WEAVE SPRINT . PAPER III

ECT and neurostimulation

The treatment that works fastest when the illness is most dangerous, and the family of stimulation techniques that grew up around it. How electroconvulsive therapy is actually delivered: the seizure and the theories of why it helps, the anaesthetic, the electrodes, and the dose that decides both how well it works and what it costs the memory. Then the course, the maintenance, and the drugs that fight it. Closing on the modern devices, from currents that never induce a seizure to electrodes left in the brain. Throughout, the Indian law that permits only the modified form.

CONTENTS

- Electroconvulsive therapy: foundations and indications
- Electroconvulsive therapy: technique and dose
- Electroconvulsive therapy: effects, consent and course
- Other neurostimulation therapies

Dr. Wilfred D'souza . Dr. Niharika Reddy

WEAVE . CENTRE FOR INTEGRATIVE PSYCHIATRY

Weave
Centre for Integrative Psychiatry

WEAVE SPRINT
PSYCHIATRY OUTREACH PROGRAMME

Psychiatry that listens before it prescribes.

Therapy-informed psychiatry for adults, children, couples and families. Online across India, in person in Mumbai.





Assessments

ADHD, autism and developmental. Anxiety, mood, attention and personality patterns.



Consultations and sessions

Psychiatric consultations, therapy sessions, medication review and couples sessions.



Family Library

Free plain-language guides for families, in five languages.

`weave.clinic/family`

REFER, OR SEND SOMEONE OUR WAY

Your patient contacts Weave directly and decides whether to book.

You remain the referring clinician. Any coordination between clinicians happens with the patient's permission.

CONTACT

`weave.clinic/contact`
`WhatsApp +91 7625004229`

Dr. Niharika Reddy and Dr. Wilfred D'souza, Consultant Psychiatrists. Runwal Forests Clinic, Kanjurmarg West, Mumbai, and online.

WEAVE.CLINIC

Routine referrals only. This is not an emergency route.

Contents

Orientation: the induced seizure and the stimulated brain 4

1 Orientation: the induced seizure and the stimulated brain 4

Electroconvulsive therapy: foundations and indications 9

2 History and evolution of ECT 9

3 Mechanism of action and neurobiological theories of ECT 15

4 Indications for ECT (depression, mania, catatonia, schizophrenia, emergencies) 22

5 Contraindications and high-risk conditions for ECT 30

Electroconvulsive therapy: technique and dose 37

6 Pre-ECT workup and anaesthesia (agents, muscle relaxant, airway) 37

7 ECT technique and electrode placement (bilateral, right unilateral, bifrontal) 46

8 Bilateral versus right unilateral ECT (efficacy versus cognitive trade-off) 53

9 Stimulus dosing, seizure threshold and adequate seizure criteria 60

10 Modified versus unmodified (direct) ECT 68

Electroconvulsive therapy: effects, consent and course 74

11 Cognitive side effects of ECT (anterograde and retrograde amnesia) 74

12 Other complications and adverse effects of ECT (cardiovascular, prolonged seizure) 82

13 Consent for ECT and legal aspects under MHCA 88

14 ECT in special populations (pregnancy, elderly, adolescents) 94

15 Course, frequency and number of ECT sessions 101

16 Continuation and maintenance ECT 106

17 Drug interactions and concurrent medications with ECT 112

Other neurostimulation therapies 119

18 Repetitive transcranial magnetic stimulation (rTMS): mechanism, indications, parameters 119

19 Transcranial direct current stimulation (tDCS) 125

20 Deep brain stimulation (DBS) in psychiatry 131

21 Vagus nerve stimulation (VNS) 137

22 Magnetic seizure therapy and emerging neuromodulation 142

23 Psychosurgery (history, current indications, ethics) 146

Cross-cutting synthesis 153

24 Cross-cutting synthesis 153

Apply and consolidate 161

25 Applying the chapter 161

26 Consolidation 169

Previously asked 174

The quick review 178

References 187

PAPER III

ECT and neurostimulation

Six bands . one complete notes document

Orientation: the induced seizure and the stimulated brain

1 · Orientation: the induced seizure and the stimulated brain

Electroconvulsive therapy is the most effective acute treatment available in severe mood and catatonic states, and its price is measured in cognition. A brief electrical stimulus crosses the skull of an anaesthetised and relaxed patient, a generalised cerebral seizure follows, and an illness unshifted by weeks of drug treatment can shift within days. From that standing and that price follows everything the chapter teaches: what the treatment is for, how it is delivered, what it costs, and who may lawfully have it.

This opening section is the chapter's **advance organizer**. It asserts no specific of its own: no dose, charge, threshold multiple, percentage or session count appears, because each belongs to its owner. What it owns is the shape: the standing of the treatment, the sequence in which the chapter delivers it, the non-ECT techniques read against it, and the Indian legal frame. The accompanying figure lays the same architecture out visually.

Most effective	No single law	Modified only
THE STANDING OF THIS TREATMENT IN SEVERE MOOD AND CATATONIC STATES	THE TRADES DIFFER BY PARAMETER AND BY PLACEMENT, SO NONE IS INHERITED	THE INDIAN LEGAL FRAME THE WHOLE CHAPTER SITS INSIDE

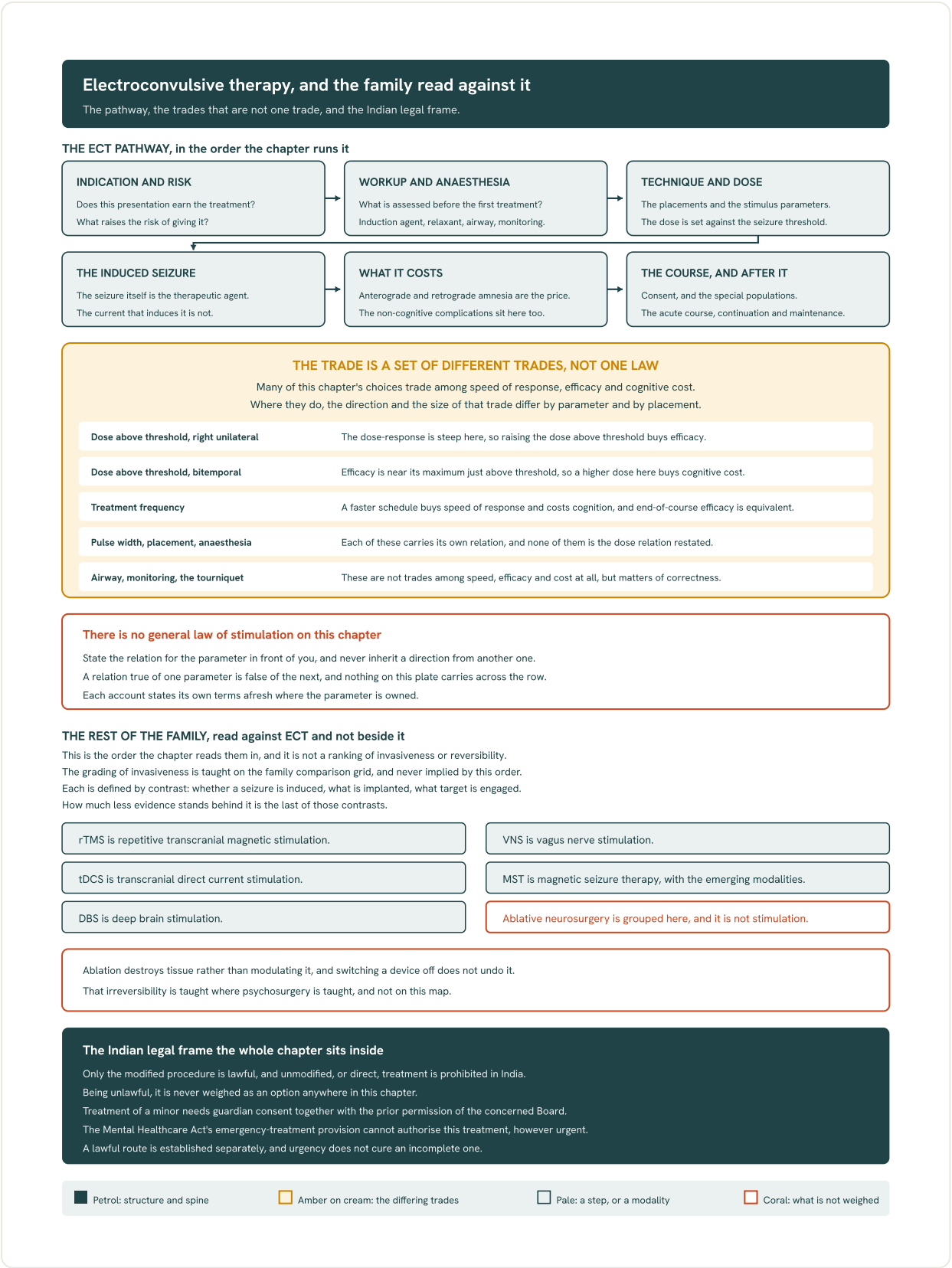


Fig 27.1. The chapter's architecture: the ECT pathway from indication through workup, technique, the induced seizure and its costs to the course and its maintenance; the trades that differ by parameter and by placement rather than following any single law; the rest of the neuromodulation family in a reading order that ranks nothing, with ablative neurosurgery grouped there but not stimulation; and the Indian legal frame the whole chapter sits inside.

1.1 What the treatment is, and where its power lies

The therapeutic agent is the **induced seizure**, not the current that induces it and not the convulsion once visible at the bedside. Modification abolishes the motor convulsion and leaves

the cerebral seizure untouched, so a treated patient lies still. Its power concentrates where illness is severe or immovable: severe depression, above all with psychotic features, food refusal or acute suicidal risk; catatonia, which responds better than almost anything in psychiatry responds to anything; acute and refractory mania; and defined presentations of schizophrenia, including the clozapine non-responder. The evidence under each belongs to the indications account.

The counterpart is a real and measurable effect on memory, and a smaller burden of non-cognitive complication, principally cardiovascular. Neither is a reason to withhold the treatment from a patient who needs it.

1.2 The structural fact the chapter turns on

Many of the choices in this chapter trade among speed of response, efficacy and cognitive cost, and where they do, the direction and the size of that trade differ by parameter and by placement. That is a structural fact, not a directional law. The chapter asserts no law covering all of these choices, because not every choice is such a trade at all: airway management, physiological monitoring and the tourniquet that isolates one limb from the relaxant are not purchases of efficacy at a cognitive price.

Its terms are stated afresh by the topic owning the parameter. The table below is not a rule but a demonstration that no rule applies across the row.

PARAMETER	WHAT THE RELATION ACTUALLY IS	STATED IN FULL BY
STIMULUS DOSE ABOVE THRESHOLD, RIGHT UNILATERAL PLACEMENT	The dose-response is steep, so raising the dose above the seizure threshold buys efficacy.	Stimulus dosing, and the placement comparison.
STIMULUS DOSE ABOVE THRESHOLD, BITEMPORAL PLACEMENT	Efficacy is already near-maximal at a low multiple of threshold, so a higher dose here largely buys cognitive cost alone.	The same accounts, stating it as differing by placement.
TREATMENT FREQUENCY	Buys speed of response and costs cognition. End-of-course efficacy is equivalent between the schedules, so a faster schedule is never listed among the choices that buy efficacy.	The acute course and its frequency.
PULSE WIDTH, ELECTRODE PLACEMENT, ANAESTHETIC CONDUCT	Each carries its own relation, none the dose relation restated.	Technique, and the workup and anaesthesia.
AIRWAY, MONITORING, THE TOURNIQUET	Not a trade among speed, efficacy and cognitive cost. Correctness, not currency.	The workup and anaesthesia.

RULE **State the relation for the parameter in front of you, and never inherit a general direction from a different parameter.** There is no general law of stimulation here. A sentence coupling how hard a treatment is made to work to what it costs in memory is never written on this chapter, however qualified, because a relation true of one parameter is false of the next, and the reader who carries it across will dose a patient on it.

MNEMONIC

DECIDE, DELIVER, LIVE WITH IT, LOOK OUTSIDE

Decide whether this patient should have the treatment at all. **Deliver** it: workup and anaesthesia, technique and dose, and the seizure that results. **Live with it**: what it costs, how consent is lawfully established, and how the course is sustained. **Look outside** to the non-ECT family, defined by contrast with what you have just read. Every section sits inside one of those movements.

