Cardiac complications after ECT

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PRIFYSGOL CAERDYDD

After the administration of electric current there is a surge of cholinergic and adrenergic stimulation

Electric current during ECT → Surge of cholinergic and adrenergic stimulation → Increased cardiac workload. Complications among elderly or those with heart problems

<u>Complications</u>:

Asystole
Bradycardia
Tachycardia
Arrhythmias
Hypertension
ECG changes
Cardiac enzyme changes

Death rates are extremely low:
They are comparable to the mortality associated with minor procedures that involve anaesthesia

Mortality

- **Mortality:** 1:10,000 patients, 1:80,000 ECTs

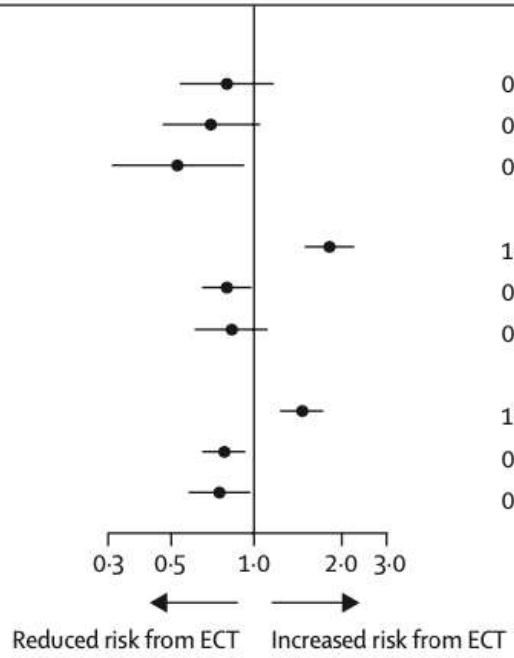
- No excess mortality after ECT: relative odds of all-cause mortality in the year after their index course of 0.87.

Risk of serious medical events in patients with depression treated with electroconvulsive therapy: a propensity score-matched, retrospective cohort study

THE LANCET Psychiatry
Volume 8, Issue 8, August 2021, Pages 686-695

Tyler S Kaster, Simone N Vigod, Tara Gomes, Rinku Sutradhar, Duminda N Wijeysundera, Daniel M Blumberger

	ECT-exposed		ECT-unexposed (ref)		Cause-specific HR (95% CI)
	n/N	Incidence (per 1000 person-years)	n/N	Incidence (per 1000 person-years)	
Death by suicide (primary outcome)					
Crude analysis	27/4982	5·84	423/62 345	7·26	0·80 (0·54–1·18)
Age-only weighting	27/4982	5·84	38/4988	8·30	0·70 (0·47–1·05)
Primary weighted analysis	27/4982	5·84	54/5304	10·90	0·53 (0·31–0·92)
Non-suicide death (secondary outcome)					
Crude analysis	111/4982	24·00	753/62345	12·92	1·86 (1·52–2·27)
Age-only weighting	111/4982	24·00	139/4988	30·10	0·80 (0·65–0·98)
Primary weighted analysis	111/4982	24·00	143/5304	29·00	0·83 (0·61–1·12)
All-cause mortality (secondary outcome)					
Crude analysis	138/4982	29·83	1176/62 345	20·18	1·48 (1·24–1·76)
Age-only weighting	138/4982	29·83	178/4988	38·36	0·78 (0·65–0·93)
Primary weighted analysis	138/4982	29·83	197/5304	39·88	0·75 (0·58–0·97)



The Cardiovascular Side Effects of Electroconvulsive Therapy and Their Management

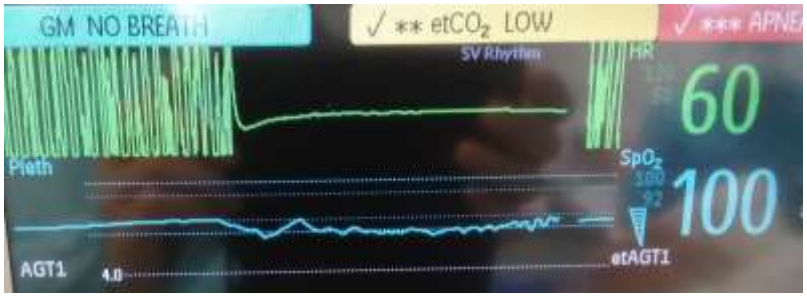
Adriana P. Hermida, MD, Mamoona Mohsin, MD,† Ana P. Marques Pinheiro, MD,* Elizabeth McCord, MD,* John C. Lisko, MD,‡ and Lyndsay W. Head, MD§*

Journal of ECT • Volume 38, Number 1, March 2022

ECT Journal Club, at 12 noon London time, first Wednesday of each month (almost each month)

Asystole

- After the electric stimulus there is initial stimulation of vagal nuclei that lead to parasympathetic surge
- This can cause bradycardia, hypotension and a transient asystole
- Q: How common is asystole during ECT (>2sec)?
  - 2%
  - 10%
  - 50%



Hase & Kettl, 2005: Asystole (>2sec) recorded 57 times out of 117 observations (48.7%).
Burd *et al*, 1998: 65.8% of a group of elderly patients experienced asystole at some time during their course of ECT (>5sec)

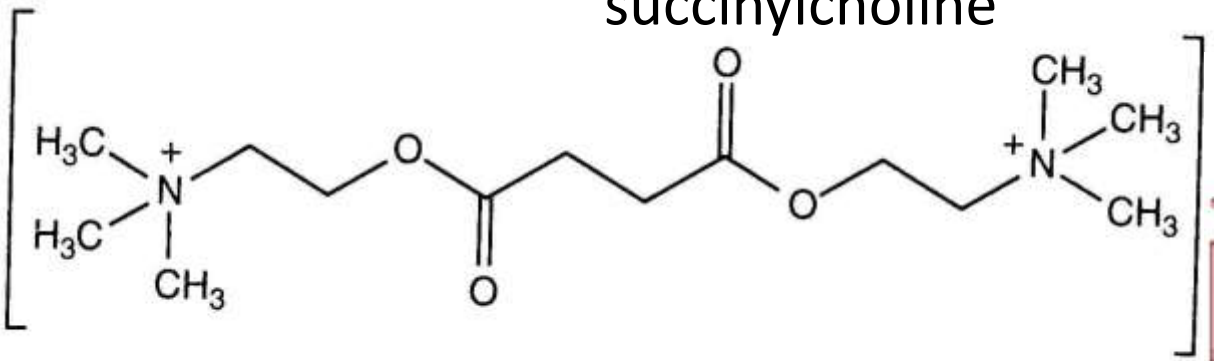
All resolved spontaneously

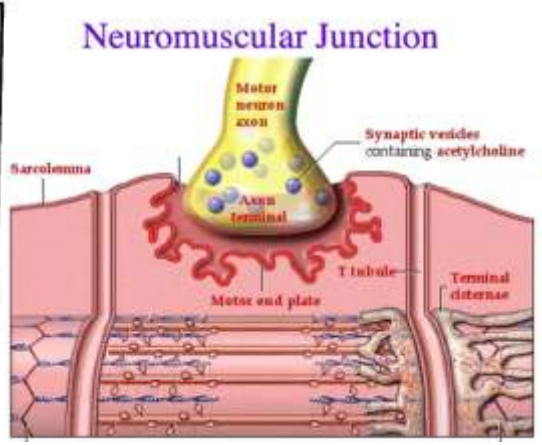
Risk factors for asystole/bradycardia

- Age > 65 years
- Use of beta blockers
- Cardiac disease
- Hypoxia
- High dose of succinylcholine
- Subconvulsive stimuli
- No anticholinergic pre-treatment
- Electrode placement

