the neck pain, headache and twitching in the scalp or upper face associated with the intervention.[4][11] In addition, placebo manipulations can affect brain sugar metabolism and MEPs, which may confound results.[55] This problem is exacerbated when using subjective measures of improvement.[11] Placebo responses in trials of rTMS in major depression are negatively associated with refractoriness to treatment.[56]

A 2011 review found that most studies did not report unblinding. In the minority that did, participants in real and sham rTMS groups were not significantly different in their ability to correctly guess their therapy, though there was a trend for participants in the real group to more often guess correctly.[57]

Animal model limitations[edit]

TMS research in animal studies is limited due to its early FDA approval for treatment-resistant depression, limiting development of animal specific magnetic coils.[58]

Society and culture[edit]

Regulatory approvals[edit]

Neurosurgery planning[edit]

Nexstim obtained 510(k) FDA clearance for the assessment of the primary motor cortex for pre-procedural planning in December 2009[59] and for neurosurgical planning in June 2011.[60]

Depression[edit]

In 2008, the US Food and Drug Administration authorized the use of rTMS as a treatment for depression that has not improved with other measures.[61][62] A number of deep TMS have received FDA 510k clearance to market for use in adults with treatment resistant major depressive disorders.[63][64][65][66][67] The Royal Australian and New Zealand College of Psychiatrists has endorsed rTMS for treatment resistant major depressive disorder (MDD).[68]

Migraine[edit]

The use of single-pulse TMS was approved by the FDA for treatment of migraines in December 2013.[69] It is approved as a Class II medical device under the "*de novo* pathway".[70][71]

Other areas[edit]

In the European Economic Area, various versions of Deep TMS H-coils have CE marking for Alzheimer's disease,[72] autism,[72] bipolar disorder,[73] epilepsy,[74] chronic pain,[73] major depressive disorder,[73] Parkinson's disease,[41][75] posttraumatic stress disorder (PTSD),[73] schizophrenia (negative symptoms)[73] and to aid smoking cessation.[72] One review found tentative benefit for cognitive enhancement in healthy people.[76]

In August 2018, the US Food and Drug Administration authorized the use of TMS in the treatment of obsessive-compulsive disorder (OCD).[77]

Coverage[edit]

United States[edit]

Commercial health insurance[edit]

In 2013, several commercial health insurance plans in the United States, including Anthem, Health Net, and Blue Cross Blue Shield of Nebraska and of Rhode Island, covered TMS for the treatment of depression for the first time.[78][79][80][81] In contrast, UnitedHealthcare issued a medical policy for TMS in 2013 that stated there is insufficient evidence that the procedure is beneficial for health outcomes in patients with depression. UnitedHealthcare noted that methodological concerns raised about the scientific evidence studying TMS for

depression include small sample size, lack of a validated sham comparison in randomized controlled studies, and variable uses of outcome measures.[82] Other commercial insurance plans whose 2013 medical coverage policies stated that the role of TMS in the treatment of depression and other disorders had not been clearly established or remained investigational included Aetna, Cigna and Regence.[83][84][85]

Medicare[edit]

Policies for Medicare coverage vary among local jurisdictions within the Medicare system,[86] and Medicare coverage for TMS has varied among jurisdictions and with time. For example:

- In early 2012 in New England, Medicare covered TMS for the first time in the United States.[87][88][89][90] However, that jurisdiction later decided to end coverage after October, 2013.[91]
- In August 2012, the jurisdiction covering Arkansas, Louisiana, Mississippi, Colorado, Texas, Oklahoma, and New Mexico determined that there was insufficient evidence to cover the treatment,[92] but the same jurisdiction subsequently determined that Medicare would cover TMS for the treatment of depression after December 2013.[93]

United Kingdom[edit]

The United Kingdom's National Institute for Health and Care Excellence (NICE) issues guidance to the National Health Service (NHS) in England, Wales, Scotland and Northern Ireland. NICE guidance does not cover whether or not the NHS should fund a procedure. Local NHS bodies (primary care trusts and hospital trusts) make decisions about funding after considering the clinical effectiveness of the procedure and whether the procedure represents value for money for the NHS.[94]

NICE evaluated TMS for severe depression (IPG 242) in 2007, and subsequently considered TMS for reassessment in January 2011 but did not change its evaluation.[95] The Institute found that TMS is safe, but there is insufficient evidence for its efficacy.[95]