1.3 The ECT pathway, in the order the chapter runs it

The ECT sections follow the sequence a resident moves through, and examiners ask about its joints.

- **Indication and risk.** Where the treatment came from and how it became modified; the competing accounts of how it works; the indications and their evidence; and the conditions that raise risk without barring it.
- **Workup and anaesthesia.** What is assessed before the first treatment, what is not routinely needed, and the anaesthetic conduct: induction agent, relaxant, airway and monitoring.
- **Technique and dose.** The placements and what distinguishes them; the parameters that define a stimulus; the threshold and the dose as a multiple of it; the placement comparison as a function of that multiple; and the modified procedure against the unmodified one.
- **The induced seizure.** What makes a seizure adequate, what to do when none follows, and what to do when one does not stop.
- **What it costs.** The anterograde and retrograde amnesias, how they are monitored and how they behave; and the non-cognitive complications.
- **The course, and life after it.** Consent as a lawful route; pregnancy, old age and adolescence; the acute course and its frequency; continuation and maintenance; and the drugs that move the threshold.

1.4 The rest of the family, read against ECT and not beside it

The chapter then reads the remaining modalities in this order: repetitive transcranial magnetic stimulation, transcranial direct current stimulation, deep brain stimulation, vagus nerve stimulation, magnetic seizure therapy with the emerging modalities, and ablative neurosurgery. That is a reading order and not a ranking of invasiveness or reversibility; the grading is taught on the comparison grid the cross-cutting synthesis owns.

Every device in this **neuromodulation** family is defined by contrast with what you have just read: whether a seizure is induced, what is implanted if anything, what target is engaged, and how

much less evidence stands behind it.

One member is not a member. The bank groups **ablative neurosurgery** with the stimulation modalities and this chapter follows it, but ablation is not stimulation. It destroys tissue rather than modulating it, and switching a device off does not undo it. That irreversibility is taught where psychosurgery is.

1.5 The Indian legal frame the whole chapter sits inside

A short list of statutory positions governs practice, each named in words, never renumbered. First, only the modified procedure is lawful. Unmodified, or direct, electroconvulsive therapy is prohibited in India. It is not the older technique, not the resource-limited compromise and not the weaker option: it is unlawful, and is never weighed as an option in this chapter. Second, treatment of a minor is gated: it requires guardian consent together with the prior permission of the concerned Board, sought in advance. Third, the Act's emergency-treatment provision cannot authorise this treatment, however urgent the clinical picture.

That third position is the trap most often examined. A patient can be in a state where the treatment is the correct and urgent answer while the emergency provision still cannot authorise it, so a **lawful route** is established separately and urgency does not cure an incomplete one. This is a point about treatment authority and not admission authority: a capacitous adult can consent to this treatment with no admission at all.

> **IN INDIA**
>
> The Indian frame is not a stricter version of a Western one but different in kind. A prohibition that elsewhere would be a professional standard is here a statutory bar, a treatment decision for a minor passes through a statutory body, and the urgency that would settle the question elsewhere settles nothing. Where a topic has an Indian answer, that is the answer.

1.6 The edges of the map: named here, taught elsewhere

An advance organizer is as much about what sits outside the frame as inside. Several bodies of material touched constantly here are owned by others, and are named rather than re-taught.

- Every Mental Healthcare Act provision touching this treatment and psychosurgery, the bar on the unmodified procedure, the minor's guardian-consent and Board-permission gate, the psychosurgery restriction and the emergency-treatment bar, belongs to the Mental health legislation and forensic psychiatry notes, each named here in words.
- The catatonia entity, the lorazepam challenge, malignant catatonia against neuroleptic malignant syndrome and the Bush-Francis rating belong to the Other psychotic disorders, delusional syndromes and catatonia notes.
- The mood-disorder algorithms, the staging of treatment resistance and the pharmacology of continued agents belong to the Mood disorders: pharmacotherapy notes.
- The schizophrenia resistance algorithm, clozapine and the antipsychotic adverse-effect burden belong to the Schizophrenia notes, which assign the clozapine-resistant evidence here.

Many of this chapter's choices trade among speed of response, efficacy and cognitive cost, and where they do the direction and size of that trade differ by parameter and by placement, so each account states its own relation; some are not such trades at all. It sits inside an Indian legal frame in which only the modified procedure is lawful, treatment of a minor is gated behind guardian consent and prior Board permission, and the emergency-treatment provision cannot authorise the treatment.

Electroconvulsive therapy: foundations and indications

2 · History and evolution of ECT

Electroconvulsive therapy is one of the few treatments in current psychiatric practice that was invented out of a therapeutic premise which was not borne out, fell into disrepute it had partly earned, and came back on evidence gathered decades later. It is also the shortest route to understanding the modern procedure: the anaesthetic, the relaxant, the monitored seizure and the documented consent were each added to answer a specific failure of what came before. Names and dates carry marks in this paper, so they are given with their attributions.

This account owns the origin and evolution of convulsive therapy: the antagonism hypothesis, the chemical convulsants, Cerletti and Bini, the introduction of anaesthesia and muscle relaxation, the mid century decline, the modern reappraisal and the Indian arc. It does not teach why an induced seizure helps; the competing neurobiological theories, none of them settled, belong to this chapter's account of the mechanism of action and neurobiological theories of electroconvulsive therapy. It does not teach the technique contrast between modified and unmodified (direct) ECT, nor the Indian legal position on the unmodified form; both belong to this chapter's account of modified against unmodified (direct) ECT. It does not narrate the leucotomy story, which belongs to this chapter's account of psychosurgery, its history, current indications and ethics. Modern stimulus parameters and electrode placements appear nowhere in this account.

1934	1938	1943
THE FIRST DELIBERATE THERAPEUTIC CONVULSION IN A HUMAN BEING, PRODUCED CHEMICALLY	THE FIRST THERAPEUTIC SEIZURE INDUCED ELECTRICALLY, IN ROME	ELECTROCONVULSIVE THERAPY AT THE CENTRAL INSTITUTE OF PSYCHIATRY, RANCHI

2.1 The convulsion as a treatment, long before there was a device

Convulsive therapy is older than psychiatry's capacity to measure a convulsion, and a candidate who begins the story in the twentieth century has already misplaced it. Camphor given by mouth to induce convulsions and cure lunacy is attributed to the Swiss alchemist Paracelsus in the sixteenth century, and convulsions induced by chemical means, specifically camphor in oil, are documented in the eighteenth and nineteenth centuries. The idea that a seizure could relieve mental illness was inherited, not invented; what the twentieth century added was the deliberate

production of one, in a named patient, for a named condition, on a stated rationale. That rationale is where the modern history begins, and it is where it first goes wrong.

2.2 Meduna and the schizophrenia and epilepsy antagonism hypothesis

In 1934 Ladislaw Meduna, a Hungarian psychiatrist, investigated a hypothetical inverse relationship between seizures and schizophrenia, drawing on neuropathological studies and on a review of a century of earlier work, and postulated a relationship between a lack of glial cells in people with schizophrenia and an overgrowth of those cells in people with epilepsy. The forename is written Ladislaw because that is how the source quoted here spells it; two other peer reviewed histories write Ladislas von Meduna, which is the form to expect in an answer, and the difference is a transliteration rather than a different person. This is the **schizophrenia and epilepsy antagonism hypothesis**, the founding premise of the entire treatment family this chapter teaches.

It did not rest on neuropathology alone. A dedicated history of the treatment in schizophrenia attributes to Gyula Nyiro, in 1929, the clinical observation from which the antagonism hypothesis followed. Different histories state what Nyiro actually reported in different ways, so the observation is attributed here without asserting any one version of its content.

The therapeutic premise was not borne out, and the two things that happened to it should be kept apart. Meduna's original theory of an antagonism between epilepsy and schizophrenia has been replaced by hypotheses about the mechanism of action of the treatment. Replaced is what the source says, and it is not the same as disproved: the same paper adds that subsequent research could not confirm the antagonism hypothesis, which is a failure to confirm rather than a refutation, and a 2022 position still argues the biological question is open on shared genetic and network findings. So the premise stopped being the reason to give the treatment, while whether epilepsy and schizophrenia stand in any antagonistic relation at all remains unsettled. Those hypotheses, none presented as settled, belong to this chapter's account of the mechanism of action and neurobiological theories of electroconvulsive therapy.

RULE **The hypothesis that produced convulsive therapy was mistaken, and that does not make the treatment mistaken.** Whether a treatment works is settled by trials of it; why it works is a question about mechanism; and the accuracy of the guess that led somebody to try it bears on neither. Convulsive therapy is psychiatry's cleanest example of a wrong reason producing a right treatment, which is why the origin story is worth knowing rather than skipping.

2.3 The chemical era, and why it ended

Meduna moved from hypothesis to patient quickly. The eightieth anniversary review dates the first human experiment, involving intramuscular camphor injections, to 2 January 1934; other histories give the day differently, and one dates the first camphor injections to the November of the preceding year, so not even the year is undisputed and that winter, rather than a day or a year, is the honest unit. Attribute the date rather than asserting it, and note that any interval measured from the start of this work inherits the same disagreement. Camphor in oil was injected into a patient with catatonic schizophrenia, causing a 60 second grand mal seizure; the patient

recovered fully after a short series of such treatments, and five more patients were treated by the end of that year. Catatonia was therefore present at the origin of the treatment and remains among its strongest indications, taught in this chapter's account of the indications for electroconvulsive therapy.

Camphor was not the agent that carried the era. After about two months the intramuscular camphor injections were replaced by pentylenetetrazol, better known as **cardiazol** and marketed elsewhere as metrazol, which was less painful and had a lower latency of seizure induction. The chemical era ended for a reason a candidate should be able to state in one clause: the interval between the injection and the seizure remained unpredictable and frightening for the patient, and it is that latency which sent Cerletti looking for an electrical alternative. Electricity did not offer a better seizure. It offered a seizure whose timing the operator controlled.

2.4 Cerletti and Bini, Rome, 1938

Ugo Cerletti and Lucio Bini performed the first electrical induction of a seizure in a psychotic patient in Rome in April 1938, after animal studies in which they defined the parameters of an electrical stimulus they judged safe. The animal work is not decoration; it is the reason the pair could argue the stimulus was survivable before applying it to a person. One history dates the first stimulation to 11 April 1938, records that it did not produce an epileptic seizure, and dates a tonic clonic seizure at a higher voltage to 20 April. The year is agreed by every source opened for this chapter; the exact date is not, a useful marker that historical detail drifts where the substance does not.

The drift is visible in the patient too. A second independent history gives the same month and year and describes the patient as a 39 year old man treated at the Clinic for Mental and Nervous Diseases in Rome. The two accounts differ on how he came to the clinic. The discovery was reported publicly in August 1939, at the Third International Neurological Congress in Copenhagen, by Professor Lucio Bini, and that record is also the reason to write the forename as Lucio, since at least one contemporary peer reviewed source writes Luigi.

The stimulus of that first treatment is worth carrying for one reason only, which is contrast. It is recorded that no anaesthesia was used and that a first shock was delivered at 80 volts for one tenth of a second. Those are historical figures from the first treatment ever given, and they describe no device in use anywhere today. What happened after that first shock is deliberately not printed: the sources disagree not only on the DAY of these events but on their STRUCTURE, one account giving a failed attempt followed by a seizure some days later and another a single session of escalating shocks, and the number and escalation of the shocks lie in sentences no row here quotes. Both disagreements are declared as gaps rather than resolved into a sequence neither source describes.

TRAP **Quoting the figures of the first treatment as though they described a modern stimulus.** They record what was applied in Rome before anaesthesia, muscle relaxation, brief pulse waveforms, seizure monitoring or dosing against a measured threshold existed. Everything a candidate is examined on regarding the modern stimulus belongs to this chapter's accounts of electroconvulsive therapy technique and electrode placement and of stimulus dosing, the seizure threshold and adequate seizure criteria. A history figure written into a technique answer is a wrong answer, not an erudite one.

2.5 The modification: a relaxant first, then anaesthesia

What separates the treatment of 1938 from the treatment given today is not the electricity but what is given alongside it. Curare came first, introduced to stop the fracture and dislocation injuries of the unmodified convulsion, and muscle relaxation and anaesthesia were both in place by the early 1950s. Sources differ on the precise years, so the decade is the honest unit and a false precision is worse than none. The result is the treatment current Indian guidance defines: **modified ECT** is the modern form of electroconvulsive therapy in which the electrical stimulus is given under general anaesthesia and muscle relaxation.