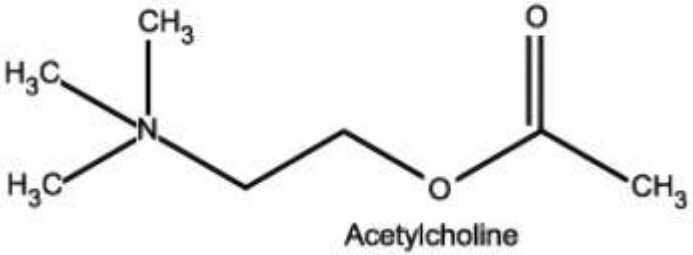
The role of succinylcholine

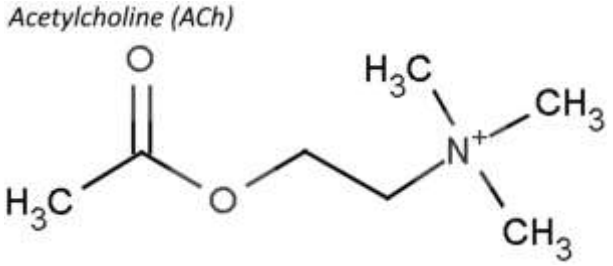
succinylcholine





succinylcholine acts by mimicking acetylcholine at the neuromuscular junction and blocking it





Because of the structural resemblance to acetylcholine, succinylcholine can cause bradycardia and asystole by stimulation of cardiac muscarinic receptors in the sinoatrial node. Even more likely to occur after re-dosing before re-stimulation

Q: Which electrode placement is least likely to cause asystole?



Bitemporal	Bifrontal	Right unilateral
11.6% (14/121)	2.6 % (2/79)	48.4 % (39/80)

The effect of electrode placement and pulsewidth on asystole and bradycardia during the electroconvulsive therapy stimulus

Patrick T. Stewart, Colleen K. Loo ✉, Ross MacPherson, Dusan Hadzi-Pavlovic
Author Notes

International Journal of Neuropsychopharmacology, Volume 14, Issue 5, June 2011, Pages 585–594, https://doi.org/10.1017/S1461145710001458

Predictors of Bradycardia During the Stimulation Phase of Electroconvulsive Therapy

Josef Nagler, MD† and Martin Geppert, MD**

(*J ECT* 2011;27: 201–206)

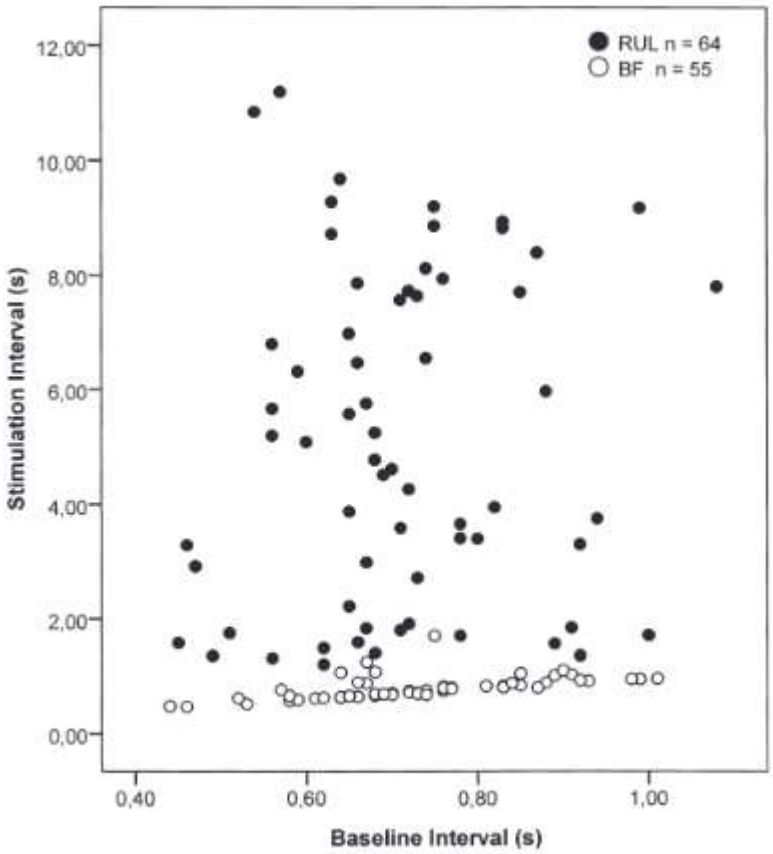
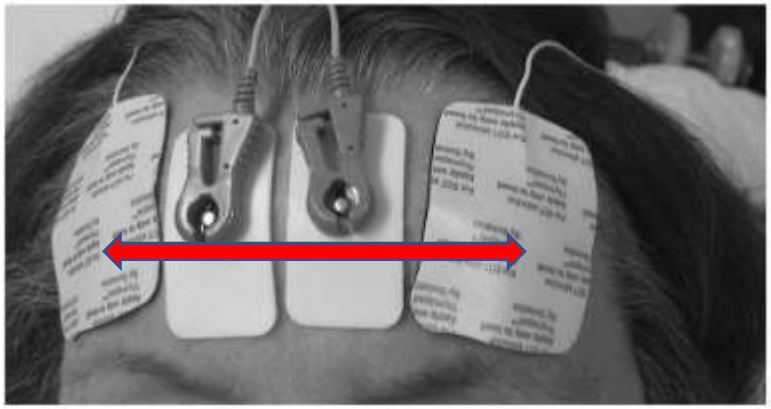


FIGURE 2. Interindividual comparison of mean stimulation intervals in RUL and BF treatment series.