In January 2014, NICE reported the results of an evaluation of TMS for treating and preventing migraine (IPG 477). NICE found that short-term TMS is safe but there is insufficient evidence to evaluate safety for long-term and frequent uses. It found that evidence on the efficacy of TMS for the treatment of migraine is limited in quantity, that evidence for the prevention of migraine is limited in both quality and quantity.[96]

Subsequently, in 2015, NICE approved the use of TMS for the treatment of depression in the UK and IPG542 replaced IPG242.[97] NICE said "The evidence on repetitive transcranial magnetic stimulation for depression shows

no major safety concerns. The evidence on its efficacy in the short-term is adequate, although the clinical response is variable. Repetitive transcranial magnetic stimulation for depression may be used with normal arrangements for clinical governance and audit."

Costs[edit]

A single TMS session for depressive disorders averages US$350. A full course of treatment could cost between US$6,000 and US$12,000, depending on the number of treatments.[98][26]

Providers[edit]

Manufacturers of the devices include Brainsway, Deymed, MagVenture, Mag&More, Magstim, Nexstim, Neuronetics, Neurosoft.[*citation needed*] Current UK providers include a few NHS Trusts and a private operator, Smart TMS. In Cyprus FDA approved treatments for depression and OCD are provided by Cyprus rTMS [99].Also the Cyprus Technological University uses rTMS in research [100]

III. Trans-cranial Direct Current Stimulation

It is a non-invasive form of treatment that uses very weak (1 to 3 mA) direct electrical current applied to the scalp. The small device is very portable and usually operated by readily available DC batteries.

A. Side effects. There are no known serious adverse effects of tDCS. It is well tolerated, with reported common side effects in the literature listing mostly minimal tingling at the site of stimulation, with a few reported cases of skin irritation.

B. Mechanism of action. Direct current polarizes current, and tDCS is believed to act via the alteration of neuronal membrane polarization, but little is known about the actual mechanism of action of tDCS.

C. Clinical studies. Preliminary research suggests that tDCS may enhance certain brain functions independent of mood; however, tDCS technology and its use in psychiatry are in the early stages of exploration.

IV. Cranial Electrical Stimulation

A. Definition. CES, like tDCS, uses a weak (1 to 4 mA) current. It is traditionally applied via saline-soaked, felt-covered electrodes clipped onto the earlobes.

B. Mechanism of action. The exact mechanism of action has not been elicited, and there is no agreement among researchers on the predominant mode of action.

C. Side effects. It is believed that the CES stimulation is not harmful, primarily due to its low voltage power supply (9-V battery) and lack of any reported adverse event by the FDA. Local skin effects, as well as a general feeling of dizziness, have been reported.

D. Clinical studies. In a meta-analysis by the Harvard School of Public Health the overall pooled result showed CES to be better than sham treatment for anxiety at a statistically significant level.

V. Magnetic Seizure Therapy

A. Definition. MST is a novel form of a convulsive treatment, given using a modified TMS device that is under development in several research institutions. The aim is to produce a seizure whose focus and patterns of spread may be controlled. MST is a convulsive treatment, in many ways similar to ECT and requires approximately the same preparation and infrastructure as ECT. It is not FDA approved.

B. Mechanism of action. Induction of a seizure is hypothesized to be the underlying event responsible for the likely multiple specific mechanisms of action of MST treatment.

C. Side effects. Adverse effects are like those of ECT, are largely connected to the risks associated with anaesthesia and generalized seizure. Studies show MST results in less retrograde and anterograde amnesia than ECT.

D. Current status in treatment algorithms. It is still an investigational protocol and treatments outside of research are not FDA approved.

VI. Vagus Nerve Stimulation

A. Definition. VNS is the direct, intermittent electrical stimulation of the left cervical vagus nerve via an implanted pulse generator, usually in the left chest wall. The electrode is wrapped around the left vagus nerve in the neck and is connected to the generator subcutaneously.

B. Side effects and contraindications. VNS is generally well tolerated. The most common side effects are voice alteration, dyspnoea, and neck pain.

C. Current status in treatment algorithms. The FDA indicated VNS for the adjunctive long-term treatment of chronic or recurrent depression in patients 18 years or older experiencing a major depressive episode in the setting of unipolar or bipolar disease who have not had an adequate response to four or more adequate antidepressant treatments.