This account stops there. How the modified and **unmodified (direct)** techniques differ in injury and in patient experience, and why the unmodified form was historically used, belong to this chapter's account of modified against unmodified (direct) ECT, as does the settled Indian position that the unmodified form is unlawful, its prohibition sitting in the Mental health legislation and forensic psychiatry notes' provision on treatment without anaesthesia and muscle relaxants. Here the unmodified technique is a historical stage and nothing else. The anaesthetic agents and the relaxant themselves belong to this chapter's account of the pre-ECT workup and anaesthesia.

AXIS	CHEMICAL CONVULSIVE THERAPY	THE FIRST ELECTRICAL ERA	MODIFIED ECT
BEGAN	1934	1938	the early 1950s
HOW THE CONVULSION WAS PRODUCED	Camphor by injection, then pentylenetetrazol	An electrical stimulus applied to the scalp	An electrical stimulus applied to the scalp
GIVEN ALONGSIDE	Nothing	No anaesthesia	General anaesthesia and muscle relaxation
WHAT FORCED THE NEXT STEP	An unpredictable and frightening latency to the seizure	Fracture and dislocation injuries of the convulsion	The current form
STATUS TODAY	Abandoned	Historical only, and unlawful in India	The standard technique

2.6 The mid century decline, and how much of it was earned

The decline is the part of this history a candidate is most often asked to explain, and the honest explanation has an earned half and an unearned half. The earned half is stated best by the American national consensus statement, in its own words. In the United States in the 1940s and 1950s the treatment was often given to the most severely disturbed patients in large mental institutions; as often occurs with new therapies it was used for a variety of disorders, frequently

in high doses and for long periods; many of those efforts proved ineffective and some even harmful; and its use as a means of managing unruly patients, for whom other treatments were not then available, contributed to the perception of an abusive instrument of behavioural control.

Some of the disrepute attaches to identifiable practices, and naming them is the honest way to teach this. In **regressive ECT**, sessions were performed several times a day until the deliberate onset of an acute brain syndrome, with memory loss, confusion, disorientation, apathy and dysarthria. That was the point of the technique, not its complication. It is abandoned, and it is the sharpest available contrast with a modern course, taught in this chapter's account of the course, frequency and number of sessions.

The unearned half is that the fall in use ran well past the abuses that provoked it. Three forces drove the decline together: effective psychopharmacological medication arriving from 1952, the development of judicial and regulatory restriction, and public protest. Use was widespread during the 1950s and 1960s, with approximately 300,000 people a year receiving the treatment in the United States, dwindled significantly during the 1970s and early 1980s under the ascendancy of pharmacological approaches and increased public protest, and recovered in the late 1980s and 1990s once it became apparent that medication was not a panacea. Those figures are United States figures and must be labelled as such; no Indian utilisation figure is carried in this account.

Attitude, not evidence, did much of the work. Negative media portrayal and the earlier misuse contributed to the negative professional and public perceptions that attitude surveys record repeatedly, and that attitude has played an important role in decreasing use of the treatment in the developed world and in reducing access to it, which constitutes a violation of psychiatric patients' right to an effective treatment. A candidate who can say that last clause out loud has understood why the history is taught at all.

2.7 The evidence led reappraisal, and why it is not finished

The turn back towards evidence has a datable institutional marker. Because research had intensified on effectiveness, indications, mechanism, adverse effects and optimum technique while remaining outside the mainstream of mental health research, the National Institutes of Health, together with the National Institute of Mental Health, convened a Consensus Development Conference on Electroconvulsive Therapy from 10 to 12 June 1985. That conference is where the treatment stopped being defended by assertion and started being assessed in public against data.

The evidence anchor came later. The 2003 systematic review and meta-analysis of electroconvulsive therapy in depressive disorders concluded that it is an effective short term treatment for depression, and probably more effective than drug therapy. This account carries the milestone only; the magnitudes and the cognitive costs belong to the items that own them.

The story has not closed. Despite that evidence base, global trends show a paradoxical decline in utilisation across the United States, Europe and Italy itself, where the procedure originated. The history therefore lands in the present rather than in an archive: the gap between what the

evidence supports and what services deliver is the live problem, which is why stigma belongs in a clinical chapter at all.

2.8 The Indian arc

India did not receive this treatment late, and an Indian candidate should be able to say so with a date. The Central Institute of Psychiatry, Ranchi, was one of the first centres outside Europe to start cardiazol induced seizure treatment, in 1938, and electroconvulsive therapy in 1943. Two things follow:

- cardiazol treatment began at Ranchi in the same year as the first electrical induction in Rome;
- India took up the chemical era and not only the electrical one, so the Indian story has the same phases as the international one.

A second institution corroborates the decade without fixing a year. An institutional history of the Government Mental Hospital, Madras, places the arrival of electroconvulsive therapy and of insulin coma therapy, including modified insulin coma therapy, in the years 1939 to 1948, and attributes the lag to the emergence of the Second World War, which held over changes that were long overdue. This is why the Ranchi record carries the date here and the Madras record the corroboration.

What followed was not passive adoption. Over the past five decades Indian research on electroconvulsive therapy has produced more than 250 publications, with a notable increase following the 1989 national workshop. The country has been a producer of the evidence base it applies, and the workshop is the turning point a candidate can name.

MNEMONIC

CAMPHOR, CARDIAZOL, CERLETTI, CURARE, CRITICISM, CONSENSUS

The arc in order. **Camphor**, the first deliberate therapeutic convulsion, on a mistaken premise. **Cardiazol**, whose unpredictable latency ended the chemical era. **Cerletti**, with Bini, the first electrical induction. **Curare**, the relaxant that began the modification anaesthesia completed. **Criticism**, the mid century decline. **Consensus**, the evidence led reappraisal that has still not restored utilisation.

Convulsive therapy began from a false premise about antagonism between epilepsy and schizophrenia, was produced first chemically by Meduna and then electrically by Cerletti and Bini in Rome, and became the modern procedure only when a muscle relaxant and then general anaesthesia were added by the early 1950s; it fell out of use for reasons partly earned by overuse and practices such as regressive ECT, partly imposed by new drugs, regulation, protest and stigma; and the reappraisal that followed left an evidence base strong enough to expose a decline in use that continues in spite of it. India entered the story at the beginning, at Ranchi, and has generated evidence about the treatment ever since.

3 · Mechanism of action and neurobiological theories of ECT

Electroconvulsive therapy is among the most effective treatments in psychiatry, and nobody has established how it works. That is the state of the evidence, not a gap in the resident's reading, and the field says so: a review of the neurobiology concludes that the cause and effect relationship between the findings and the therapeutic effects could not be established with absolute certainty, and it is not very prudent to assume a single mechanism that can explain the therapeutic effect. The examinable answer is therefore not a mechanism but a set of competing accounts, each with the observations behind it and the observation it cannot reach, none promoted to a fact.

Owned here in full: why the induced seizure helps, across the anticonvulsant and seizure-threshold, neuroendocrine and hypothalamic, monoamine and receptor-sensitivity, neurotrophic and neuroplastic, and network and connectivity accounts. Named and not re-derived, each with its own account in this chapter: the seizure threshold and how the stimulus is set against it; the placements; the cognitive adverse effects; the indications outside depression; and magnetic seizure induction. The division is strict: the seizure appears here only as a candidate therapeutic mechanism, never as a dosed event.

five	unclear	62 patients
COMPETING ACCOUNTS, OF WHICH THE NETWORK ACCOUNT IS THE NEWEST	THE EXACT NEUROBIOLOGICAL MECHANISMS, IN A REVIEW'S OWN WORD	IN THE TITRATION STUDY THAT SPLIT THE ANTICONVULSANT EFFECT FROM RESPONSE

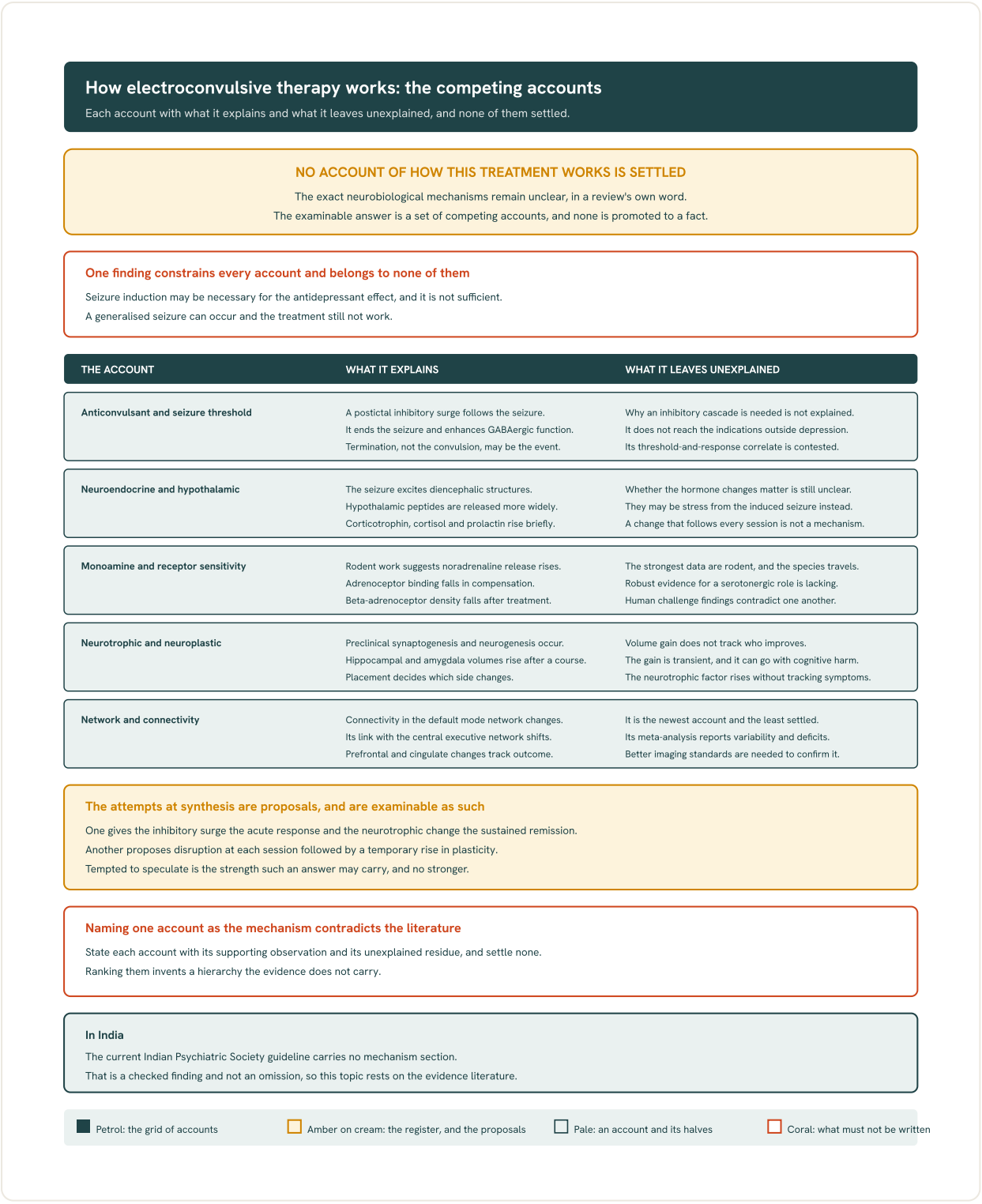


Fig 27.2. The competing neurobiological accounts of how electroconvulsive therapy works, each set beside the observation that supports it and the residue it leaves unexplained, with the finding that seizure induction may be necessary and is not sufficient constraining all of them, the attempts at synthesis stated as proposals, and no account marked as settled.

3.1 The register this topic is written in

The confidence available to every account is fixed first: the commonest error is not naming the wrong theory but naming a real one in the wrong voice. An independent review reaches the same place: the exact neurobiological mechanisms underlying the efficacy of the treatment remain unclear. A candidate who writes that the mechanism of action is the anticonvulsant hypothesis has contradicted the literature, whatever else the answer contains.