Mean distance between electrodes = 10.3cm

Bifrontal placement partially avoids the brainstem nuclei

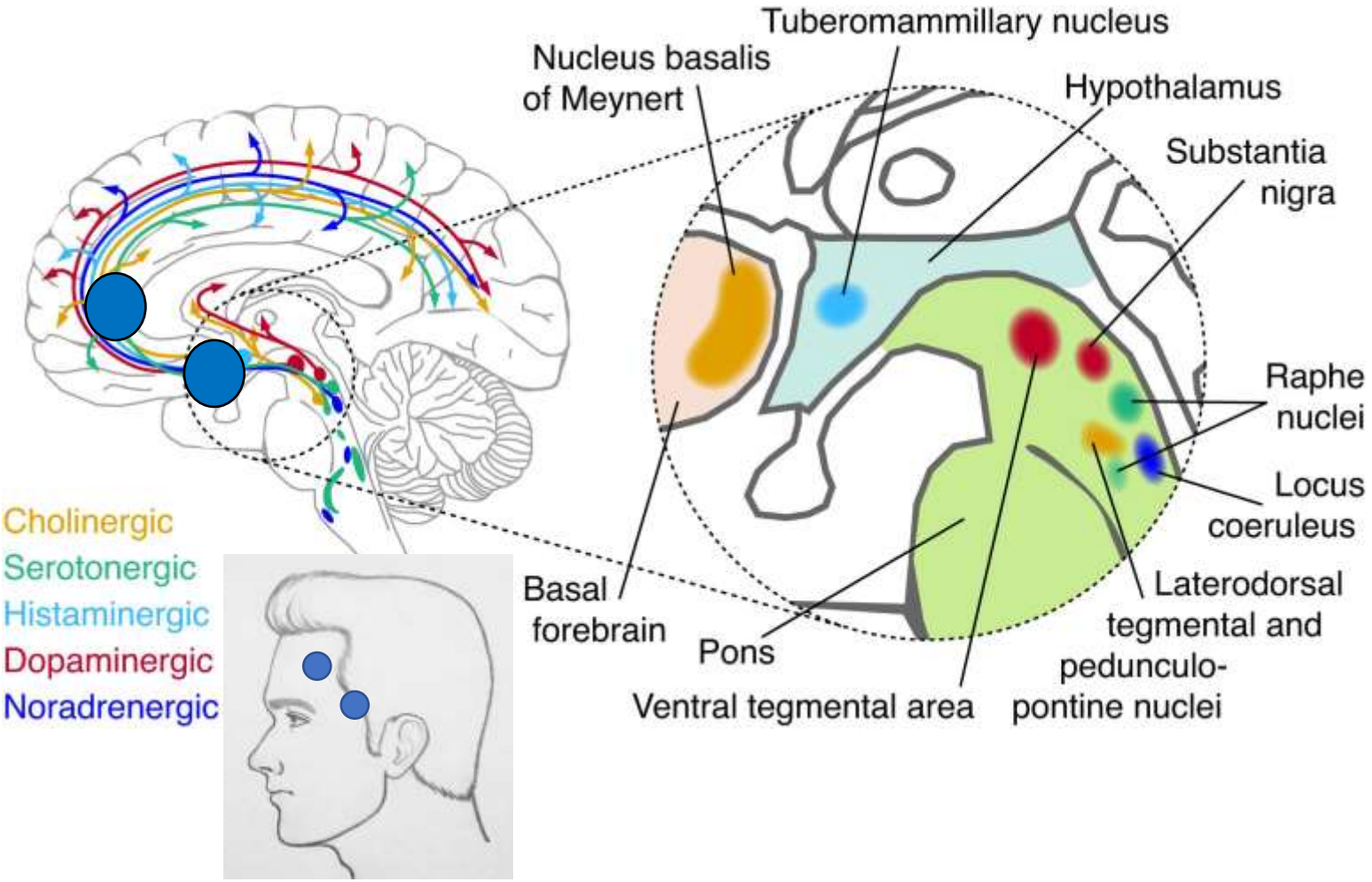
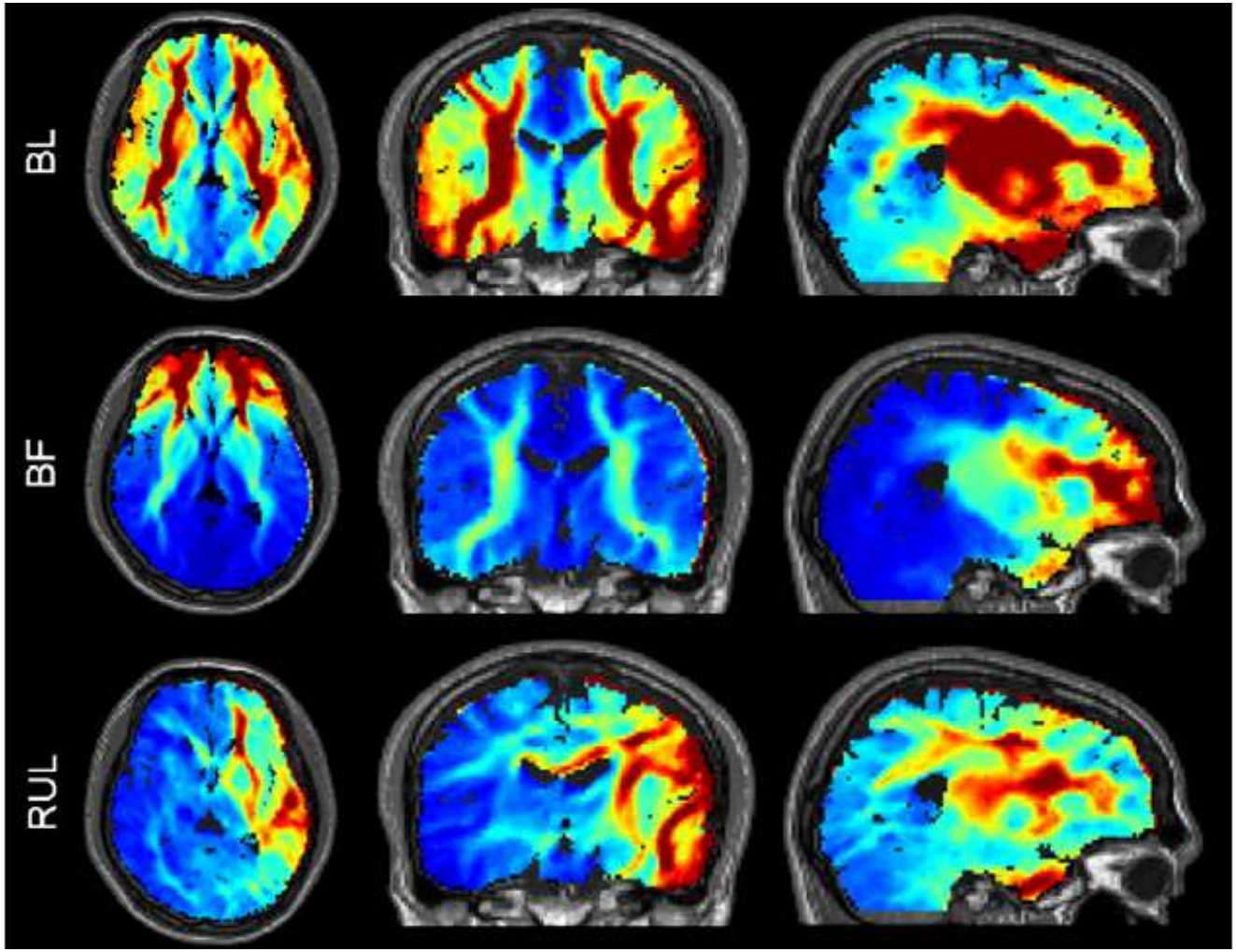


Image from van den Brink et al, Frontiers in Human Neuroscience, 2019

Regional electric field induced by electroconvulsive therapy in a realistic finite element head model: Influence of white matter anisotropic conductivity

Won Hee Lee [a,b], Zhi-De Deng [b,c], Tae-Seong Kim [d], Andrew F. Laine [a], Sarah H. Lisanby [b,e], Angel V. Peterchev [b,f,*]



Prevention of asystole and bradycardia

- Avoid pre-treatment with beta-blockers
- Treat with Glycopyrrolate (anticholinergic)
- Routine treatment with Glycopyrrolate is still debated and may not be necessary
- Asystole usually resolves on its own without any clinical consequence
- Use Bifrontal ECT
- Use a different muscle relaxant

Hypertension and tachycardia

- After the initial parasympathetic surge, there is a sympathetic surge that causes hypertension and tachycardia
- BP increases on average to 179/97, pulse rate to 126 bpm
- Usually well tolerated but in high risk patients antihypertensive medication should be used
- The ideal agent should have rapid action and quick elimination
- Q: Which beta-blocker is the most suitable for use in ECT?

Esmolol

Ultra-short-acting beta-one antagonist

Half-life 9 minutes

Supported by a literature review:

Review > Br J Anaesth. 2014 Jul;113(1):43-51. doi: 10.1093/bja/aeu153.

