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"Electroconvulsive therapy in patients with cardiac implantable electronic devices: a case report and systematic review of published cases"

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Abstract

Objective—To report the case of a 54-year-old man with recurrent depressive disorder with multiple medical co-morbidities having a dual-chamber pacemaker, treated successfully with 11 sessions of electroconvulsive therapy, and to conduct a systematic review of published cases documenting the use of electroconvulsive therapy in patients with cardiac implantable electronic devices for treating major psychiatric disorders.

Methods—We searched electronic databases (MEDLINE, PubMed, Google Scholar, Embase, Cochrane Library, PsycINFO, and Crossref) and included studies reporting on the use of electroconvulsive therapy in patients with cardiac implantable electronic devices.

Results—35 publications across 53 years (1967-2021) reported on 76 patients (including current report) who received a pooled total of 979 modified ECT sessions. The most common adverse events were premature ventricular contraction and hypertension. There have been no reports of serious adverse effects that necessitated the cessation of ECT.

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Contribution by the authors:

Dr Abhiram PN performed literature search, data analysis and drafted the original work.
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Dr Suma T. Udupa, Dr. Ravindra N. Munoli and Dr. Samir Kumar Praharaj have critically revised the work and have managed the case.

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Conclusion—ECT is a safe and efficacious treatment for major psychiatric disorders, and the presence of cardiac implantable electronic devices should not delay or deter the use of ECT in these patients.

Keywords

Cardiovascular diseases; CIED; Depression; ECT; Pacemakers

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Introduction

Electroconvulsive therapy (ECT) is a non-invasive brain stimulation technique that has proven efficacy in depression, schizophrenia, and other major psychiatric disorders.[12] It is a safe and well-tolerated procedure. Adverse effects with ECT are commonly mild and can be managed symptomatically.[3] Presence of medical co-morbidity often makes the management of psychiatric disorders more challenging by negatively impacting the treatment responsiveness, tolerability, morbidity, and mortality. Therefore, ECT holds a special significance in this population. With the reasonable modification of the procedure, ECT can be safely adapted for use in major psychiatric disorders with co-morbid medical conditions, including cardiac disorders.[4] Advancement in the management of arrhythmias and heart failure has resulted in the increased use of cardiac implantable electronic devices (CIEDs) such as pacemakers and Implantable Cardioverter Defibrillators (ICD).[5] Extant literature reports that therapeutic use of ECT in patients with CIED poses a unique challenge at various levels, including electrical interference caused by ECT stimulus, hemodynamic alterations during the ECT, and myopotentials that can be sensed by the device or response of CIED to benign arrhythmias that might arise after ECT.[6–9] With the advances in the treatment of cardiac conditions, there has been a steady increase in the pacemaker implantation rates, and so too does the frequency with which these patients present for ECT. This demands for a comprehensive understanding of safe and efficacious utilization of ECT in patients with CIED

. Although there have been case reports and case series, there are no controlled studies evaluating the management of CIEDs during ECTs. We report a case with recurrent depressive disorder with an implanted pacemaker who was successfully treated with a ECT and conducted a systematic review of the published cases.

Case report

Mr.A, a 54-year-old married gentleman, presented with a history of pervasive sadness, anhedonia, anergia, insomnia, reduced appetite, hopelessness, and suicidal ideations with a suicide attempt with high intent to die in the last six months. He had the first episode of depression at the age of 49 following the death of his spouse. Since then, he has had four episodes of depression with a history of multiple suicide attempts of high intentionality and lethality. He has a family history of schizophrenia in first-degree relatives and suicide in a third-degree relative.. His medical history included three cerebrovascular accidents (at the ages of 16, 31 and, 47 years), hypothyroidism, bronchial asthma, benign prostatic hypertrophy, intervertebral disc prolapse, dyslipidaemia, and ischemic heart disease (IHD)

controlled on regular medication. At the age of 48 years, he was diagnosed with ischemic heart disease and underwent a percutaneous coronary intervention with stent to the right coronary artery. Three years later he developed recurrent syncope and was found to have sinus pauses and sick sinus syndrome and thus a dual-chamber demand pacemaker (Endurity Core PP2140, Abbott Plymouth, MN) was placed in April 2019. The atrial and ventricular sensitivities were 0.5 MV and 2.0 MV, respectively, with a base rate of 60/min. The post-operative period was uneventful.