One structural finding constrains every account and belongs to none of them. Seizures induced by low-dose right unilateral stimulation lacked antidepressant efficacy, so seizure induction may be necessary but insufficient for antidepressant effects. A generalised seizure occurred and the treatment did not work. Any account that treats the bare occurrence of a seizure as the therapeutic event is under-determined, and the question becomes what kind of seizure, where it began and what followed it. The dose that makes right unilateral stimulation effective, and the placement comparison, belong to this chapter's accounts of stimulus dosing and of the placements.

RULE **State each account with its supporting observation and its unexplained residue, and settle none of them.** These accounts describe changes at different levels of organisation and on different timescales, and several may be describing parts of one process without any having been shown to cause the benefit. Ranking them invents a hierarchy the evidence does not carry.

IN INDIA

There is no Indian guideline position to quote here, and that is a checked finding, not an omission. The current Indian Psychiatric Society clinical practice guideline for electroconvulsive therapy declares its aim as enabling consistent, safe and effective practice in applicable psychiatric disorders; its full text carries no mechanism section and no neurobiological hypothesis, and every hit on the word anticonvulsant is pharmacological. Current Indian guidance controls Indian practice questions; on mechanism it has nothing to control, so this topic rests on the evidence literature.

3.2 The anticonvulsant hypothesis, stated as the hypothesis

The **anticonvulsant hypothesis** is a claim about therapeutic action and must be learnt in that form. It contends that the antidepressant effect is mediated by a postictal inhibitory surge in regions of the prefrontal cortex, which both terminates the immediate seizure and enhances GABAergic function. It derives from the GABA deficit model of affective disorders, so it is a hypothesis about inhibition rather than convulsion.

So read, it explains two things nothing else here explains as neatly: why the therapeutic event might be the **postictal inhibitory surge** and the seizure's termination rather than the convulsion, and why a stimulus initiating seizure activity in prefrontal regions might work when one initiating in motor cortex does not. The word throughout is might.

TRAP **Writing "the anticonvulsant hypothesis is that the rise in seizure threshold across a course correlates with clinical response" as the definition.** That states a contested empirical correlate and calls it a definition, in the one topic where nothing may be presented as settled. The hypothesis is the postictal inhibitory account above; the correlate is a separate observation, taught next, and disputed by the literature of the people who proposed it.

3.3 The contested correlate: does the rise in threshold track response

Across a course the seizure threshold tends to rise, though not in every patient, and the exception matters here because the argument below is built on the size of the rise. That rise, its magnitude

and its clinical consequences belong to this chapter's account of stimulus dosing, the seizure threshold and adequate seizure criteria, and are not restated. What belongs here is the mechanistic question laid on top of it: if the treatment works by being anticonvulsant, the size of the anticonvulsant effect ought to predict who improves. Two literatures answer differently.

AXIS	POSITION A: THE RISE TRACKS OUTCOME	POSITION B: IT DOES NOT
THE CLAIM	The magnitude of the change in seizure threshold over the course is associated with therapeutic outcome, particularly with right unilateral treatment, in the review that advanced the hypothesis	No relation between the rise in threshold and either therapeutic response status or speed of response, in a prospective titration study of 62 depressed patients
ITS AUTHORS' CAVEAT	Evidence associating the anticonvulsant and antidepressant effects has accrued, but critical experiments augmenting and blocking the anticonvulsant action in relation to efficacy should still be conducted	The same study confirms the anticonvulsant effect and concludes only that it is not tightly coupled to therapeutic efficacy
STANDING NOW	Subsequent investigations have not replicated key findings of the anticonvulsant hypothesis, and studies have failed to reproduce the correlation between seizure threshold increases and antidepressant response	

Position B is the more useful record, because it separates what a careless answer welds together: the anticonvulsant effect is real and was confirmed in the same study that refused the correlation, while the coupling of that effect to who improves is what was not found. Two further lines of negative evidence sit beside the failed replications, both at review level: spectroscopy studies of GABA concentration report either no change or increases unrelated to antidepressant outcome, and a study of plasma neuroactive steroids that modulate GABA receptor function found no change. That is what makes the correlate contested rather than merely old.

3.4 What the anticonvulsant account does not explain

Two conceptual limits come from the same review that recorded the failed replications. The account does not provide a rationale for why an inhibitory cascade should be necessary for antidepressant response, and it does not account for the treatment's efficacy in psychiatric disorders other than major depression. The second limb is load-bearing: the indications taught in this chapter's account of the indications and the evidence base include catatonia, mania and treatment-resistant schizophrenia, and a mechanism framed wholly as an antidepressant mechanism cannot carry them. Offering it as the mechanism offers a mechanism for one indication only, which is why the plural in theories is not a hedge.

3.5 The neuroendocrine and hypothalamic hypotheses

The **neuroendocrine hypothesis** is the oldest formal account here, best quoted in its originating form. Its authors postulated in 1980 that the antidepressant efficacy of convulsive therapy results

from the increased release and more widespread cerebral distribution of hypothalamic peptides with behavioural effects. Its base was the observation that neuroendocrine abnormalities characterise endogenous depression and are reversed by convulsive therapy.

What it observes is well established and frequently examined. The treatment has been hypothesised to enhance release of hormones from the hypothalamus into cerebrospinal fluid and blood through excitation of diencephalic structures, and there is a transient and selective increase in secretion of adrenocorticotrophic hormone, cortisol and prolactin that returns to baseline in a few hours. The prolactin surge is the item most residents are asked about, and it is a genuine consequence of the induced seizure.

PITFALL

Offering the prolactin surge as the mechanism. The source that records it declines to: whether these changes in hormone levels have therapeutic implications, or are manifestations of stress related to the induced seizure and secondary to other changes in the brain, is still unclear, and it is not known how changes in hormone levels affect the amelioration of symptoms. A change that follows every treatment is evidence that the treatment was delivered; it becomes a mechanism only when shown to differ with clinical outcome, and that step has not been taken.

3.6 The monoamine and receptor-sensitivity accounts

The **receptor-sensitivity account** reads the treatment as an antidepressant of a familiar kind, acting on monoamine transmission and receptor sensitivity. Its strongest data are preclinical, and the species travels with the claim. Results from studies on rodents suggest that the therapeutic effect may be mediated through an increased release of noradrenaline, indicated by a compensatory reduction in alpha-2 adrenoceptor binding, most marked in frontal cortex regions connected to the amygdala and hippocampus. The classical companion is a controlled rodent experiment in which electroconvulsive shock produced down-regulation of both the cyclic AMP response and beta-adrenoceptor density in rat cortex. Human data in the same literature are observations rather than mechanisms: reduced dopamine transporter binding is reported in major depressive disorder, and a treatment series elevates cerebrospinal fluid metabolites of monoamine neurotransmitters.

The serotonergic limb is where answers most often overreach, and the source is blunt. Robust evidence for the direct involvement of the serotonergic system in electroconvulsive therapy is lacking at this point of time, and further research in this domain might throw light on its implications. The same source calls the rodent receptor-sensitivity data ambiguous and the human challenge findings contradictory. No receptor-subtype direction or binding figure is carried, because none is grounded well enough to print.

3.7 The neurotrophic and neuroplastic account

This is the account a resident is most likely to state as fact, and the one whose caveats are most explicit. **The neurogenesis hypothesis** holds that the therapeutic effects depend on increasing the number of neurons or the connections among neurons, on a preclinical base:

- synaptogenesis markers;
- granule cell and mossy fibre sprouting;
- newborn cells in the dentate gyrus;
- growth factor signalling including brain-derived neurotrophic factor.

One observation inside it speaks to the seizure's standing as the therapeutic agent, and the review draws the inference itself: epileptic seizures induce similar neurogenesis effects in the absence of electrical stimulation, so if neurogenesis drives efficacy, this argues for the seizure, and not the electric field, driving efficacy.

The human test has been indirect, through blood **brain-derived neurotrophic factor**. A systematic review and meta-analysis of the preclinical and clinical literature concluded that electroconvulsive stimulation in rodents and the treatment in humans increase concentrations of this factor, but this is not consistently associated with changes in behaviour; in its human pooling, plasma rose and serum did not. A later review summarised two recent meta-analyses as finding that the factor increases after treatment, but with considerable heterogeneity and no clear relationship between change in the factor and change in depressive symptoms, and across treatment-resistant depression interventions peripheral levels increased overall with no association with depressive symptoms, making peripheral measurements of the total factor inadequate predictors of treatment response. The newest meta-analysis explains the disagreement rather than resolving it: the anaesthetic used and the time the blood sample was taken greatly influence measured levels, and the biological matrix and specific methodological variables affect the measurement, reinforcing the need to standardise future studies.

The structural limb carries the most orderly finding and the sharpest caveat. The largest neuroimaging dataset on this treatment showed a highly significant dose-dependent effect on hippocampal volume scaling with the number of sessions, with lead placement determining laterality: bilateral stimulation changed both hippocampi similarly, while right unilateral stimulation acted more focally on the right. A meta-analysis of limbic structures agrees: both right and left hippocampal and amygdala volumes increased after treatment, and meta-regression found age, the percentage responding and the percentage in remission negatively associated with volume increase in the left hippocampus.

Then the caveat that governs the limb. In the same dataset greater enlargement went with worse outcome, and that negative relation lost significance once session number was controlled for, because non-responders received more sessions. Either reading refuses the assumption the field had carried: this represents a major deviation from the common assumption of a positive association between treatment-induced volume enlargement and clinical improvement, and gross volume increase of the hippocampus by itself is not a meaningful biomarker for positive therapeutic response. The authors' own word for it is epiphenomenon, and the meta-analysis reaches the same caution: the treatment increased brain volume in the limbic structures, and the clinical relevance of that increase needs further investigation.

The change also does not last: follow-up imaging at six months showed hippocampal volumes had decreased back to pre-treatment levels, and the change was unrelated to antidepressant or cognitive outcomes. And it does not run in one clinical direction: two studies have shown that increases in hippocampal volume with treatment correlated with adverse cognitive effects, the mechanism-side counterpart of this chapter's account of the cognitive adverse effects. A change that is real, orderly, reversible, unrelated to benefit and sometimes associated with harm is a poor candidate for what makes the treatment work.

3.8 The network and connectivity accounts

The **network and connectivity account** is the newest and least settled, and its observations are functional rather than structural. Whole-brain analysis revealed significant effects on functional connectivity in various networks, including the default mode network, the central executive network, the sensorimotor network and the cerebellar posterior lobe, with connectivity increased within the default mode network and between it and the central executive network, aligning with previous reports that the treatment can normalise the connectivity between them. Its outcome linkage is regional rather than global: specific prefrontal and cingulate connectivity changes have been linked to therapeutic outcomes.

Its meta-analysis states its limits before its findings. There was considerable variability as well as a few key deficits in the preprocessing and analysis of the neuroimages and in the reporting of results across the included studies. What it reports is bounded in the same breath: the brain regions noted are reasonably specific and distinguished and had significant changes in resting-state functional connectivity after a course for depression, and more studies with better neuroimaging standards should be conducted to confirm these results.

One trial supplies the dissociation that earns the account its place. Right hippocampal connectivity increased, or normalised, after the series and correlated with depressive symptom reduction, while the hippocampal subfields were unchanged relative to the comparison group. Read the second half precisely, because it is not a claim that the volumes stayed still: the subfield volumes rose but did not separate the patients from the comparison group, so what failed was the discrimination and not the change. Function moved with the illness where structure, having moved, did not distinguish anyone. The conclusion those authors drew carries its later fate with it: they suggested that increased hippocampal functional connectivity and volumes may be biomarkers for response, and the volume part of that suggestion is what the larger dataset above did not support.

3.9 Holding the accounts together without settling them

The attempts at synthesis are proposals, and are examinable as such. Abbott and colleagues argue for integrating the anticonvulsant and neurogenesis hypotheses, and the shape of the integration is temporal: they contend that a frontal inhibitory surge and subsequent reductions in metabolism and perfusion are key to an acute response, but that neurotrophic changes in the medial temporal lobes are required for sustained remission. That gives each account a phase rather than a verdict.

A second model runs the same logic across the whole course. In response to the disruptive effects of each session, the brain exhibits a temporary enhancement of neuroplasticity, leading to increased brain tissue volume and enhanced connectivity, and the combination of disruption and neuroplastic effects leads to a rewiring of neural circuits associated with depressive symptoms. Its own review reports it as a proposed model, and it is written here in that voice; its corollary about electric field dosing belongs to this chapter's accounts of stimulus dosing and of the placements.