D. Patient selection. VNS is approved as an adjunctive long-term treatment for chronic or recurrent depressive episodes in adults with a major depressive episode who have not had a satisfactory response to four or more adequate antidepressant trials. The efficacy of VNS in other disorders is unknown.

E. Dosing. The optimal dosing for psychiatric applications of VNS is still largely an area of investigation. The published studies do not identify optimal dosing parameters like time on, time off, frequency, current, or pulse width.

VII. Deep Brain Stimulation

The procedure involves placement of small-diameter brain "leads" (e.g., approximately 1.3 mm) with multiple electrode contacts into subcortical nuclei or specific white matter tracts. The surgeon drills burr holes in skull bone under local anaesthesia and then places the leads, guided by multimodal imaging and precise stereotactic land marking. Later, the "pacemaker" (also known as an implantable neurostimulator or pulse generator) is implanted sub dermally (e.g., in the upper chest wall) and connects it, via extension wires tunnelled under the skin, to the brain leads.

A. Indications. It is used to treat people with advanced Parkinson's disease, dystonia, and essential tremor whose symptoms are no longer controlled by medication.

B. Outcome with deep brain stimulation

1. Obsessive-compulsive disorder. DBS has been shown to have clinically significant symptoms reduction in patients with intractable OCD. DBS is placed at the ventral anterior limb of the internal capsule and adjacent ventral striatum (VC/VS).

2. Major depression. Functional neuroimaging research implicates the subgenual cingulate cortex as a node in circuits involved in the normal experience of sadness, symptoms of depressive illness, and responses to depression treatments. The treatment is in early stages and being studied but

chronic DBS for up to 6 months showed sustained remission of depression in a small number of patients. The advent of DBS in psychiatry has created tremendous interest and considerable research activity. DBS may therefore be accepted by patients who would not choose to undergo lesion procedures (although the reverse is also true). With all of its advantages, DBS requires that patients be treated by highly specialized teams willing and able to provide long-term care.

Assessment Procedure

Multiple choice questions based assessment after successful completion of theory and practical sessions

SRI LAKSHMI NARAYANA INSTITTUTE OF MEDICAL SCIENCE – VALUE ADDED COURSE –MEDICAL EDUCATION PROJECT

BRAIN STIMULATION TECHNIQUES – A DETAILED REVIEW 2020

SL.NO	YEAR	ROLL.NO	NAME OF CRRI	SIGNATURE
1.	2020	U15MB307	KASANKANTH.V	
2.	2020	U15MB308	KADHIRAVAN.G	
3.	2020	U15MB309	KAVIN SHANMUGAVEL.R	
4.	2020	U15MB311	KISHORE.K	
5.	2020	U15MB313	LAKSHMI.M	
6.	2020	U15MB315	LINDA EVANS.M	
7.	2020	U15MB316	MADHAVA SRIRAM.N	
8.	2020	U15MB317	MAHALAKSHMI.M.N	
9.	2020	U15MB327	NAGALAKSHMI.P	
10.	2020	U15MB320	MALAINESAN.E	

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SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

Candidate Name		Assessor Name	
Date of Assessment		Assessor Position	

Brain stimulation techniques – A detailed review

MULTIPLE CHOICE QUESTIONS

Course Code: PSYC12

I. ANSWER ALL THE QUESTIONS

1. The first use of *convulsive therapy* for the treatment of a psychiatric disorder in modern times is attributed to
A. Ladislaus von Meduna
B. A. E. Bennett
C. Egas Moniz
D. Kurt Schneider

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A. Harold Sackeim
B. Luigi Bini and Ugo Cerletti
C. D. Goldman
D. G. Holmberg and S. Thesieff

3. The sequece of administration of medications in anesthesia for ECT is:
A. Atropine---thiopentone/methohexitol---succinylcholine
B. Succinylcholine---atropine---thiopentone/methohexitol
C. Atracurium---succinylcholine---atropine
D. Atropine--- succinylecholine---thiopentone/methohexitol

4. As per the current evidence, which statement is NOT correct?
A. Bilateral ECT is superior in efficacy to unilateral ECT.
B. Unilateral ECT is more likely to cause cognitive deficits.
C. Brief-pulse ECT delivery is associated with decreased cognitive deficits.
D. Unilateral ECT is administered to the non-dominant hemisphere