Beta-blocking agents during electroconvulsive therapy: a review

E Boere [1], T K Birkenhäger [2], T H N Groenland [3], W W van den Broek [2]

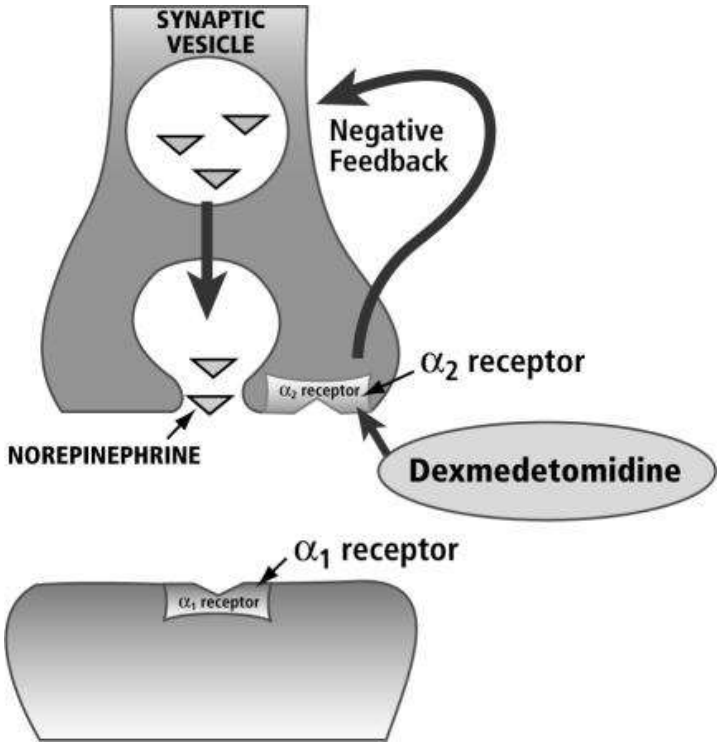
Other drugs used to treat hypertension and tachycardia

Calcium channel blockers

Dexmedetomidine:

α2 pre-synaptic receptor agonist, causes sedation and analgesia. These receptors regulate the release of norepinephrine through a negative feedback mechanism.

Similar to **clonidine**'s action



Seizure quality rating scale
(adapted from Sartorius et al, 2020)

Seizure Quality Rating Scale

- **Visible seizure duration** (duration of visible muscle contractions)
  - Less than 10s **- 0**
  - 10-15s **- 1**
  - Over 15s – **2**

- **EEG seizure duration** (duration of seizure activity on the EEG)
  - Less than 20s **- 0**
  - Over 20s – **1**

- **Mid-ictal amplitude** (If the overall amplitude of the seizure activity on the EEG is low, medium, or high)
  - Low (<50%) – **0**
  - Medium (50%-99%) **- 1**
  - High (100%) – **2**

- **Interhemispheric coherence** (Is there overall coherence between each hemisphere on the EEG)
  - No – **0**
  - Yes – **1**

- **Postictal suppression** (whether there is a clear end point and flat line to mark the end of seizure activity on the EEG)
  - Poor – **0**
  - Fair **- 1**
  - Good – **2**

- **Peak heart rate** (maximum heart rate measured during the seizure)
  - Less than 100 – **0**
  - 100-124 **- 1**
  - >125 – **2**

Effect of anaesthesia on hypertension and tachycardia

- Propofol causes hypotension, vasodilation, decreased cardiac output, and a reduction in sympathetic response and seizure duration.
- Ketamine may cause hypertension and tachycardia

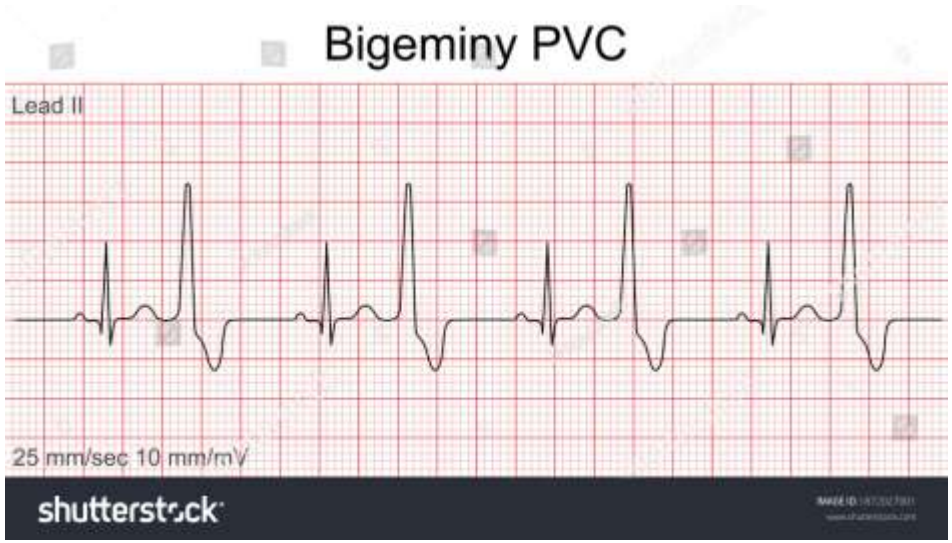
TABLE 2. Hypertension and Tachycardia

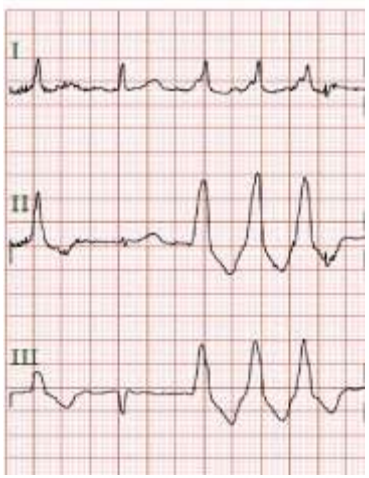
Considerations
Thorough cardiac evaluation before initiating ECT including evaluation for HTN, CHF, conduction abnormalities, HR or rhythm abnormalities, coronary artery disease
Prophylaxis with antihypertensive drugs in presence of cardiac risk factor as above
Prophylaxis with antihypertensive drugs if patient experiences substantial HTN and/or tachycardia during ECT
Choice of anesthesia and antihypertensive drug on a case-by-case basis per requirement
Avoid β-blocker use unless risk of not taking it is greater than consequences of its interaction with ECT

However: Beta-blockers may be associated with asystole

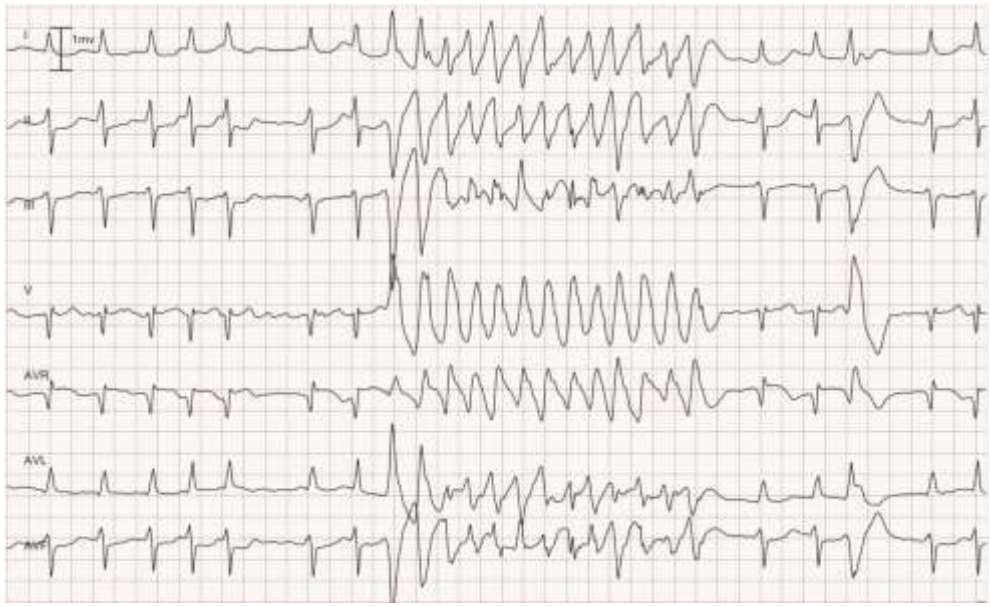
Arrhythmia is the most common complication of ECT