The register the field chose for its own synthesis is worth copying. Integrating the evidence, one review writes that it is tempted to speculate that the treatment may have neuroplastic effects resulting in the modulation of connectivity between and among specific large-scale networks that are altered in depression. Tempted to speculate is the strength an answer may carry, and no stronger.

MNEMONIC

A NEW MAN NEEDS NETWORKS

Anticonvulsant, **NEW**roendocrine, **MAN**oamine, **NEE**urotrophic, **NETWORK**. A sound-alike, not an initialism; the order is the order in which they are taught here, not of date and not of merit. Note in particular that the anticonvulsant account heads the mnemonic while the neuroendocrine hypothesis is the oldest formal account carried on this page, so the sequence is not chronological.

No account of how electroconvulsive therapy works is established, and the field says so in its own words; the seizure may be necessary and is not sufficient; the anticonvulsant hypothesis is the postictal inhibitory account, not the disputed threshold-and-response correlate; the neuroendocrine rise, the rodent receptor changes, the neurotrophic factor and the hippocampal volume gain are real observations not shown to track benefit; and connectivity, the newest account, states its own methodological limits beside its findings, so the strongest register available is that of its reviewers, who were tempted to speculate.

4 · Indications for ECT (depression, mania, catatonia, schizophrenia, emergencies)

An indication for electroconvulsive therapy settles one question only, whether this presentation earns the treatment, and never the second, whether there is a lawful route to give it. The set is short and stable in outline across the major guidelines: severe depression, particularly with psychotic features, food refusal, stupor or acute suicidal risk; catatonia; acute and refractory mania; schizophrenia, where the sources openly disagree; and the psychiatric emergency, where urgency is highest and the statutory position most easily misread.

Owned here in full: why each indication earns ECT, its evidence, and the distinction between a clinical emergency and a statutory route. Named and not re-derived: the risk profile and the contested status of absolute contraindication; the acute course and its schedules; continuation and maintenance treatment; consent as it is taken in Indian practice, where the lawful routes are enumerated; and the special populations. The catatonia entity, its signs and aetiology, the lorazepam challenge, the malignant catatonia against neuroleptic malignant syndrome

differential and the structured rating scale belong to the Other psychotic disorders, delusional syndromes and catatonia notes. The mood ladders are the Mood disorders: pharmacotherapy notes'; the resistance algorithm and clozapine itself the Schizophrenia: management, antipsychotics and adverse effects notes'.

0.91 EFFECT SIZE FAVOURING REAL OVER SIMULATED ECT IN DEPRESSION	**80 to 100%** RESPONSE ACROSS THE CATATONIA SERIES, THE STRONGEST INDICATION HERE	**Grade B** THE BEST GRADE ANY CLOZAPINE AUGMENTATION STRATEGY REACHES

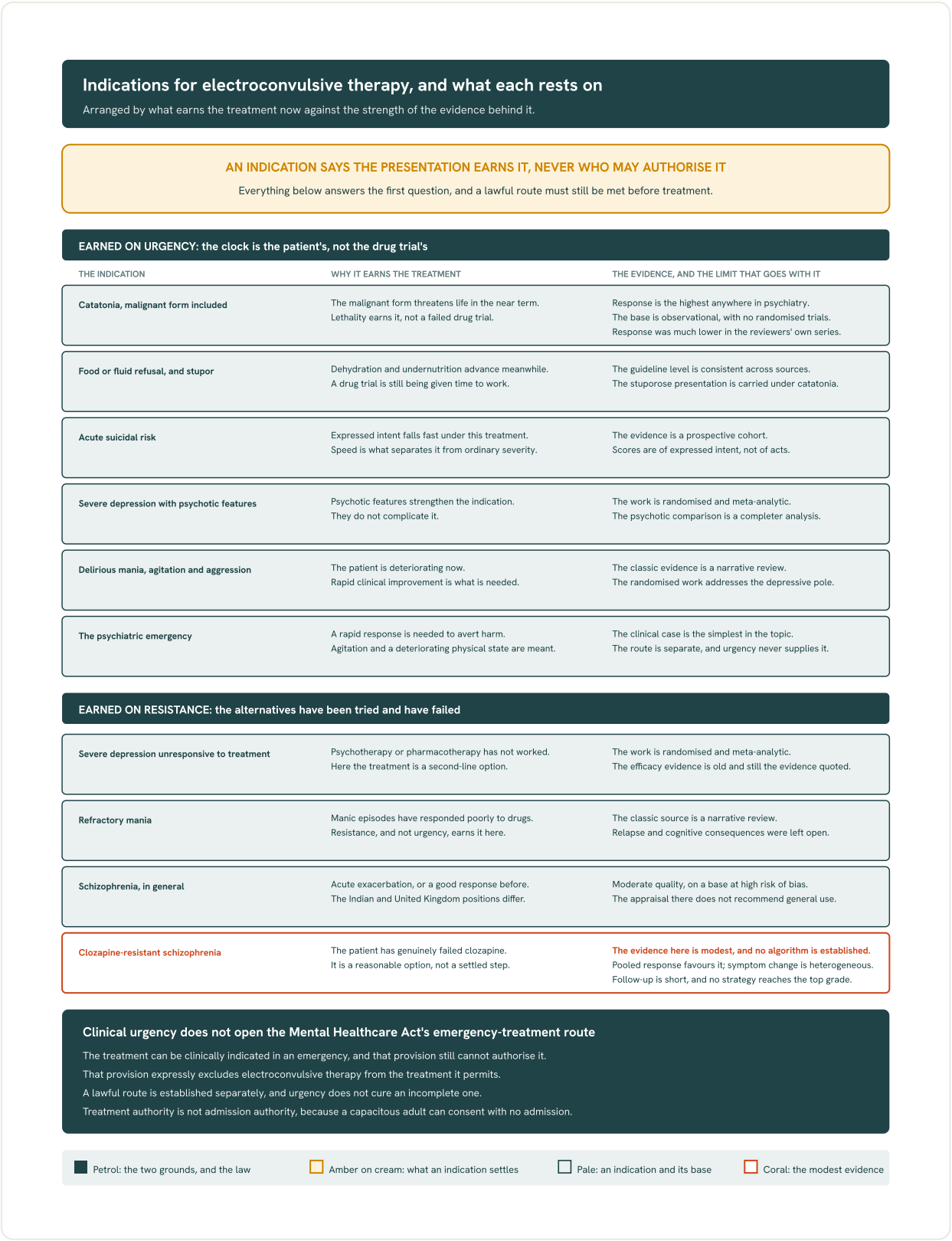


Fig 27.3. The indications for electroconvulsive therapy set out by what earns the treatment now against the strength of the evidence behind each: catatonia, food or fluid refusal, acute suicidal risk, psychotic depression, delirious mania and the psychiatric emergency on urgency; unresponsive depression, refractory mania, schizophrenia and the clozapine-resistant patient on resistance, that last one modest and with no established algorithm; and, along the foot, the statutory point that clinical urgency does not open the emergency-treatment route.

4.1 What an indication settles, and what it leaves open

An **indication** is a clinical judgment that a presentation is of the kind ECT treats and that benefit against risk favours giving it. It is not an authority to treat. Everything below answers the

first question; the second belongs to this chapter's account of consent, and the statutory provisions it turns on are named in words here and numbered only in the Mental health legislation and forensic psychiatry notes.

Weight the guideline before leaning on it. The current Indian Psychiatric Society guideline controls Indian practice questions, and records its own method: it was prepared by a consensus-based recommendation method, no formal systematic literature search was conducted, and it states in terms that it is a guidance document for practitioners rather than a directive or mandatory instruction. Where an effect size is at stake the number therefore comes from the trial, and the guideline carries the practice position.

RULE **An indication says the presentation earns ECT; it never says who may authorise it.** Each must still meet a lawful route before a treatment is given, and a strong answer to one question is no answer to the other.

4.2 Severe depression: food refusal, stupor and acute suicidal risk

Depression carries the strongest evidence, and within it one group of features moves ECT from a later option to a first one. A peer-reviewed review of the guideline position states it in a sentence: most guidelines recommend ECT as the first-line treatment for a severe depressive episode with psychotic features, catatonia, high suicide risk, or food or fluid refusal, and as a second-line treatment for a severe episode unresponsive to psychotherapeutic or pharmacological intervention. **Food refusal** earns its place because it puts a physiological clock on the decision: dehydration and undernutrition advance while a drug trial is still being given time to work. The Indian guideline's wording for it is poor oral intake, and its Table 1 depression rows read poor oral intake, high suicidal risk, a high level of distress requiring rapid symptom remission, psychotic features, melancholic features, peripartum depression, treatment resistance, and severe mixed affective features.

Depressive **stupor** needs care, because no source in this fragment's evidence list uses that term: both grounded statements carry it under catatonia, where a stuporose depressive presentation sits nosologically. It is written here as the catatonic pole of a severe depressive episode, immobility with failing intake being an indication on two grounds at once.

The efficacy evidence is old and still the evidence quoted, from the United Kingdom ECT Review Group's meta-analysis. Real ECT beat simulated ECT, 6 trials and 256 patients, standardised effect size minus 0.91 with a 95% confidence interval of minus 1.27 to minus 0.54. ECT beat pharmacotherapy, 18 trials and 1144 participants, standardised effect size minus 0.80 with a 95% confidence interval of minus 1.29 to minus 0.29. The negative sign is the direction of benefit, the effect being measured on depression scores.

Psychotic features strengthen the indication rather than complicate it, and that is measured. The collaborative remission study reported on 253 patients: remission among completers **87%** overall, **95%** in psychotic against **83%** in non-psychotic depression, with improvement more robust and appearing sooner in the psychotic patients. Those are completer rates, which the source says of itself.

Acute suicidal risk earns ECT on speed, which is what distinguishes it from ordinary severity. Expressed suicidal intent was scored before every session in 444 patients with unipolar depression: 131 patients, 29.5%, reported suicidal thoughts and acts at baseline, and the score fell to 0 after 1 week, or three treatments, in 38.2% of them, after 2 weeks, or six treatments, in 61.1%, and by the end of the course in 80.9%. The authors' conclusion is examinable: for a patient at risk, ECT should be considered earlier than at its conventional last-resort position.

TRAP **Quoting the NICE technology appraisal as current guidance for ECT in depression.** Its recommendations on depression were removed from that document and replaced by the depression guideline published in 2009, while its recommendations on catatonia, prolonged or severe manic episodes and schizophrenia did not change. It remains quotable for those and never for depression.

4.3 Catatonia: the strongest indication, on the weakest design

Catatonia earns ECT on two grounds worth keeping apart: response, better here than anywhere else in psychiatry, and mortality, since in the malignant form it is the near-term lethality of the presentation, not the failure of a drug trial, that earns the treatment. The guideline's catatonia rows carry that split, listing catatonia resistant to a benzodiazepine trial, a good response in past episodes, and malignant catatonia with a risk of imminent mortality.

A systematic review of ECT in catatonic patients reports **response rates ranging from 80% to 100%** across the retrospective observational studies, effective in all forms of catatonia including after benzodiazepines have failed, and above any other treatment in psychiatry. Inside the same review, a record review of 178 patients across 270 episodes used ECT alone in 55 patients, about 30%, resolving catatonic symptoms in 85%; where malignant catatonia was suspected, response was 89%, 9 of 11.

The counterweight comes from the same review. In the reviewers' own series ECT improved 59% of cases although pharmacotherapy had failed in 85% of the patients, attributed to a high prevalence of psychotic disorders, to ECT being reached only after two months of pharmacotherapy, to prior antipsychotic exposure, and to neurological comorbidity in one third. Both halves belong: the indication is the strongest on the list, and its design limit is the limit of its whole evidence base, these being observational series rather than randomised trials.

The boundary is exact in both directions. The entity, the lorazepam challenge, the differential against neuroleptic malignant syndrome and the structured rating scale are named here and taught in the Other psychotic disorders, delusional syndromes and catatonia notes. The technique stays on this chapter, split three ways: why the presentation earns ECT, which is this account; the stimulus dose, which is its dosing account; and the schedule, including the accelerated daily or initially twice-daily one used in life-threatening presentations, which is its account of the acute course. The assembled protocol for malignant catatonia has exactly one owner there, and no second copy is built here.