He was diagnosed with recurrent depressive disorder, current episode severe depression without psychotic symptoms as per the International Classification of Diseases 10th edition (ICD-10).[10] He had a history of failed trials of several antidepressants, including sertraline, paroxetine, and augmentation with mirtazapine, bupropion, and lithium in adequate doses and duration. We treated him with desvenlafaxine 50 mg, which was gradually increased up to 200 mg per day. After two months, due to ongoing suicidal ideas and poor response to treatment, he was admitted and considered for ECT. We obtained an opinion from a cardiac electrophysiologist regarding reprogramming the pacemaker. The following precautions were advised and were followed during the ECT: 1) Interrogation of the device pre-and post- ECT. The device might have to be changed to asynchronous pacing mode if the patient is pacemaker dependent; 2) Adequate muscular relaxation and isolation of right arm to minimize the possibility of dislocating pacemaker and to avoid strong myopotentials; 3) To avoid any electronic devices or electrical stimulus within 25 cm of the proximity of pulse generator to prevent electromagnetic interference. Bi-temporal electrode placement was deemed safe for this, and; 4) Electrical isolation of patient and pacemaker with the strict grounding of the equipment.

Before ECT, interrogation of pacemaker was done. The patient was deemed not to be pacemaker-dependant, with just 2% pacing requirement, 1:1 atrioventricular conduction, good battery status, and thus no reprogramming was necessary. All the precautions mentioned above were followed along with the cardiology team with a defibrillator, and percutaneous pacing paddles on standby for necessary interventions. Anaesthesia was induced with intravenous thiopentone sodium (4 mg/kg) and succinylcholine (0.5-1 mg/kg) with premedication with glycopyrrolate (0.2 mg). The seizure was induced using a Nivique-VR device (Nivique Meditech Pvt Ltd, Bangalore) thrice weekly with bi-temporal electrode placement. The seizure threshold was calculated using the titration method. The blood oxygen level was kept at greater than 95%. When the patient's muscles were relaxed, the charge of 120-360 millicoulombs (800 milliAmp of current; Pulse width of 1 millisecond and Pulse frequency of 125 pulses per second) was delivered and the patient had adequate motor seizures (mean seizure duration of 36.1 seconds with a range of 26 to 57 seconds).. The patient was monitored in the post-operative ICU for 30 minutes after each session and had a mean recovery time of 9 minutes (range of 8 to 10 minutes). Notable post-ictal physiological effects were transient sinus tachycardia (6/11 sessions) lasting for an average duration of 8.8 minutes and hypertension (6/11 sessions; mean systolic blood pressure=163.3mm Hg and mean diastolic blood pressure=103.3 mmHg, 5 minutes after seizure termination) which was self-limiting. He also had post ECT desaturation (defined as fall in O_2 saturation less than 90%) in three sessions to 80% for 2-3 seconds managed by the anaesthesia team with supplemental oxygen. He had sweating and hypoglycaemia

(Random blood sugar =69 mg/dl) in one session, which was managed by DNS infusion. His baseline Hamilton Depressive Rating Scale (HDRS-21) score was 27. After the second ECT treatment, improvements in depressive symptoms and suicidal ideas were observed. Subjectively, the patient was sleeping better and began to feel more optimistic. ECT was continued. He showed a response of more than 50 % improvement in HDRS after three sessions. After eight sessions of ECT, he reported mild memory disturbances despite the improvement in mood. Cognitive functions were evaluated serially with the B4ReCoDe ECT test(specific battery for assessing ECT-related cognitive adverse effects), and it showed no cognitive deficits.[11] Baseline was 113 and post ECT, it was 109. He attained remission with good functioning after 11 sessions of ECT. The patient was discharged and followed up for two months, during which he was maintaining well but reported mild subjective memory disturbances as a persisting adverse effect._No arrhythmias or pacemaker malfunction was detected during and after the ECT sessions.