- Premature atrial contractions
- Premature ventricular contractions
- Bigeminy
- Ventricular tachycardia
- Heart block





Triplet



Ventricular tachycardia

Which electrode placement is associated with the lowest frequency of arrhythmias?

The effect of electrode placement and pulsewidth on asystole and bradycardia during the electroconvulsive therapy stimulus

Patrick T. Stewart, Colleen K. Loo ✉, Ross MacPherson, Dusan Hadzi-Pavlovic
Author Notes

International Journal of Neuropsychopharmacology, Volume 14, Issue 5, June 2011, Pages 585–594, https://doi.org/10.1017/S1461145710001458

No instances of bigeminy with bifronal ECT

Methohexital and propofol are associated with a reduction in arrhythmias compared to thiopental

Premedication with atropine may decrease the occurrence of premature atrial contractions

Atrial fibrillation (AF)

- There are case reports of new-onset AF after ECT
- There are cases of chronic AF converting to sinus rhythm after ECT
- Due to the risk of embolization with cardioversion, Petrides & Fink (1996) recommend anticoagulation of AF patients during ECT courses

Recommendations

TABLE 3. Arrhythmia

Considerations
Continuous ECG monitoring during ECT
More caution in patients age >60 at risk
If possible, stop or stabilize medicines that interfere with cardiac conduction
Have a readily available defibrillator
Consider BF stimulus in case of recurrent, prolonged arrhythmias

Cardiac enzymes

- 3 small studies (<100 cases)
- Cardiac troponin / cardiac troponin I
- 4-5% of cases had transient elevations, MI diagnosed in two cases. (Martinez *et al*, 2011)
- "*Given the physiologic stress associated with both anaesthesia and ECT, a mild elevation in troponin is not unexpected and may be clinically insignificant*"
- "*In the absence of symptoms, routine collection of troponins is not clinically indicated*"

ECT has been given to patients with pre-existing cardiovascular disease

- Heart failure
- Abdominal aortic aneurisms
- Cerebral aneurisms
- Aortic stenosis
- Pulmonary embolism
- Findings are from case reports only and do not provide proof of safety

Safety of Electroconvulsive Therapy in Patients With a History of Heart Failure and Decreased Left Ventricular Systolic Heart Function

Fernando A. Rivera, MD, Maria I. Lapid, MD,† Shirlene Sampson, MD,† and Paul S. Mueller, MD**

(J ECT 2011;27: 207-213)

- 35 patients treated at the Mayo clinic 1995-2009
- Median age 77 years
- Left ventricular ejection fraction 40%-15%
- All tolerated ECT well
- i.v. beta-blockers given to 26 patients

Conclusions

- Heart rate and blood pressure increase by 20-30%
- Older patients are at a higher risk
- Most complications are minor and manageable
- Cardiac disease increases risk for depression and depression increases risk for cardiac disease. These patients need effective treatments
- With appropriate caution and management, high-risk patients can be safely treated with ECT

Part 2: Pacemakers

Electroconvulsive Therapy in Patients With Cardiac Implantable Electronic Devices

A Case Report and Systematic Review of Published Cases

Abhiram Narasimhan Purohith, MD, Sivapriya Vaidyanathan, MD,* Suma T. Udupa, DNB,* Ravindra N. Munoli, MD,* Sheena Agarwal, MBBS,* Mukund A. Prabhu, MD, DM, PDF,† and Samir Kumar Praharaj, MD**

From the Departments of *Psychiatry and †Cardiology, Kasturba Medical College, Manipal, Manipal Academy of Higher Education, Manipal, India.