4.4 Mania, acute and refractory

The manic indication is not only about resistance. The Indian guideline's mania rows are a need for rapid clinical improvement, persisting or clinically significant agitation and aggression, treatment-resistant mania, severe mixed affective symptoms, and delirious mania. Some are urgency claims and some resistance claims, earning the treatment differently: agitation and delirious mania because the patient is deteriorating now, resistance because the alternatives have failed.

The classic evidence is a review of published experience over fifty years, which concluded that ECT is associated with remission or marked clinical improvement in 80% of manic patients and is effective for patients whose manic episodes have responded poorly to pharmacotherapy. Cite it for what it established, not as current practice: it is a narrative review rather than a meta-analysis, more than thirty years old, and its authors left relapse, cognitive consequences and the choice of placement for further study. Its observations on the seizure threshold in mania and on course length belong to this chapter's dosing and schedule accounts.

The modern randomised counterpart addresses the depressive pole, which is the trap here. A systematic review with exploratory meta-analysis of randomised trials in treatment-resistant bipolar disorder found ECT similarly effective for treatment-resistant bipolar depression as for treatment-resistant unipolar depression, odds ratio 0.919 with a 95% confidence interval of 0.44 to 1.917. It supports the refractory affective indication and says nothing about mania. The United Kingdom appraisal reaches mania from the other side, its surviving recommendation 1.1 permitting ECT only to achieve rapid and short-term improvement of severe symptoms after an adequate trial of other treatment options has proven ineffective, or where the condition is considered potentially life-threatening, in catatonia and in a prolonged or severe manic episode.

4.5 Schizophrenia, and the clozapine-resistant patient

Here two authoritative sources say different things, and the skill is to state both with their provenance rather than pick.

QUESTION	INDIAN PSYCHIATRIC SOCIETY GUIDELINE (2023)	NICE TECHNOLOGY APPRAISAL 59 (2003, UPDATED 2009)
SCHIZOPHRENIA IN GENERAL	Listed: acute psychosis requiring high-intensity management; acute exacerbations of positive psychotic or affective symptoms; good response in past exacerbations; postpartum psychosis	Recommendation 1.9: the current state of the evidence does not allow the general use of ECT in the management of schizophrenia to be recommended
RESISTANT AND CLOZAPINE-RESISTANT	Listed as treatment-resistant schizophrenia and as clozapine augmentation in clozapine-resistant schizophrenia	Not addressed by its terms; the statement is about general use

The randomised evidence for treatment-resistant schizophrenia is thinner than either position implies. The Cochrane review pooled 15 studies and 1285 participants and rated moderate-quality evidence that added ECT improves medium-term clinical response against standard care

alone, risk ratio 2.06 with a 95% confidence interval of 1.75 to 2.42, in 819 participants across 9 studies, with no clear advantage on other outcomes and 14 of 15 studies, 93.3%, at high risk of bias. Against clozapine added to standard care, the risk ratio for clinically important response was 1.23 with a 95% confidence interval of 0.95 to 1.58, from 162 participants in a single low-quality study, no clear difference either way.

For the clozapine-resistant patient the pooled result is favourable and the appraisals of it are not. The largest meta-analysis found adjunctive ECT superior on study-defined response at the post-ECT assessment, 53.6% against 25.4%, risk ratio 1.94 with a 95% confidence interval of 1.59 to 2.36 and a number needed to treat of 3, while remission at that assessment was 13.3% against 3.7%, a number needed to treat of 13 with a 95% confidence interval of 6 to 100. Response is common, remission is not, and the remission interval is too wide to carry precision. Heterogeneity was high: I-squared of 86% for symptomatic improvement at the post-ECT assessment and 95% at endpoint.

The best-known randomised trial is small and single-blind. In an intent-to-treat sample of 39 participants, 20 given ECT plus clozapine and 19 clozapine alone, 50% of the ECT plus clozapine patients met the response criterion and none of the clozapine group did; response in the crossover phase was 47%. Its own conclusion anchors what this chapter must say: further research is required to determine the persistence of the improvement and the potential need for maintenance treatments.

Three appraisals keep that at its true strength.

- A synthesis of six systematic reviews concluded that ECT probably augments response to clozapine, but that whether it leads to cognitive adverse effects cannot be determined because the certainty of the evidence is very low.
- A review of 46 studies of 25 augmentation strategies called ECT highly promising while adding that its conclusions are tempered by generally short follow-up periods and poor study quality.
- A meta-review of 21 meta-analyses found that no strategy met Grade A criteria and that augmentation with ECT for persistent positive symptoms met Grade B.

RULE **State the clozapine-resistant indication as modest, and say that no algorithm is established.** The effect sizes are real and the certainty behind them is not: heterogeneity is high, the trial base small and mostly single-blind, follow-up short, no strategy at the top grade. Offer it as a reasonable option in a patient who has genuinely failed clozapine, never as a step on a settled ladder.

4.6 The psychiatric emergency, and the authority it does not confer

The clinical half is the simplest statement in the topic. ECT is a first-line treatment when a rapid or definitive response is needed to avert harm to self or others, and acute suicidal risk, agitation, catatonia and a deteriorating physical status secondary to a psychiatric condition are among the situations meant. The list is examples rather than a closed set, and the guideline adds that every

indication is individualised against clinical need, the patient's preferences and the putative risk of adverse effects.

The statutory half is where a resident is most likely to be taught something unlawful, and the error is precise enough to name. ECT can be clinically indicated in an emergency, and the Mental Healthcare Act's provision on emergency treatment cannot authorise it, because that provision expressly excludes electroconvulsive therapy from the treatment it permits. Both statements are true and not in conflict. What follows is that a lawful route must be established separately, and that urgency does not cure an incomplete route. No emergency doctrine supplies the authority the Act withholds.

The second half is the one most often lost: TREATMENT authority is not ADMISSION authority. A capacitous adult can consent to ECT with no admission at all, so urgency never converts the question into whether the patient must first be admitted. The routes, and what each does and does not authorise, are enumerated in this chapter's account of consent for ECT in Indian practice. Every provision of the Act touching ECT and psychosurgery, including those on emergency treatment, the nominated representative and the statutory gate for a minor, belongs to the Mental health legislation and forensic psychiatry notes: named in words here, numbered only there.

TRAP **Writing emergencies into the indication list and stopping there.** A list that says ECT is indicated in an emergency, with nothing beside it, teaches a resident to reach for the Act's emergency-treatment provision and treat under it, which is not lawful. The paired error runs the other way: assuming that because that route is closed, an admission must be arranged first. It need not be, if the adult before you has capacity and consents.

MNEMONIC

NOT EATING, NOT MOVING, NOT SAFE, NOT WORKING

Not eating is food or fluid refusal and the clock it starts. **Not moving** is catatonia, the malignant form earning the treatment on mortality. **Not safe** is acute suicidal risk, where speed of relief is the point. **Not working** is treatment resistance across depression, mania and schizophrenia. A fifth line belongs with the four: none is a lawful route, and each still needs one.

4.7 Each indication at its true strength

INDICATION	THE EVIDENCE, AND THE LIMIT THAT GOES WITH IT
SEVERE DEPRESSION WITH PSYCHOTIC FEATURES	Randomised and meta-analytic; the psychotic comparison is a completer analysis
FOOD OR FLUID REFUSAL, AND THE STUPOROSE PRESENTATION	Guideline-level and consistent; carried nosologically under catatonia
ACUTE SUICIDAL RISK	Prospective cohort; scores are of expressed intent, not of acts
CATATONIA, MALIGNANT FORM INCLUDED	Very high response but entirely observational, and much lower in the reviewers' own series, whose size is not stated
ACUTE AND REFRACTORY MANIA	A narrative review; the randomised work addresses the depressive pole
SCHIZOPHRENIA, GENERAL	Two authoritative sources disagree; moderate quality on a base at high risk of bias
CLOZAPINE-RESISTANT SCHIZOPHRENIA	Modest, no algorithm: pooled response and remission favour it with no heterogeneity, the heterogeneity belongs to the continuous symptom-change outcome, and follow-up is short
THE PSYCHIATRIC EMERGENCY	Clinically clear; the statutory route is separate and never supplied by urgency

PITFALL

Answering with one undifferentiated list. These indications do not stand on evidence of one kind: catatonia on observational series, depression on randomised and meta-analytic work, mania on a narrative review, the clozapine-resistant patient on a small and heterogeneous trial base. An answer that grades them reads as clinical judgment; one that recites them cannot say which would survive a challenge.

ECT is indicated where the presentation is of the kind it treats and benefit against risk favours it: severe depression, most strongly with psychotic features, food refusal or acute suicidal risk; catatonia, on the strongest response evidence in psychiatry and the weakest design; acute and refractory mania; schizophrenia, where the Indian and United Kingdom positions differ and both are stated; the clozapine-resistant patient, modest with no established algorithm; and the psychiatric emergency, where the clinical case is strongest and the Act's emergency-treatment provision still cannot authorise treatment.

5 · Contraindications and high-risk conditions for ECT

A contraindication is a statement that a treatment must not be given at all, and the difficulty here is that the field does not agree on whether electroconvulsive therapy has one. Two positions are in print, each from a named source with a date, and a candidate who has met only

one will be wrong in one of two directions: barring a patient who could have been treated, or taking a patient with an intracranial mass and papilloedema to the suite. This account states both, gives the physiology behind the older one, and teaches each condition that raises risk beforehand with its modification.

Owned here is the risk profile before treatment: the contested status of absolute contraindication, the intracranial question the dispute turns on, and the high-risk conditions. Named and not re-taught, each owned by another account in this chapter: the complications that actually occur, by the other complications and adverse effects of electroconvulsive therapy; the workup and anaesthetic conduct that manage these risks, by the pre-ECT workup and anaesthesia, its agents, muscle relaxant and airway; the clinical grounds, by the indications for ECT; and pregnancy, age and comorbidity as populations, by ECT in special populations. Risk beforehand against complication afterwards: two objects, not one.

1980	74 percent	one patient
THE YEAR POSITION TWO WAS CONCLUDED, AND STILL A COMMON EXAM KEY	COMPLICATIONS IN THE REPORTS OF ECT WITH AN INTRACRANIAL NEOPLASM BEFORE 1984	THE WHOLE DIRECT EVIDENCE ON ECT WITH RAISED INTRACRANIAL PRESSURE ALREADY PRESENT

5.1 Two positions, with their provenance and their dates

Both are written out, because the disagreement is real and this account does not settle it.

Position one is the modern task-force position: there are no absolute contraindications to electroconvulsive therapy, and every listed condition is a risk to be quantified and managed with the anaesthetist. It reaches this page through a peer-reviewed anaesthesia review of 2022, which attributes it to the American Psychiatric Association consensus statement of 2001. The two halves must not be split: in the very next breath the same passage names uncontrolled hypertension, coronary artery disease, congestive heart failure, aortic stenosis, implanted cardiac devices, atrial fibrillation, obstructive lung disease, asthma and increased intracranial pressure with or without mass lesions as conditions carrying a relatively high risk that may result in death during treatment. Position one is not a claim that nothing is dangerous; it is a claim about where the danger is adjudicated.

The position holds at the current end. A review of electroconvulsive therapy in intracranial pathology, published in 2025, concludes that no absolute contraindications have been established, frames the transient rise in intracranial pressure as the reason the question keeps being asked, and calls its own evidence base very sparse, mostly case series and short retrospective studies. It also states what modern practice is: a decision made case by case by a team involving both the psychiatric and the neurosurgical services.

Position two is that raised intracranial pressure with a space-occupying lesion is an absolute contraindication. It is standard in Indian teaching and common as an examination key, and it has a provenance: it is the conclusion of the last large review before the modern case literature, by Maltbie and colleagues in 1980, recorded as such by the 2020 review that set out to test it.

A limitation is shared, and neither side is graded above the other on it: neither the task-force text of 2001 nor the review of 1980 was opened at source. What is established is what a later peer-

reviewed paper reports each to hold.

RULE **Write both positions with their dates, and never present "no absolute contraindication" as settled and uncontested.** The modern position is a claim about method, that risk is quantified and managed rather than made prohibitive in advance. A reader given it as a licence, without what the older position was protecting against, has been taught something unsafe.

5.2 The physiology that makes position two reasonable

Position two is not superstition, and its mechanism is examinable. Electroconvulsive therapy induces a rise in cerebral blood flow, in intracranial pressure, in intraocular pressure and in cerebral oxygen consumption. Where compliance is normal those rises are transient and settle. The whole of position two is what the same rises mean in a patient whose compliance has already been consumed by a mass.