Methods

Search Strategy and study selection

The electronic search for relevant peer-reviewed English language articles was conducted using electronic databases (MEDLINE, PubMed, Google Scholar, Embase, Cochrane Library, PsycINFO, and Crossref) by two authors (APN and SV) independently. Doubts and conflicts were resolved by another author (SKP). The combinations of keywords ‘Electroconvulsive therapy’ or ‘ECT’ with keywords ‘CIEDs’, ‘cardiac rhythm management devices’, ‘pacemakers’, ‘implantable cardiac defibrillator’, ‘cardioverter’ were used. All research reports, including case reports, case series, and retrospective chart reviews, were included. The full text of these articles was retrieved electronically. Additionally, a manual search of cross-references was carried out to identify potentially relevant articles.

Extraction of data

The data extracted from the studies included details such as author names, year, details of the patient, medical diagnoses, psychiatric diagnosis, psychotropic medications, indication for ECT, indication for CIED, type of CIED, anaesthetic agents used during ECT, procedures during ECT, modifications of the procedure, adverse events and recommendations. We included studies from the year 1968 to May 2021.

Data synthesis and analysis

Quality appraisal of the reported cases was done to look for adequacy of description of the case, medical comorbidities, CIED, modification of procedures during ECT, and adverse events. Pooled data were summarized using descriptive statistics, with means and standard deviations for continuous variables and frequencies and percentages for dichotomous variables.

Results

Publication characteristics

We identified a total of 31 case reports, 4 case series publications across a span of 53 years (1967 to 2020), reporting on 75 patients. We were unable to retrieve one case report.[12] We did not find any published report on ECT in patients with cardiac resynchronization therapy device. We did pooled analysis on a total of 76 patients including our current report.

Quality appraisal

The case reports were graded on the adequacy of description of the case, medical comorbidities, CIED, modification of procedures during ECT, and adverse events. Depending on the description of the above five parameters, case reports were appraised. The overall quality of reported cases ranged from good (all five parameters are described) to moderate (few parameters are missing). Of the patients, 58 (78%)had an adequate description of psychiatric morbidity, co-morbid cardiac condition, details of CIED, and the modification carried out during ECT sessions; whereas 12 (16%) did not have the description of psychiatric illness and the indication for ECT, 8 (11%) did not have the description and pacing mode of CIED and the details of modifications done on the mode of CIED during ECT session, and 14 (18.6%) did not have the details of psychotropic and the medication for cardiac conditions.

Results of pooled analysis

Seventy-six patients (Female = 37, Male=39), with a mean age of 71.2 (SD 12.5, range 31-95) years have received a pooled total of more than 979 modified ECT sessions (806 sessions in patients with pacemakers, 148 sessions in patients with ICDs, 25 sessions in a patient with both pacemaker and ICD) (See supplemental table 1). The majority of patients (76%, N=57) were above the age of 65 years. Sixty-one patients had a pacemaker, thirteen patients had ICD, one patient had both a pacemaker and an ICD device, and one patient had an ICD and cardiac contractility modulator (CCM). The most common indication for pacemaker implantation was atrioventricular block (43.5%, N=27), followed by sinus node dysfunction (27.4%, N=17), whereas ventricular tachycardia was the most common (64.2%, N=9) indication for ICD implantation among patients. Among those with a pacemaker, dual-chamber and dual-sensing was the most common pacing mode (51.6%, N=32), followed by ventricular pacing and ventricular sensing mode (27.4%, N=17). The comorbid medical conditions included hypertension (26.3), had ischemic heart disease (14.4%), and congestive cardiac failure (9.2%). Psychiatric diagnoses were unipolar depression in 57 (75%), bipolar depression in 5 (6.5%), and one patient had NMDA receptor encephalitis and severe catatonia for which ECT was given. About 50% of patients who had unipolar depression, either single episode or recurrent episodes, had treatment resistance. Anticholinergic medication before ECT was given in 37 patients (48.7%), and in the rest, it was avoided. Thiopental sodium was the most commonly used anaesthetic agent during ECT (59.2 %, N=45). Among patients who had a pacemaker, in 45 (77.4%), the pacing mode was unchanged during the ECT sessions. In most patients who had ICD (71.4%, N=10), the device was deactivated before ECT sessions and later reprogrammed. In the pooled total of 831 ECT sessions given to patients with a pacemaker, 668 sessions have been carried out