5. Which of the following drugs is associated with lower seizure thresholds when administering ECT?
A. Lithium
B. Anticonvulsants
C. Benzodiazepines
D. Barbiturates

6. What is the minimum seizure duration required for effectiveness of ECT?
A. 1 to 3 seconds
B. 5 to 10 seconds
C. 30 to 90 seconds
D. 180 to 200 seconds

7. What is the best accepted placement of electrodes in unilateral ECT?
A. Bifrontotemporal
B. Paritotemporal

C. Occipital
D. D'Elia position

8. What is considered as the gold standard for confirmation of seizure in ECT?
A. Cuff method
B. Electroencephalography (EEG)
C. Electromyogram (EMG)
D. Galvanic Skin Response (GSR)

9. What is the average mortality rate with ECT (modified)?
A. 3-4 per 100,000
B. 10-25 per 100,000
C. 10-20 per 10,000
D. 50-60 per 1000,00

10. Factors predisposing to postictal confusional state include, all EXCEPT:
A. Sine wave ECT
B. High-dose ECT
C. Existing CNS disease
D. Multiple ECT
E. A younger age group

11. Which is the best unit for quantification of ECT stimuli?
A. Millicoulombs (mC)
B. Joules
C. Watts
D. Volt

12. Which of the following is NOT a recommended preparation for ECT procedure?
A. Informed consent in writing
B. Pre-ECT investigations
C. Morning bath, cleaning the oil from the head, overnight fast
D. Premedication with an anticholinergic agent
E. Administration of an anticonvulsant 30 minutes before ECT

13. The most common indication of ECT is:
A. Schizophrenia
B. Generalized Anxiety Disorder
C. Manic episodes
D. Major depression

14. rTMS is found to have antidepressant properties when applied to
A. Temporal– parietal regions of the cortex
B. Dorsolateral prefrontal cortex (DLPFC)
C. Occipital cortex
D. Parieto-occipital cortex

15. The most persistent adverse effect of ECT is
A. Retrograde amnesia
B. Fractures
C. Seizures
D. Hypertension

SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

Candidate Name	KISHORE.K	Assessor Name	DR. AGILA
Date of Assessment	18.5.2020	Assessor Position	ASSISTANT PROFESSOR

UNIVERSITY REG NO:- 015MB311.

10/15

Brain stimulation techniques – A detailed review

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SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

Candidate Name	KAVIN SHANMUGAVEL.R	Assessor Name	DR. AGILA
Date of Assessment	18.5.2020	Assessor Position	ASSISTANT PROFESSOR