The chain from the general rise to the specific danger runs in a fixed order, first proposed by Carter in 1977. Seizure activity raises blood pressure and cerebral blood flow; that increases the oedema around the tumour; the oedema raises intracranial pressure further; neurological signs follow. The 2020 review treats this as the likely cause of the more prominent adverse events in its own series, and records that a corticosteroid was given to 12.5 percent of its patients to reduce the risk of acute oedema, saying only that it is therefore possible such premedication lowers the risk. No agent dose belongs here: drug conduct is the workup and anaesthesia item's.

Two honesties travel with this mechanism. No magnitude is available: no grounded source states how large the rise in intracranial pressure is, and the review describing the rises says the mechanisms behind them are incompletely understood, so direction and transience are taught and size is not carried. And the word herniation appears in none of the sources this account rests on. The grounded chain ends at neurological signs; herniation is the endpoint the older teaching was written to prevent, and naming it as the feared outcome is legitimate while asserting it as a documented consequence is not. That gap is declared, not closed. The same sentence grounds the ocular limb, which is why raised intraocular pressure and retinal detachment sit in the set below.

5.3 The outcome data each position was reading

Position two was a reading of outcomes, not an opinion. In reports published up to 1984 on treatment in patients with intracranial neoplasms, complications were recorded in **74 percent** of 38 patients and death in **8 percent** of 29 patients. The denominators differ, so these are not two proportions of one sample. The source supplies the confound itself: some who died had aggressive tumours such as glioblastoma, so attribution to the treatment is not clean. The figures reach this page through a safety review of 2025 reporting an earlier review, which bounds them without doubting them.

The modern series behind position one is small. Across reports from 1984 to 2019 there are 40 patients. Adverse effects occurred in **15 percent** and all were reversible; none died from treatment; the authors judged it beneficial in 36 of the 40 patients, which is 90 percent. The decisive sentence sits in that paper: only one patient was reported to have had raised intracranial

pressure before the procedure, and came through without adverse effect. That is the closest the literature comes to answering this item's question, and it is one patient.

TRAP **Reading the difference between the two eras as pure progress in technique.** The comparison is not like for like and the modern authors say so. The tumour was known beforehand in 72.5 percent of modern cases, allowing precautions, and neurological symptoms preceded treatment in 12.5 percent of them against 45.7 percent of the older group, which held more aggressive tumours. The modern series is safer partly because it is a different population.

5.4 Where Indian practice actually sits

The current Indian guideline, of 2023, gives its high-risk conditions in a table headed *clinical conditions requiring caution while administering ECT*, grouped into four systems, and its running text introduces the physical examination as the way to identify any **relative contraindications**. Raised intracerebral pressure with an intracerebral space-occupying lesion heads the cerebral group. The Indian guideline therefore sits with position one, and that framing is itself the finding.

Its operative instruction is sharper than either slogan. For a symptomatic intracranial mass, and for recent ischaemic or haemorrhagic stroke, it says treatment may warrant postponement until the risks of complications are minimised: a relative bar with a condition attached. The same row states that cerebral implants and foreign bodies are not an absolute contraindication and that treatment may be given in consultation with neurologists. Its electrode-placement instructions belong to this chapter's account of ECT technique and electrode placement, its relaxant instruction to the workup and anaesthesia item.

IN INDIA

Two Indian facts point in opposite directions and both are true. Indian *teaching* writes raised intracranial pressure with a space-occupying lesion as an absolute contraindication; the current Indian *guideline* does not. Give the guideline position, and name the taught position with its date.

5.5 How the intracranial condition is actually found

The condition this item turns on is usually not looked for in advance. Routine neuroimaging before a course is not mandatory and its yield is very low: one retrospective study found a single abnormal scan preventing treatment out of 108 scans done as routine pre-treatment imaging, which is 0.93 percent. Selective imaging is suggested for patients at risk: a prior aneurysm, a heritable aneurysm disorder, headache, and age above 60 with hypertension or smoking. What triggers imaging afterwards is any new neurological symptom: significant lateralisation of the seizure, a transient postictal paralysis, aphasia or a secondary seizure.

What is mandatory is the bedside examination. The Indian guideline of 2023 makes fundoscopy a mandatory part of the pre-treatment physical examination. That is the practical link: **papilloedema** is what raised intracranial pressure looks like at the bedside, and the examination exists, in the guideline's words, to identify relative contraindications and prevent complications.

5.6 The high-risk conditions, and the modification that manages each

The set below is explicitly open: the safety review supplying the last row introduces its own list with the word *include*, marking it as not closed. Neither list is the answer to what the high-risk conditions are.

SYSTEM GROUP	CONDITIONS NAMED	MODIFICATION THAT MANAGES IT
CEREBRAL	Raised intracerebral pressure with an intracerebral space-occupying lesion; unstable cerebral aneurysm; recent intracerebral haemorrhage or stroke; and phaeochromocytoma, which sits in this row only because the source table's column layout puts it here, its hazard being a catecholamine-driven hypertensive surge rather than anything cerebral	Postponement of a symptomatic mass and of recent stroke until risks are minimised; shunt patency confirmed; blood pressure held with short-acting parenteral antihypertensives in aneurysm
OCULAR	Raised intraocular pressure; retinal detachment	The induced rise in intraocular pressure is the mechanism, transient in the ordinary case
CARDIAC	Recent myocardial infarction; poor cardiac output; unstable arrhythmias; high anaesthetic risk (ASA level 4 or 5); third degree burns	Wait 4 to 6 weeks after infarction; blood pressure controlled with short-acting agents; anticoagulation considered in valvular disease; restimulations limited in heart failure, with functional capacity assessed; pacemakers safe with cardiology consultation
NEUROMUSCULAR	Spinal injuries; bone fractures; recurrent dislocations	Managed through the relaxant and the technique, both owned by the workup and anaesthesia item
NAMED BY THE MODERN REVIEW	Recent myocardial infarction; intracranial aneurysm above a threshold size; proximal deep vein thrombosis; phaeochromocytoma; systemic infection	Avoided or given with high caution on a favourable risk-benefit ratio; deep vein thrombosis singled out for extreme precaution, fatalities being associated with it

Three entries need their detail, because each is where a memorised line goes wrong. The **infarction window** is the plainest conflict here after the intracranial one: the Indian guideline of 2023 says consider waiting 4 to 6 weeks; the safety review of 2025 writes it as an infarction within the last 4 weeks. The disagreement is stated, not resolved.

The **aneurysm** is not theoretical. Across 27 published cases, treatment was safe with secured and unsecured aneurysms up to 2.1 cm, with one fatal exception: a patient died of subarachnoid haemorrhage after rupture of an anterior cerebral artery aneurysm of 1.9 cm, 2 days after a tenth right unilateral treatment, the peri-ictal pressure having risen sharply despite a short-acting agent. The review states that treatment is not universally safe in aneurysm and that risk rises

with size. Note the consequence for any threshold: the paper names aneurysms larger than 1 cm as high risk, yet the fatal one was larger and safe cases are reported above it. Size is a gradient, not a cut-off.

The **mass** is not one thing. A mild mass effect does not appear to preclude treatment provided there is no midline shift, and the largest tumour reported without adverse effect measured 8.1 by 7.6 by 4.1 cm. That is what makes position one operable at the bedside. It does not retire position two, and this account must not be read as doing so: position two's claim is that the PRESENCE of an intracranial mass is an absolute bar, and recasting that claim as really being about midline shift would resolve the chapter's flagship conflict in the permissive direction on evidence that is a series of case reports. No source here defines when a mass effect stops being mild, none quantifies a midline shift, and no such threshold is asserted, so the discriminator is not available as a bedside rule.

PITFALL

Printing any of these lists as the complete set. The task-force passage quoted earlier for position one adds conditions the Indian table does not carry as such, so severe pulmonary compromise, decompensated heart failure and severe valvular disease belong on any honest version. A vascular malformation liable to rupture with a pressure surge is often taught here too; no source grounded for this account names it, so it is flagged rather than printed as established.

5.7 Pregnancy: on the risk list, not the contraindication list

Pregnancy is not a contraindication to electroconvulsive therapy, and the distinction this item exists to draw appears here in its cleanest form. Two instructions travel with that: pregnancy testing in all women of reproductive age, and fetal exposure to anaesthetics minimised. The risk side is stated alongside, not instead: guidance holds that risks may be enhanced during pregnancy, in older people and in children and young people, and fatalities including fetal deaths have been reported after treatment in the presence of pregnancy among other conditions, with the caveat that attribution is not established in every case. How treatment is then modified belongs to this chapter's account of ECT in special populations, and the statutory position on minors to its account of consent for ECT and the legal aspects under the Mental Healthcare Act.

5.8 What stands in place of a contraindication list

Guidance from the appraisal tradition does not answer this item with a list at all. It requires a documented assessment of the risks and potential benefits to the individual, and names what that assessment must weigh:

- the risks associated with the anaesthetic;
- current comorbidities;
- anticipated adverse events, particularly cognitive impairment;
- the risks of not having the treatment.

It gives a procedure, not a set of barred conditions, which is itself evidence about where the guideline tradition sits.

The last element is the one a contraindication list silently omits, and it is why the modern position can be stated as risk quantification rather than prohibition: a patient in catatonic stupor who is not eating carries a risk from not being treated that no table of conditions records. The cognitive limb belongs to this chapter's account of the cognitive side effects of ECT; owned here is the framework.

MNEMONIC

Two dates, four systems, one patient

Two dates: position two is 1980, position one is 2001 and later. **Four systems**: the Indian caution table runs cerebral, ocular, cardiac and neuromuscular, and the list is open. **One patient**: the single reported case treated with raised intracranial pressure already present is the whole of the direct evidence.

5.9 The anaesthetic considerations, and the consequence this item must point at

Every condition here is managed with the anaesthetist rather than adjudicated alone, which is what position one means in practice. The high anaesthetic-risk class in the Indian cardiac group is an anaesthetic judgement inside a psychiatric guideline, and managing the aneurysm patient's blood-pressure surge is an anaesthetic act. The evaluation and investigations, fasting and the medication review, pre-oxygenation and the airway, the induction agent and muscle relaxation in full all belong to this chapter's account of the pre-ECT workup and anaesthesia. No agent, no dose and no step of the sequence is restated here.

One consequence must be pointed at rather than left implied. A patient in whom a cerebrovascular event is managed rather than barred carries a neuromuscular-blockade consequence, and that consequence belongs in full to the workup and anaesthesia item, which states its window and grounds it.

RULE **Do not carry the word "recent" across from this item to the blockade question.** Recency here marks the cardiovascular and cerebral risk of the induced seizure, highest close to the event and falling with time. The neuromuscular-blockade risk after a cerebrovascular event does not run in that direction, and it is not discharged by the event ceasing to be recent. Nothing here says a patient is past the blockade risk because the stroke is old; that window is the workup and anaesthesia item's to state and ground.

Electroconvulsive therapy has two positions on contraindication and both are written with their dates: no absolute contraindication, from the task-force tradition of 2001 and the reviews following it, and the intracranial mass as an absolute bar, from the last large review before the modern literature in 1980, still standard teaching and a common key. The mechanism behind the older position is the induced rise in cerebral blood flow and intracranial pressure, and the chain from seizure to peritumoural oedema to neurological signs. The current Indian guideline files raised intracranial pressure under caution and instructs postponement rather than prohibition; every other high-risk condition is quantified and managed with the anaesthetist, each carrying its own modification.

Electroconvulsive therapy: technique and dose

6 · Pre-ECT workup and anaesthesia (agents, muscle relaxant, airway)

Modified ECT is a general anaesthetic given in order to induce a seizure, which puts most of the treatment's physical risk in the anaesthetic room rather than at the stimulus dial. The psychiatrist neither prescribes nor gives it, but selects the patient, orders the workup and reads a seizure the induction agent has shaped. What is examinable is what must be settled before a course, and when the usual relaxant must not be used.

Owned here: the evaluation and its investigations, fasting and the medication review, pre-oxygenation and the airway, the induction agents, neuromuscular blockade in full, the anticholinergic question, the tourniquet method, and recovery. Named but not re-derived: the seizure threshold and stimulus dose, which belong to this chapter's account of stimulus dosing; the conditions that raise risk beforehand, to the risk profile; the interactions, to concurrent medication.