without changing the pacing mode (80.3%), and in 66 ECT sessions, the pacing has been changed to asynchronous mode. Whereas in the pooled total of 148 ECT sessions given to the patients with ICD, in 71 sessions, the device was deactivated (48%), and in 29 sessions, the device was left active during ECT. There was one instance of inappropriate ICD shock delivery in the recent case report[13].

In three-fourth of patients (75%), ECT sessions were uneventful. In 25% of patients, there were adverse effects immediately after ECT administration (Table 1). The premature ventricular contraction was the most common adverse effect noted, which recovered spontaneously. Hypertension was the commonest (7.1%) adverse effect that needed medical intervention in 70 sessions out of 979 pooled ECT sessions which was easily managed without any serious consequences. There have been no reports of serious adverse effects that necessitated the cessation of ECT administration.

Discussion

ECT and cardiovascular risk

It has been about 65 years since the advent of permanent cardiac pacing for therapeutic use in its modern sense. There has been a paradigm shift in the design language with increasing reduction of hardware, thereby leading to the diminution of complications.[14,15] With the advance in anesthesiological procedures and continuous monitoring of heart rate, blood pressure, oxygen saturation, and electrocardiogram, the cardiovascular morbidity of ECT has also reduced over time.[9,16] Safety and efficacy of ECT have been advocated in the elderly population with co-morbid medical conditions, and our pooled analysis emulates the same.[17] Though the overall risk of cardiovascular complications during ECT is low, it still forms the commonest source of morbidity due to ECT.[9,16] The electrical stimulus and subsequent seizure induce a brief yet intense hemodynamic response, leading to complications in those who already have compromising cardiovascular functioning due to pre-existing conditions.[18,19] The hemodynamic response thereby increases the myocardial oxygen demand by two- to three-fold; hence, cardiovascular complications are more common in those who have coronary artery disease.[20,21] Immediate centrally mediated parasympathetic drive after ECT stimulus can result in bradycardia and transient asystole, whereas, the ictal sympathetic surge can result in benign and self-limiting cardiac arrhythmias.[21] Hypertension and tachycardia are common and generally time-limited adverse effects of ECT, which can be controlled by prophylactic beta-blockers to extenuate the sympathetic surge even in those with pre-existing cardiac conditions; and the same was observed in our pooled analysis.[22] With pre-treatment stabilization of cardiac rhythm, ECT can be safely given in patients with arrhythmias.[4] Most of the patients in our pooled analysis had co-morbid high-risk cardiac conditions; still, ECT has been largely uneventful and been efficacious.

ECT in patients with CIED

Pacemakers can be of either single-chamber, dual-chamber or biventricular types. ICD can be of single-chamber, dual-chamber or, one coupled with cardiac resynchronization device. The procedure of ECT in patients with CIED bears challenges at various levels.

a) The ECT stimulus, in theory, can result in electromagnetic interference (EMI), which can intercede the function of CIED, leading to oversensing and noise reversion depending on the programming of the device.[23–25] The clinical consequence of this will be an inhibition of pacing resulting in bradycardia/asystole or an increase in the ventricular pacing/atrial arrhythmia depending on the pacemaker's mode. This can be inconsequential or problematic depending on the pacemaker dependency of the patient. The ICD, on the other hand, can detect a certain frequency of EMI as ventricular arrhythmia and consequently deliver an inappropriate shock.[23–26] However, practically, the risk of EMI with a brief duration of ECT stimulus is less.[24,26]