J ECT, 2022

The heart conduction system

Pacemaker indications

Sick sinus syndrome

Syncope

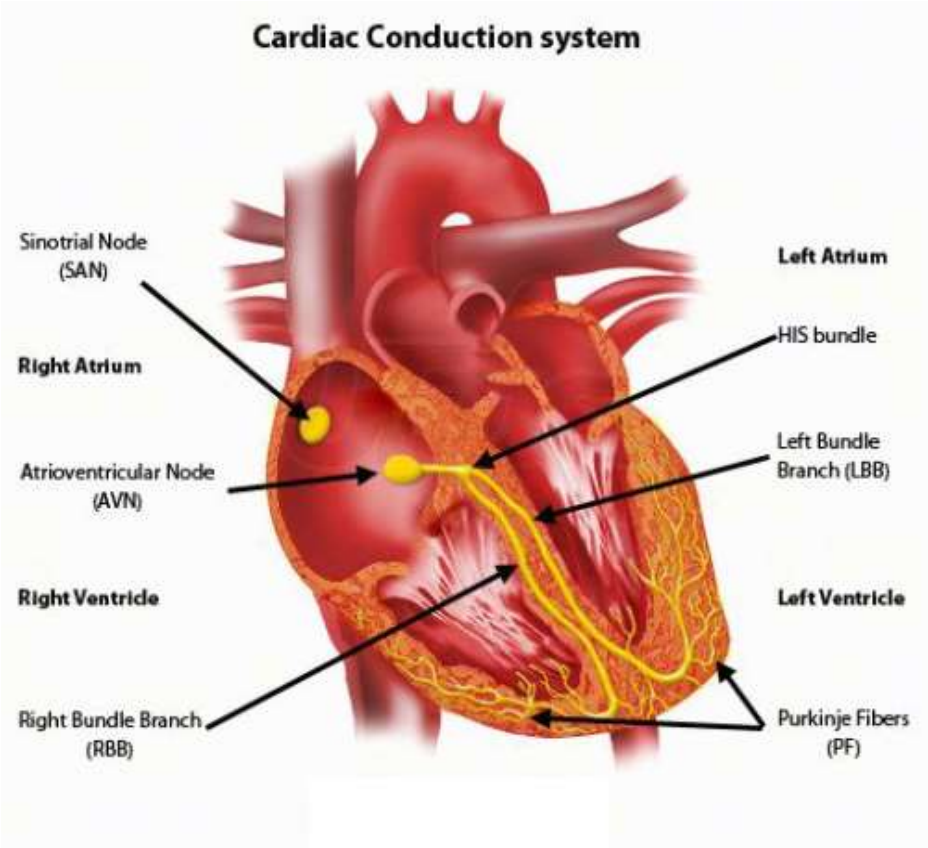
Bradycardia

Cardiac arrest

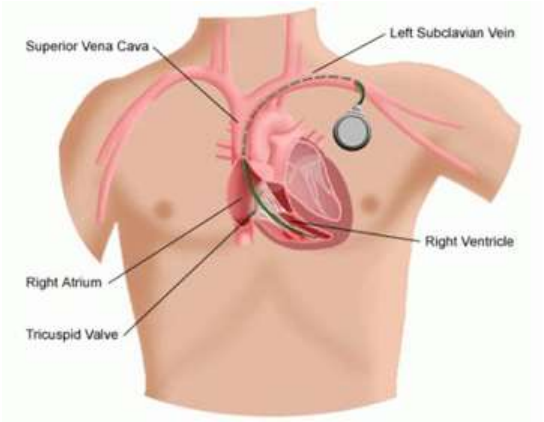
Heart block

Ventricular tachycardia

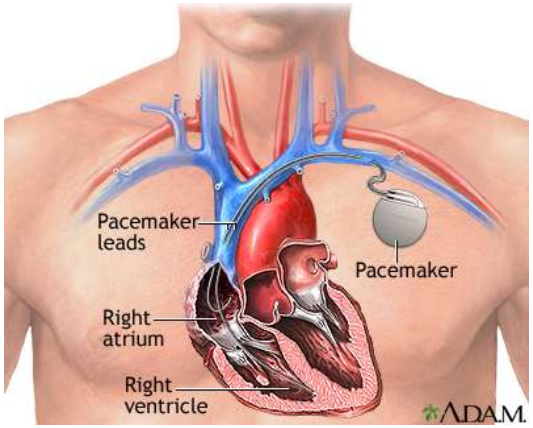
Heart failure



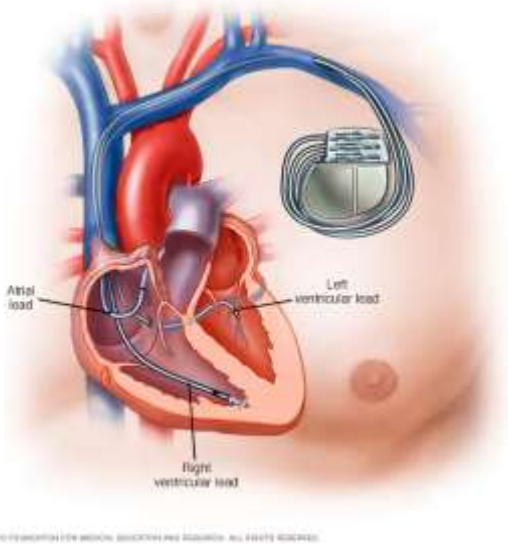
Types of pacemakers



Single lead: a single lead is attached in the right atrium or right ventricle

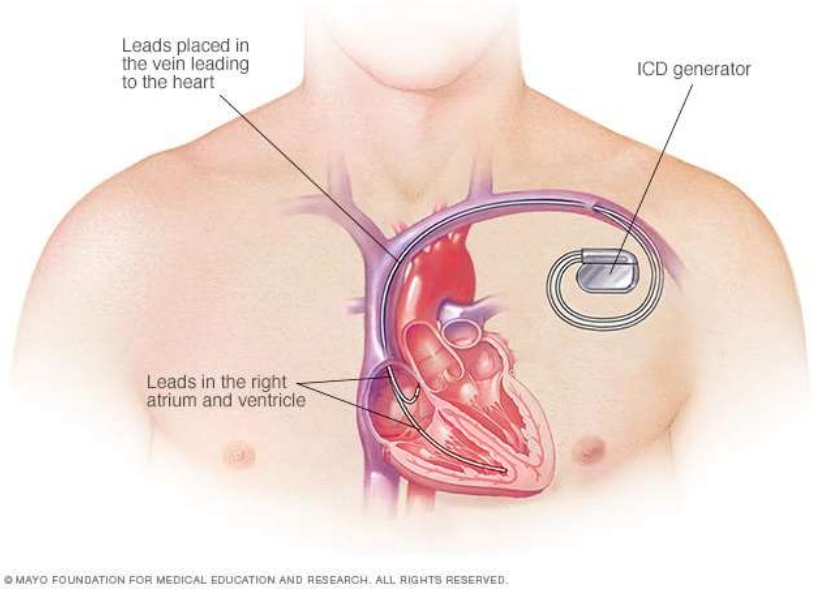


Dual lead: There is one lead in the right atrium and one in the right ventricle



Biventricular pacemaker: There is one lead in the right ventricle and one in the left ventricle. There can be a third lead in the right atrium as well.

Implantable cardioverter-defibrillator (ICD)

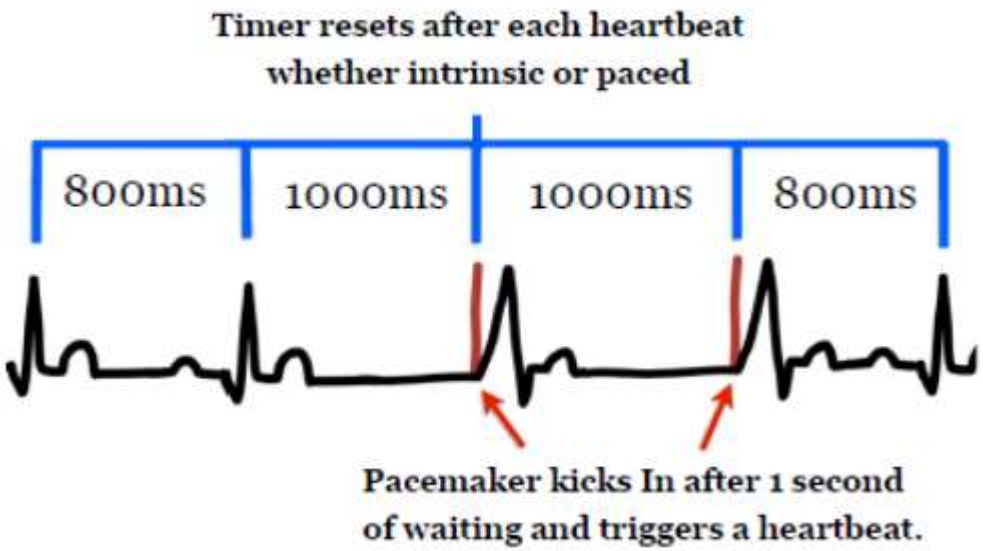


An ICD continuously monitors the heartbeat and delivers electric shocks, when needed, to restore a regular heart rhythm. Many devices combine a pacemaker and ICD

Sensing and pacing

- Sensing: Pacemaker leads sense the heart electrical activity. It “knows” that the heart has contracted.
- Pacing: If the pacemaker does not “sense” a beat, it will deliver a small electrical charge to trigger a heartbeat.

Base rate (the slowest heart rate allowed)



If the pacemaker detects physical activity, it gently increases the heart rate. Does this increase happen during seizure?

What can go wrong during ECT?

- Implantable Cardioverter Defibrillator (ICD) can trigger a shock (defibrillation) around the ECT
- Electromagnetic interference can affect sensing and the pacemaker can misinterpret the muscular or electrical activity as normal heart beats and stop pacing
- Fasciculations in skeletal muscles during muscle relaxation can inhibit the pacemaker (Modern pacemakers are not affected)
- Electrical activity can damage the pacemaker

- Thirty-five publications across 53 years (1967–2021) reported on 76 patients
- Did not find any published report on ECT in patients with cardiac resynchronization therapy device.
- The overall quality of reported cases ranged from good (all 5 parameters are described) to moderate (few parameters are missing).
- 8 (11%) did not have the description and pacing mode
- 61 patients (806 sessions) with pacemakers,
- 13 patients (148 sessions) with ICDs,
- 25 sessions in one patient with pacemaker + ICD
- Most patients (76%, n = 57) were older than 65 years

Indications for pacing / ICD

- For pacemaker implantation: 1) atrioventricular (AV) block (43.5%, n = 27),
  2) sinus node dysfunction (27.4%, n = 17),
- For ICD: Ventricular tachycardia was most common indication (64.2%, n = 9)
- Most common pacing mode: dual chamber with dual sensing pacemakers (51.6%, n = 32), followed by ventricular pacing with ventricular sensing mode (27.4%, n = 17).