UNIVERSITY REG. NO: - 115MB309

11/15

Brain stimulation techniques – A detailed review

MULTIPLE CHOICE QUESTIONS

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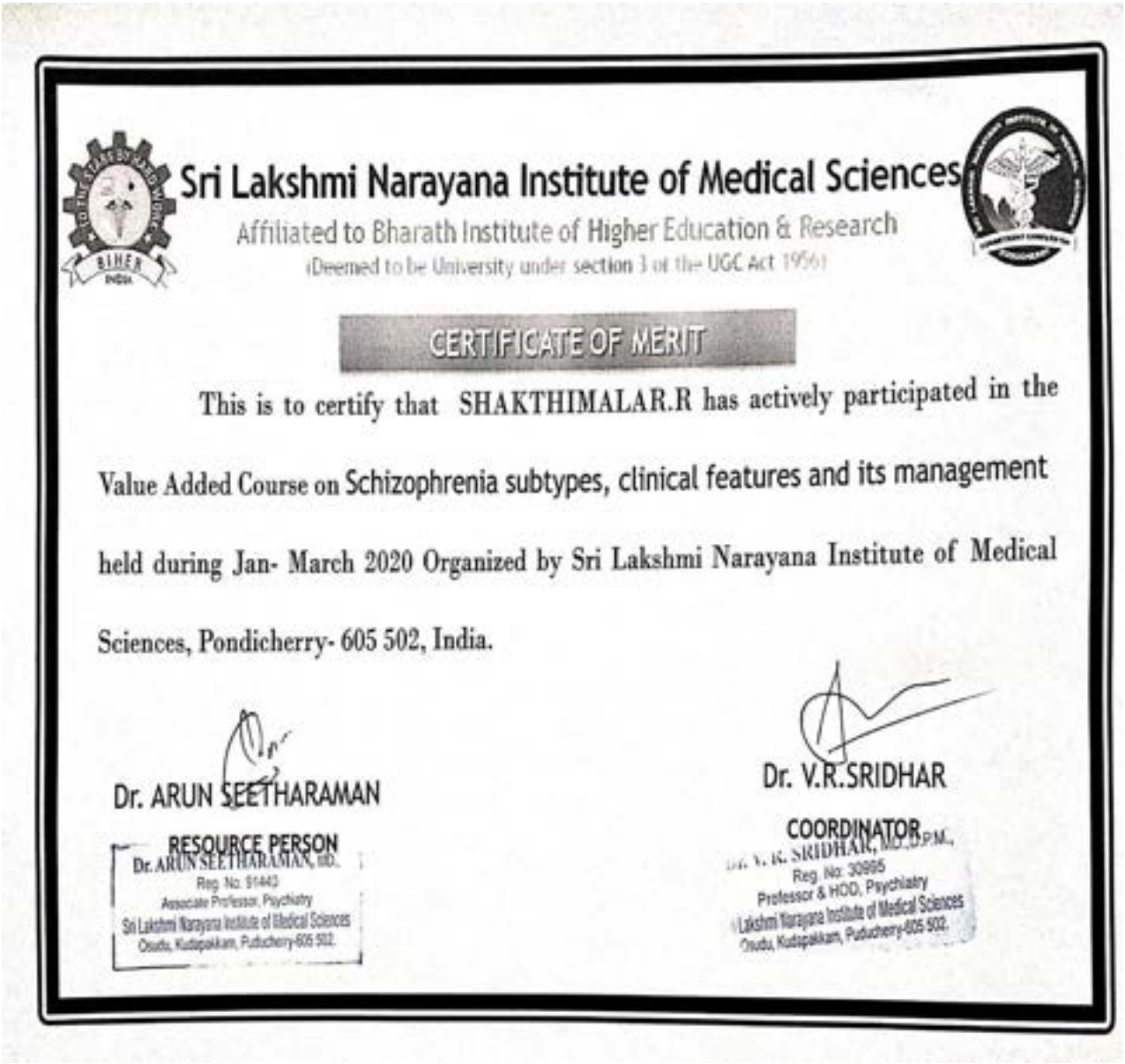

Sri Lakshmi Narayana Institute of Medical Sciences

Affiliated to Bharath Institute of Higher Education & Research

(Deemed to be University under section 3 of the UGC Act 1956)

CERTIFICATE OF MERIT

This is to certify that SHAKTHIMALAR.R has actively participated in the Value Added Course on Schizophrenia subtypes, clinical features and its management held during Jan- March 2020 Organized by Sri Lakshmi Narayana Institute of Medical Sciences, Pondicherry- 605 502, India.

Dr. ARUN SEETHARAMAN

RESOURCE PERSON
Dr. ARUN SEETHARAMAN, MD.,
Reg. No. 91442
Associate Professor, Psychiatry
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Kudapakkam, Puducherry-605 502.

Dr. V.R.SRIDHAR

COORDINATOR
Dr. V. R. SRIDHAR, M.D., D.P.M.,
Reg. No. 30995
Professor & HOD, Psychiatry
Lakshmi Narayana Institute of Medical Sciences
Osudu, Kudapakkam, Puducherry-605 502.