b) A myopotential is an electric signal arising in a skeletal muscle that is falsely picked up as a depolarization signal by the CIED. It is caused by the over sensing of muscle potentials by the unipolar atrial channels of a dual chamber-pacemaker as the atrial sensitivity setting is usually more sensitive than ventricular sensitivity. The fasciculation produced by depolarizing muscle relaxants can cause complete inhibition of pacemakers due to over sensing.This can lead to long asystolic pauses and ventricular tachycardia in pacemaker-dependent patients.[27,28] It is recommended that the risk of oversensing and far-field sensing can be eliminated by reducing the sensitivity of pacemaker leads of unipolar configuration or changing over to bipolar configuration if necessary.[29,30] Nonetheless, newer pacemaker generators with special shielding and bipolar sensing have largely replaced the older unipolar systems, and hence the concern of myopotentials being sensed by the pacemaker and extracorporeal EMI is obsolete now.[29,31,32] Changing over to asynchronous pacing modes eliminates the risk of over sensing, but the prospect of competitive atrial or ventricular rhythm can prevail.[29] Most of the available guidelines on the perioperative management of CIED recommend converting the pacemaker to asynchronous mode and turning off the anti-tachycardia function of the ICD during ECT.[26,30,33] Despite the recommended guidelines, as reflected in our pooled data, in the overwhelming majority of ECT sessions, the pacing mode was unchanged, and there was no reporting of any adverse event due to this. Suzuki et al. have discussed the possibility of the increment in sinus node discharge due to sympathetic surge during ECT resulting in ventricular fibrillation due to the "R-on-T" phenomenon in a pacemaker with an asynchronous pacing mode.[34,35] However, this has been attributed to sensing failure, and there have been no reports of asynchronous mode triggering ventricular tachyarrythmias.[36]

c) Most modern pacemakers also have a rate-adaptive pacing system that responds to the increased physical or metabolic demand.[37] Rarely, this algorithm can inappropriately sense excessive movements due to inadequate muscle relaxation during the ictal phase and mechanical movements of the chest wall due to manual ventilation during anaesthesia induction.[7] However, no adverse events have been noted even when rate-adaptation mode was not altered during ECT as per our pooled analysis.

With the previously published literature and available recommendations, it can be concluded that in a pacemaker-dependent patient, it is best to convert the pacing to asynchronous mode during ECT and to ensure adequate control of tachycardia after ECT; whereas, in patients with a stable intrinsic rhythm, it is best to follow various methods to reduce oversensing without converting the pacemaker to asynchronous mode.[26,29,30,36,38]

d) In addition to the above-discussed susceptibilities, ICDs have an additional challenge of inappropriate shock delivery due to inappropriate sensing of EMI as ventricular dysrhythmia (ADD) or sinus tachycardia exceeding the tachycardia detection rate, and this can result in psychological distress.[29,33,39] The available guidelines recommend turning off the anti-tachycardia function of ICD during ECT, and it is reflected in our pooled data, wherein the majority of sessions, the devices were inactivated before ECT.[26,29,30,38] Among the small minority of sessions, ICD was not deactivated, and there was one instance of inappropriate shock delivery which has been attributed to the failure of the anti-tachycardia therapy inhibition by the magnet.[13]With the available guidelines and previously published literature, it can be concluded that it is prudent to turn off the anti-tachycardia pacing function of the ICD before ECT.[26,29,30,33,38]

Magnet application is often used in the perioperative period to change the functioning modes of CIED. However, it has to be emphasized that the response of different CIED for magnet application is variable. Generally, for pacemakers, magnet application results in asynchronous pacing with a fixed pacing rate and fixed atrioventricular delay. However, there are exceptions where the magnet behaviour will be device-specific depending on the manufacturing design, and magnet application might not result in asynchronous pacing but only selective suspension of atrial anti-tachycardia pacing.[26,33,38] The response to magnet application in ICD is again device-specific. Still in general, it will disable the tachycardia detection function of the ICD, but it will not render the pacing function of ICD into asynchronous mode.[26] Most guidelines recommend having a pacemaker/ICD magnet available during the procedure where it can come in handy should significant pacemaker inhibition occur during ECT.[26,30,33,38] There is no data on ECT in patients implanted with the modern subcutaneous ICD. The sensing vectors in these devices can be larger, spanning from the proximal/distal electrode ring of the lead to the pulse-generator, and therefore are prone to myopotential over-sensing from the chest wall muscles. However, the commonest source of inappropriate shocks in these devices is still T-wave over-sensing.[40] This is particularly important during ECT, and it may be prudent, intuitively, to switch off the defibrillatory function during ECT in such patients. Finally, Cardiac Resynchronisation Therapy (CRT) devices exist with pacing function alone (CRT-P) or with defibrillation function combined (CRT-D). Hence, the precautions during ECT in patients with CRT should be similar to those discussed previously.