What was done to the pacemaker/ICD?

Pacemakers (61 patients):

The pacing mode was unchanged during the ECT sessions in 45 patients (77.4%). No adverse effects

For 66 ECT sessions pacing was changed to asynchronous mode (no sensing)

ICDs (13 patients):

In 10 out of 13 patients who had ICD, the device was deactivated before ECT sessions and later reprogramed

One reported instance of inappropriate ICD shock delivery

Adverse events in 25% of cases

- Premature ventricular contraction was the most common adverse effect but it recovered spontaneously.
- Hypertension was the most common (7.1%) adverse effect that needed medical intervention (in 70 out of 979 ECT sessions), easily managed without any serious consequences.
- No reports of serious adverse effects that necessitated stopping ECT.
- Other complications: Atrial flutter, atrial fibrillation, tachycardia, one transient ventricular tachycardia

TABLE 1. List of the Reported Adverse Effects

Author	Age	Sex	Type of CIED	Action	Adverse Event	No. Sessions	Possible Cause	Management
Ballenger[13]	50	M	PM (VVI)	Unchanged	Atrial flutter	1	Anticholinergic premedication	Cardioversion and procainamide.
Alexopoulos et al[14]	78	M	PM (VVI)	Unchanged	PVC	1	ECT	None
Regestein et al[15]	76	F	VOO	VOO	PVC	3	ECT	None
Silverman et al[16]	80	M	PM	NR	PVC	1	ECT	NR
Goldberg et al[17]	54	M	ICD	Deactivation	Hypertension	8	ECT	Labetalol
	65	M	ICD	Deactivation	Tachypnea	1	Unknown	Furosemide
					Hypertension	1	ECT	Ephedrine
					Wide complex tachycardia	1	Unknown	Magnesium sulphate and neosynephrine
Lapid et al[18]	78	M	ICD	Deactivation	Tachycardia	1	Anticholinergic premedication	Esmolol
Dolenc et al[6]	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[19]	56	M	ICD and CMD	Deactivation	Hypertension	12	ECT	Esmolol prestimulation for initial 4 ECT and post-ECT nitroglycerin and hydralazine later.
Davis et al[20]	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[12]	49	F	ICD	Unchanged	Inappropriate ICD shock delivery	1	ECT mediated EMI	Deactivation of ICD in subsequent ECTs
Magula et al[21]	64	F	ICD	NR	Hypertension	38	ECT	Labetalol and nitroglycerin
					Transient VT	8	ECT	Lignocaine
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; LBBB, left bundle branch block; NR, not reported; PM, pacemaker; PVC, premature ventricular contraction; SVT, supraventricular tachycardia; VVI, ventricular demand pacing; VOO, asynchronous ventricular pacing.

Recommendations from paper and ECT Handbook

- Discuss with cardiologist and electrophysiologist
- Patients with pacemakers can be treated safely. No need to switch off device
- Glycopyrrolate if needed
- Ensure proper muscle relaxation
- Use bifrontal ECT
- Patients with cardioverted defibrillator (ICD): a cardiology technician should be present. Switch off or leave on? It is possible to leave it active, as the benefit of shocking a life-threatening arrhythmia outweighs the risk of delivering a wrong shock (Bryson *et al*, 2015).

BRIEF CLINICAL REPORT LETTER

Automatic Implantable Cardioverter Defibrillator in Electroconvulsive Therapy

Bryson, Ethan O. MD*; Popeo, Dennis M. MD†; Briggs, Mimi C. BA†; Pasculli, Rosa M. BA†; Kellner, Charles H. MD†

Author Information

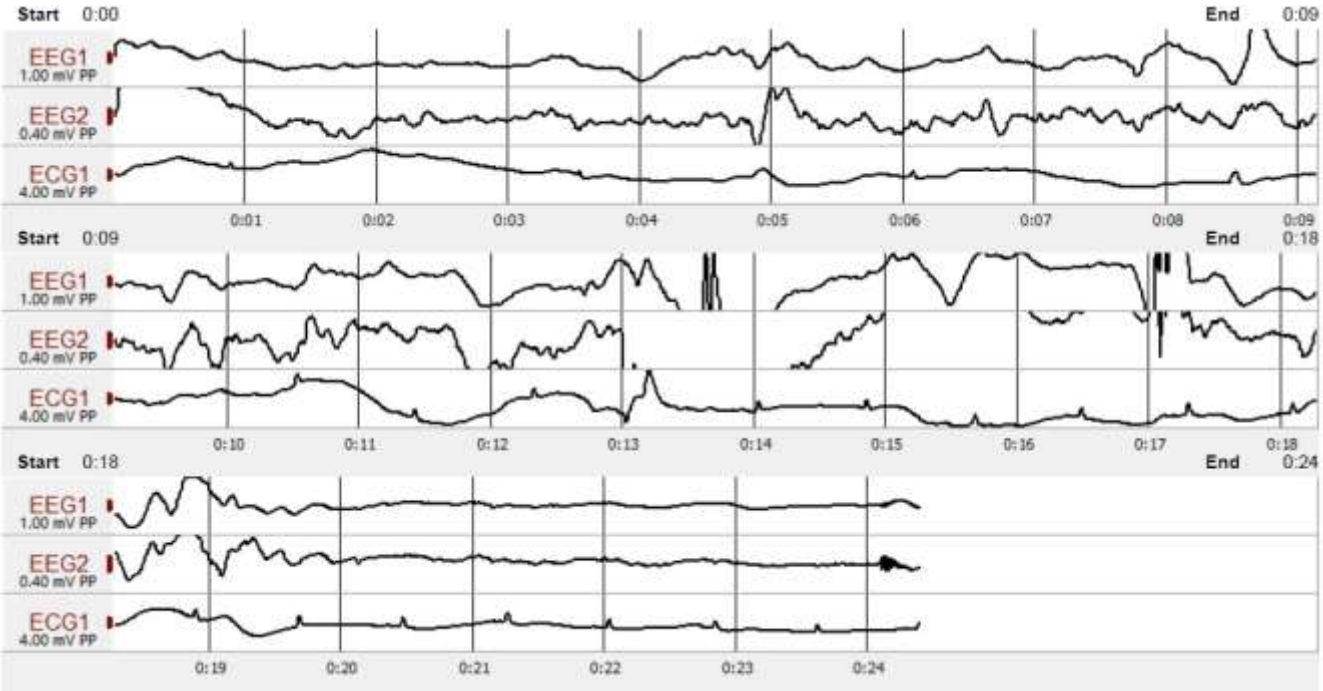
The Journal of ECT: March 2015 - Volume 31 - Issue 1 - p e22

- 85-year-old woman with treatment refractory depression and an implanted cardioverter-defibrillator. Dual chamber at 60 beats per minute
- Before the first ECT treatment, the device was deactivated via magnet applied to the chest wall, and no hemodynamic changes were noted
- The magnet was removed from the chest wall immediately after the end of seizure activity
- No ventricular arrhythmias or tachycardia at the first 4 treatments
- Decision was made to leave the device active for the remaining treatments