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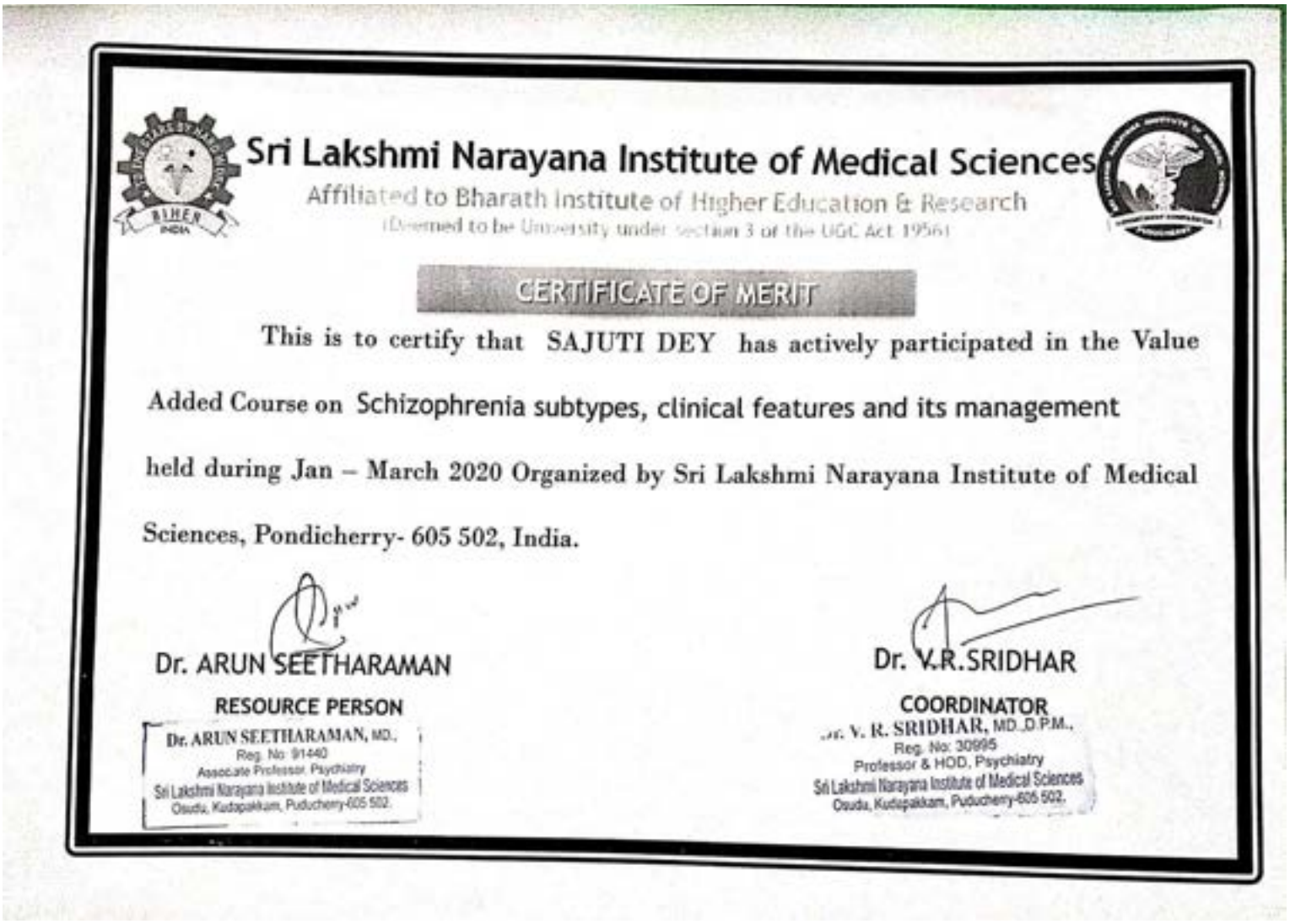

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Dr. ARUN SEETHARAMAN
RESOURCE PERSON
Dr. ARUN SEETHARAMAN, MD.,
Reg. No: 91440
Associate Professor, Psychiatry
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Kudapakkam, Puducherry-605 502.

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COORDINATOR
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SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

Student Feedback Form

Course Name: **BRAIN STIMULATION TECHNIQUES**
Subject Code: **PSYC12**

Name of Student: _____ Roll No.: _____

We are constantly looking to improve our classes and deliver the best training to you. Your evaluations, comments and suggestions will help us to improve our performance

SI. NO	Particulars	1	2	3	4	5
1	Objective of the course is clear					
2	Course contents met with your expectations					
3	Lecturer sequence was well planned					
4	Lectures were clear and easy to understand					
5	Teaching aids were effective					
6	Instructors encourage interaction and were helpful					
7	The level of the course					
8	Overall rating of the course	1	2	3	4	5

**** Rating: 5 – Outstanding; 4 - Excellent; 3 – Good; 2– Satisfactory; 1 - Not-Satisfactory***

Suggestions if any:

SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

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Course Name: **BRAIN STIMULATION THERAPIES**
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Name of Student: KASANKANTH · V Roll No.: U15MB307

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Sl. NO	Particulars	1	2	3	4	5
1	Objective of the course is clear			✓		
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Signature

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Suggestions if any:

Signature

Date:

SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

Date: 30-06-2020

From

Dr.V.R.Sridhar
Professor and Head,
Department of Psychiatry,
Sri Lakshmi Narayana Institute of Medical Sciences
Bharath Institute of Higher Education and Research,
Chennai.