The intensity of cardiovascular changes during ECT may also vary with pulse width and electrode placement changes. Bifrontal electrode placement, compared to bitemporal, has lesser effects on heart rate due to lesser current density in more posterior midline brain structures. The risk of bradycardia and asystole has also been lesser with bifrontal electrode

placement.[41,42] Therefore it is prudent to consider bifrontal electrode placement in patients with CIED.

Strengths and limitations

The lack of randomized controlled data for the best recommendation in this area is not unforeseen as the number of patients with CIED presenting for ECT is not overwhelming.Hence the small sample might not reflect the true effects. The incidence of 'far-field' sensing of various extracorporeal and intra-corporeal artifacts by the CIED cannot be possibly controlled across the group if a randomized controlled study is designed. Multiple factors related to co-morbid cardiac conditions, variations in the CIED design, sensitivity of sensing leads, cardiac autonomic response during ECT preclude homogeneity and equivalence across the group. Hence good quality case reports and case series are the mainstays of evidence (e.g., Dolenc et al. [6], Macpherson et al. [7]). Kokras et al. [8] in 2011 have published a review and pooled analysis of 63 patients. Our systematic review has all published case reports (N=76 patients) that confirm the largely proven evidence of the safety of ECT in patients with comorbid cardiac conditions. However, the publication bias arising from adverse outcomes not being reported should be considered.

General considerations for the clinical practice

The clinical aspects to be considered while giving ECT in a patient with CIED, based on the reviewed literature, have been summarized in Table 2.

Conclusion

ECT is a time-tested, safe, and efficacious treatment for major psychiatric disorders, and the presence of CIED need not be an absolute contraindication for ECT. Adhering to safety recommendations will ensure the effective harness of the therapeutic potential of ECT. It is recommended that ECT should be given after converting the pacemaker to the asynchronous mode in a pacemaker-dependent patient with prompt detection and control of tachyarrhythmia in post-ictal phase, the synchronous mode in patients with good intrinsic rhythm, and to deactivate ICD and reprogram after ECT under the care of multi-disciplinary team involving psychiatrist, anaesthesiologist, cardiologist and cardiac electrophysiologist. Of note, there are reports of ECT being administered safely in patients with transvenous ICD without prior reprogramming, but this decision is perhaps best individualised, weighing the potential risk of over-sensing induced inappropriate shocks.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

List of reported adverse effects.