The experience of Declan McLoughlin's group

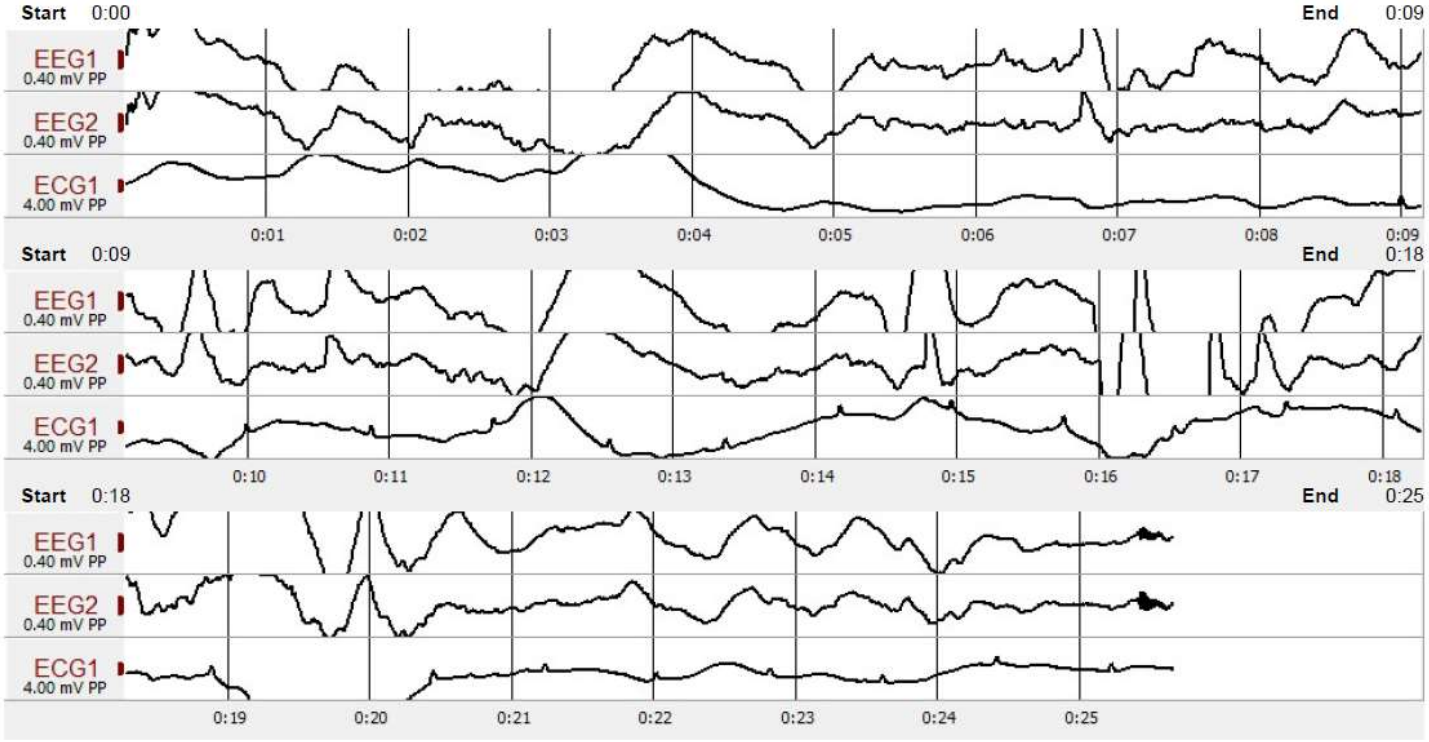
- "Our experience with regular pacemakers is that there is no major issue and we proceed as normal.
- For implantable defibrillators, one needs a cardiac technician to turn off the device just before the ECT session and then turn it back on again."

Cardiff case. 1st stimulation, 126mC



Bradycardia during the first 11 seconds, no action, proceed to 2nd stimulation

Cardiff case, 2nd stimulation, 184mC



Second stimulation is followed by 9s asystole, then sinus rhythm, but after 5 minutes the rate slows down to 29/min. Escape rhythm generated in the AV node. Transferred to the general hospital, one week later implanted with pacemaker.

"This lady has a dual-chamber pacemaker. It is programmed to sense her SA node rate and to pace the ventricles accordingly. If her SA node rate increases due to exercise/stress/emotion, then the pacemaker rate will increase accordingly. Her rate will not drop below 60bpm."

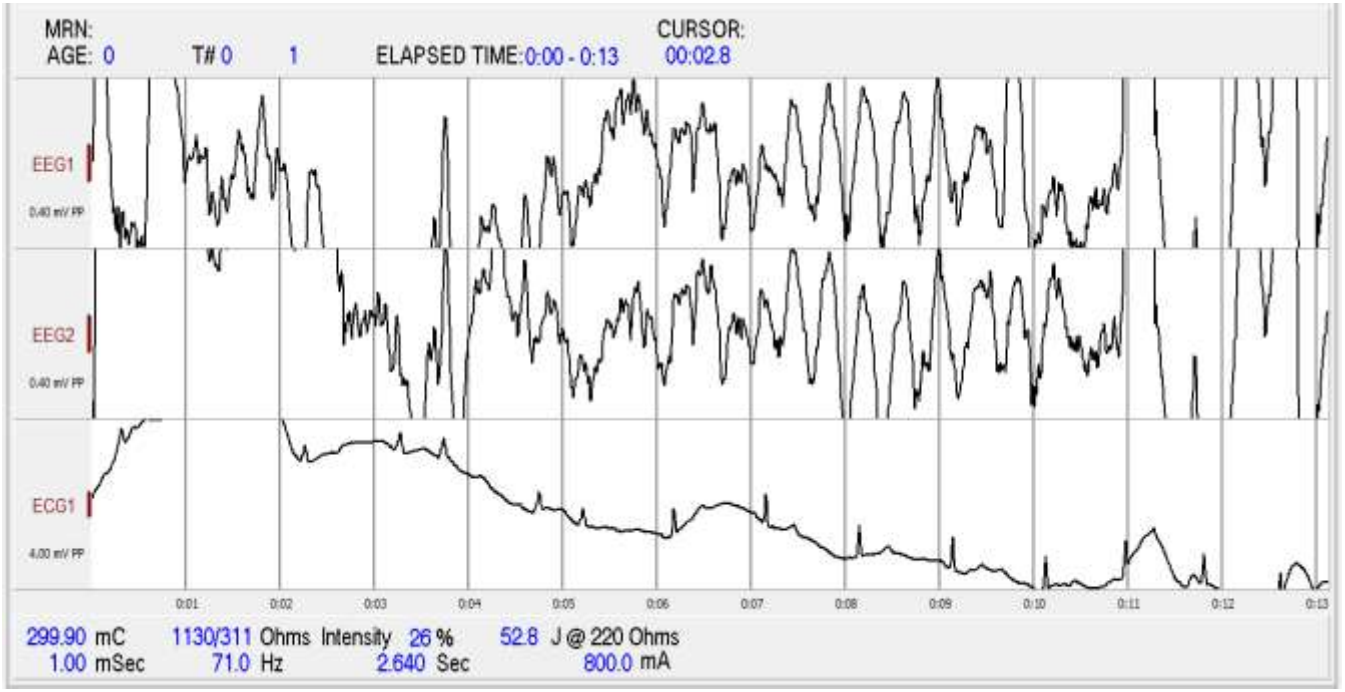


Figure: ECT with pacemaker left on. No problems around ECT so far.