Through Proper Channel

To

The Dean,
Sri Lakshmi Narayana Institute of Medical Sciences
Bharath Institute of Higher Education and Research,
Chennai.

SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

Sub: Completion of value-added course: Brain stimulation techniques – A detailed review

Dear Sir,

With reference to the subject mentioned above, the department has conducted the value-added course titled: **Brain stimulation techniques – A detailed review**. We solicit your kind action to send certificates for the participants that is attached with this letter. Also, I am attaching the photographs captured during the conduct of the course.

Kind Regards,

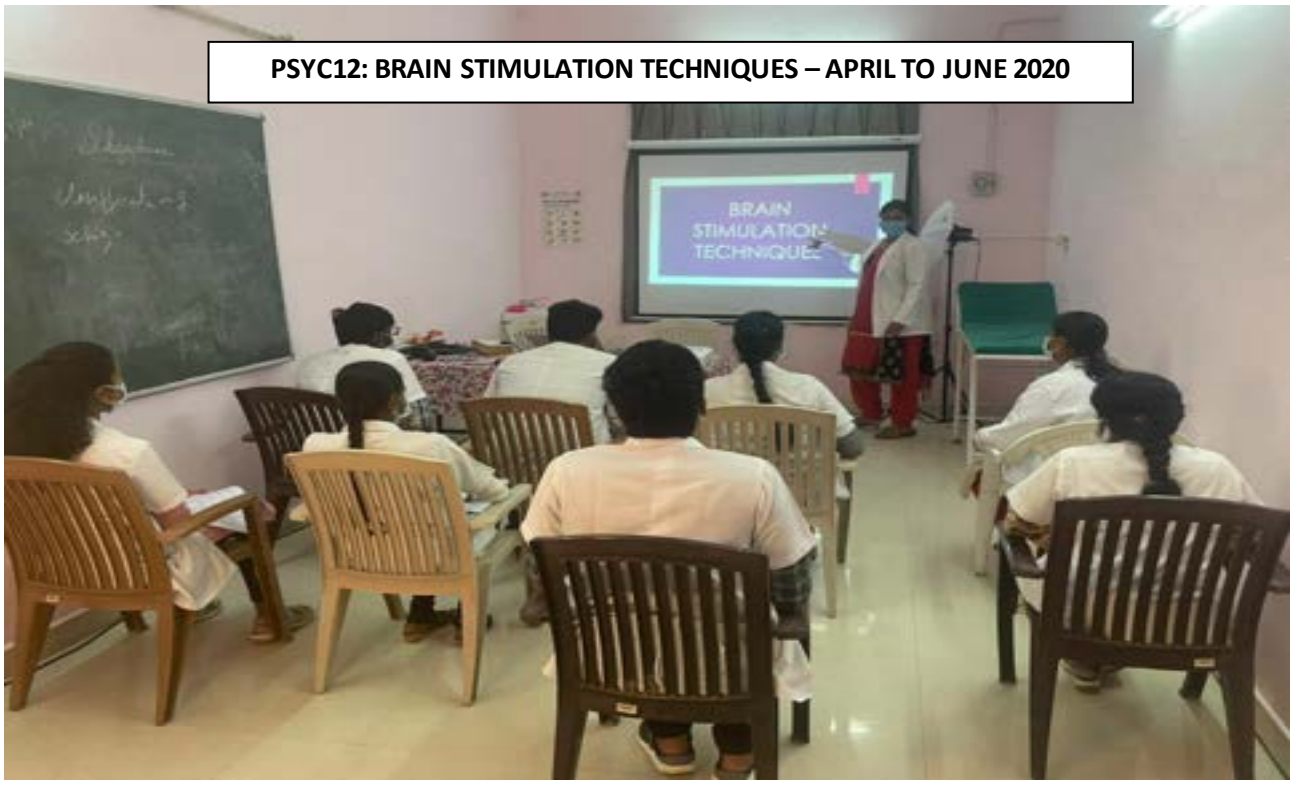
Dr. V. R. SRIDHAR, MD.,D.P.M.,
Reg. No. 30995
Professor & HOD, Psychiatry
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Kudapakkam, Puducherry-605 502.

Dr. Sridhar

Encl: Certificates

Photographs

SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH



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