Author	Age	Sex	Type of CIED	Action	Adverse event	No of sessions	Possible cause	Management
Ballenger et al.[17]	50	M	PM (VVI)	Unchanged	Atrial flutter	1	Anticholinergic premedication	Cardioversion and procainamide.
Alexopoulos et al.[22]	78	M	PM (VVI)	Unchanged	PVC	1	ECT	None
Regestein et al.[23]	76	F	VOO	VOO	PVC	3	ECT	None
Silverman et al.[28]	80	M	PM	NR	PVC	1	ECT	NR
Goldberg et al.[20]	54	M	ICD	Deactivation	Hypertension	8	ECT	Labetalol
	65	M	ICD	Deactivation	Tachypnoea	1	Unknown	Furosemide
					Hypertension	1	ECT	Ephedrine
					Wide complex tachycardia	1	Unknown	Magnesium sulphate and Neosynephrine
Lapid et al. [20]	78	M	ICD	Deactivation	Tachycardia	1	Anticholinergic premedication	Esmolol
Dolenc et al.[20]	80	F	PM (DDD)	Unchanged	PVC	37	ECT	None
	72	M	PM (DDD)	Unchanged	PVC	15	ECT	None
	89	M	PM (DDDR)	Unchanged	PVC	108	ECT	None
	58	F	ICD	Unchanged	Tachycardia, rate dependent LBBB and Hypotension	1	Anticholinergic premedication	Esmolol
	87	M	PM (DDD)	Unchanged	SVT	1	Unknown	Amiodarone and Esmolol
MacPherson et al. [7]	86	F	PM (DDD)	Unchanged	Atrial fibrillation	1	Unknown	Hydralazine and Esmolol
Lynch et al. [33]	56	M	ICD & CMD	Deactivation	Hypertension	12	ECT	Esmolol prestimulation for initial 4 ECT and Post ECT Nitroglycerin and Hydralazine later.
Davis et al. [33]	68	M	ICD	Deactivation	Tachycardia, Hypertension	1	ECT	Esmolol and Labetalol
	87	F	ICD	Deactivation	Tachycardia, Hypertension	9	ECT	Labetalol
Kokras et al.[8]	62	M	PM (DDDR)	DDD	Hypertension	1	Anticholinergic premedication	Glyceryl trinitrate and clonidine
Streckenbach et al. [14]	49	F	ICD	Unchanged	Inappropriate ICD shock delivery	1	ECT mediated electromagnetic interference	Deactivation of ICD in subsequent ECTs
Magula et al. [14]	64	F	ICD	NR	Hypertension	38	ECT	Labetalol and Nitrogycerin
					Transient VT	8	ECT	Lignocaine

Author	Age	Sex	Type of CIED	Action	Adverse event	No of sessions	Possible cause	Management
Current report	55	M	PM(DDDR)	Unchanged	Tachycardia	5	Anticholinergic premedication	None

CMD - Cardiac contractility modulator, DDD- Dual chamber dual sensing, DDDR - Dual chamber dual sensing rate modulated, ECT - Electroconvulsive therapy, ICD - Implantable cardioverter defibrillator, LBBB - Left bundle branch block, NR - Not reported, PM - Pacemaker, PVC - Premature ventricular contraction, SVT - Supraventricular tachycardia

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Table 2
Summary of clinical considerations for ECT in patients with CIED.

1. Before ECT

- Multidisciplinary team liaison involving psychiatrist, anaesthesiologist, cardiologist, cardiac electrophysiologist.
- Device interrogation
  - ✶Type of CIED, model, and manufacturer details
  - ✶Indication
  - ✶Battery longevity and condition of the leads.
  - ✶Programming details and pacemaker dependency.
  - ✶Magnet rate and rhythm if planning to use a magnet,
  - ✶History of malfunctions or cardiac events.
- Assess patient's underlying rhythm and rate - assess the need for back up pacing.
- Anticholinergic if indicated - Preferably glycopyrrolate.
- Pacemaker - Asynchronous mode in pacemaker dependent patient/ turn off rate adaptation if excessive chest wall movement with breathing support is a concern.
- ICD - Deactivation of anti-tachycardia function.
- Ensure the availability of competent authority to handle CIED during ECT if necessary.

2. During ECT

- Proper grounding of all monitoring devices.
- Adequate electrical insulation of patient.
- Ensure that electronic devices are placed in safe distance away from the CIED.
- Monitor cardiac rhythm and peripheral pulse oximetry and disable artefact filter in ECG monitor if available.
- Ensure proper muscle relaxation to avoid myopotentials and use non-depolarizing muscle relaxants if succinylcholine-induced fasciculation is of concern.
- Avoid subthreshold stimulus.
- If EEG monitoring is not available to record the seizure, place the cuff in the lower limb to observe the seizure movement.

3. After ECT

- Monitor vitals and prompt detection and treatment of tachyarrhythmia and hypertension if indicated.
- Interrogation of CIED by competent authority.
- Turn on the rate adaptation and adjusting the pacing mode of pacemaker if modified before ECT.
- Re-enable the anti-tachycardia function ICD if modified before ECT.