2, 6, 8 HOURS OF FASTING FOR CLEAR LIQUIDS, LIGHT MEALS, AND FRIED OR FATTY FOOD	**7 to 10** DAYS AFTER A DENERVATING INJURY AT WHICH HYPERKALAEMIC RISK PEAKS	**Not mandatory** THE ROUTINE PRE-ECT BLOOD PANEL, PER THE CURRENT INDIAN GUIDELINE

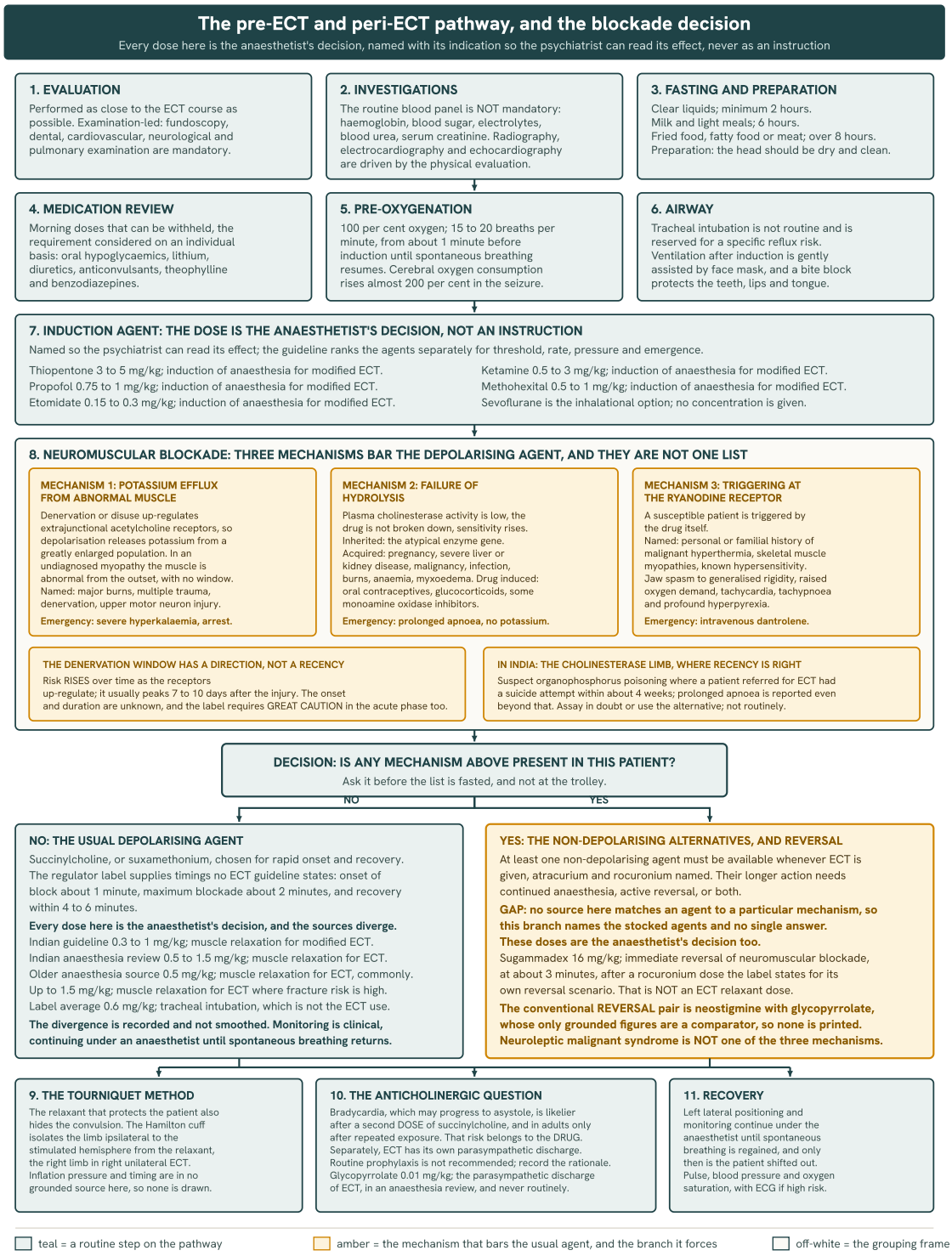


Fig 27.4. The pathway before and around a treatment, with neuromuscular blockade drawn as a decision rather than a default: on one branch the usual depolarising agent, on the other the non-depolarising agents that must be stocked, taken when any one of the three mechanisms named on the plate bars the depolarising agent. Three things are deliberately absent from that branch. No grounded source here matches a particular alternative agent to a particular barring mechanism, so it names the stocked class and declares the gap, not one drug. It carries no relaxant dose for any alternative agent, the one rocuronium figure being the reversal label's own antecedent and marked as not an ECT dose. And the neuroleptic malignant syndrome preference is not here, because it is not one of the three mechanisms and the item's text records that succinylcholine given for ECT in it is described as safe. Every dose shown is named with its indication as the anaesthetist's decision rather than as an instruction.

6.1 The evaluation before a course

The **pre-ECT evaluation** is a joint fitness-for-anaesthesia assessment whose first rule is timing: the guideline requires it be performed as close to the ECT course as possible, because a workup done weeks earlier certifies a patient who no longer exists.

The examination is asked as it stands. Fundoscopic examination is mandatory along with other systemic examinations, and so are dental evaluation for loose or missing teeth, cardiovascular examination for arrhythmias, assessment for neurological comorbidities and pulmonary clinical evaluation. Fundoscopy is the window on raised intracranial pressure, taken up by the risk-profile account; loose teeth matter because the masseter contracts whatever the relaxant does.

6.2 The investigations: what is required, and what is not

Candidates fail this by reciting a battery. The guideline states that for general anaesthesia haemoglobin, blood sugar, electrolytes, blood urea and serum creatinine would facilitate detection of common risk-enhancing comorbidities but are not mandatory, and radiography, electrocardiography, echocardiography and other tests are indicated on the physical evaluation.

RULE **Pre-ECT investigation is driven by the clinical evaluation, not by a fixed list.** The mandatory content sits in the examination, not the laboratory; the tests that earn a place are those a finding asked for, an electrocardiogram because an arrhythmia was heard, a potassium level because the relaxant question is live.

6.3 The morning of a treatment: fasting and the medication review

Fasting is graded by what was eaten, and the intervals are quoted, not recalled: ECT is given fasting, after **2 hours from clear liquids, 6 hours from milk and light meals, and over 8 hours from fried food, fatty food or meat**. The same guideline table adds one preparation step alongside the intervals, outside the passage quoted here: the head should be dry and clean for the procedure, which is a scalp and electrode-contact requirement and not a fasting one.

The review is drug by drug that morning. The morning dose of oral hypoglycaemics, lithium, diuretics, anticonvulsants, theophylline and benzodiazepines can be withheld, considered on an individual basis. The opposite list is forgotten: scheduled antihypertensives, thyroxine, antiemetics, antireflux drugs, antianginals, bronchodilators and anticholinergics should not be withheld and can be taken orally 2 hours before the procedure with small sips of water. Which drug moves the threshold belongs to concurrent medication.

6.4 In the suite: pre-oxygenation, the airway and what must be stocked

Pre-oxygenation answers a measured demand, because cerebral oxygen consumption increases almost 200% during the seizure, so the recommendation is ventilation with 100% oxygen at 15 to 20 breaths per minute, from approximately 1 minute before induction until spontaneous breathing resumes. Hyperventilation may prolong the seizure; its threshold effect belongs to stimulus dosing.

Intubation is not routinely required unless there are specific risk factors for reflux, ventilation after induction is gently assisted with a face mask, and a bite block protects the teeth, lips and

tongue. That source predates current consensus and is carried for want of a plainer one. The bite block is the step most often left out of an answer: the stimulus contracts the masseter by a route the relaxant misses. The room is stocked: at least one non-depolarising agent such as atracurium or rocuronium must be available alongside succinylcholine.

6.5 The induction agent: rankings that do not run in the same order

The induction agent decides how easily a seizure is obtained, how long it runs, how the circulation behaves and how fast the patient wakes, and the guideline gives these as separate rankings.

RANKING	ORDER AS THE GUIDELINE GIVES IT
SEIZURE THRESHOLD, PROCONVULSANT TO ANTICONVULSANT	ketamine, etomidate, methohexital, sevoflurane, thiopentone, propofol
PROPENSITY TO INCREASE HEART RATE	ketamine, methohexital, thiopentone, etomidate, propofol
PROPENSITY TO INCREASE MEAN ARTERIAL PRESSURE	ketamine, etomidate, methohexital, thiopentone, propofol
EMERGENCE TIME, LONGEST TO SHORTEST	ketamine, etomidate, thiopentone, methohexitone, propofol, sevoflurane

Read together they show the trade: ketamine sits at the proconvulsant end of the threshold ranking and the worst end of rate, pressure and emergence, and propofol the reverse on threshold, rate and pressure, with sevoflurane alone shorter than propofol on emergence. Each figure below is the anaesthetist's decision, named so the psychiatrist can read its effect on the seizure, never as an instruction.

- **Thiopentone.** 3 to 5 mg/kg for induction in modified ECT, owned by the anaesthetist and not taught here as a prescribing instruction: fast acting, reduces seizure duration less than propofol, increased arrhythmia risk.
- **Propofol.** 0.75 to 1 mg/kg for induction in modified ECT, owned by the anaesthetist: shortens seizure duration and raises the threshold, but better haemodynamic stability in cardiovascular risk, and quicker emergence, against injection pain.
- **Etomidate.** 0.15 to 0.3 mg/kg for induction in modified ECT, owned by the anaesthetist: prolongs seizure duration and may reduce the threshold, useful where it is high, against a hyperdynamic response, longer emergence and adrenocortical suppression.
- **Ketamine.** 0.5 to 3 mg/kg for induction in modified ECT, owned by the anaesthetist: seizure effect unclear with a modest enhancing effect, sedative and analgesic, against emergence phenomena, poorer haemodynamic stability and raised intracranial pressure.
- **Methohexital.** 0.5 to 1 mg/kg for induction in modified ECT, owned by the anaesthetist: the gold standard, whose problem in India is sparse availability.
- **Sevoflurane.** The inhalational option, comparable to thiopentone, useful where venous access is difficult, with better haemodynamic stability, enhancing muscle relaxation, and in

pregnancy reducing uterine contraction. Enhanced muscle relaxation is listed as a general advantage of the agent, not as a property of its use in pregnancy. The guideline gives no concentration, so none is supplied.

Propofol with ketamine, called ketofol, can balance seizure duration against haemodynamic effect; short-acting opiates or dexmedetomidine spare dose but need more evidence in ECT; and the differential effects of anaesthetic agents are dependent on their dose. Calling an agent proconvulsant is therefore a statement about a dose.

6.6 Neuromuscular blockade, part one: the depolarising agent

Succinylcholine, or suxamethonium, is the depolarising blocker for modified ECT, chosen for rapid onset and recovery. The label supplies timings no ECT guideline states: blockade develops in about 1 minute, maximum blockade may persist about 2 minutes, and recovery follows within 4 to 6 minutes. Read the middle figure as the label writes it: what persists for that interval is the MAXIMUM of the block and not the block itself, so nothing on this page promises breathing when it elapses. Its average of 0.6 mg/kg is for tracheal intubation, not ECT, an indication owned by anaesthetic practice and named only so it is never taken for ECT. Monitoring is clinical: it continues under an anaesthetist until spontaneous breathing is gained.

The ECT dose is not agreed, and the divergence is recorded, not smoothed. The Indian psychiatry guideline gives **0.3 to 1 mg/kg** for relaxation in modified ECT, owned by the anaesthetist. An anaesthesia review gives **0.5 to 1.5 mg/kg** for relaxation in ECT, also owned by the anaesthetist. An older one gives 0.5 mg/kg as the commonest relaxant dose in ECT, owned by the anaesthetist, and alone names who needs more: up to 1.5 mg/kg for relaxation in ECT in severe cachexia, osteoporosis or skeletal injury, owned by the anaesthetist. Fragile bones need more relaxation, not less.

6.7 Neuromuscular blockade, part two: the mechanisms that bar the depolarising agent

The situations that contraindicate a depolarising blocker do not share a mechanism, and the difference is not academic, because the emergency each produces is treated differently. The routes are potassium efflux from abnormal muscle, driven after denervation or disuse by up-regulated **extrajunctional acetylcholine receptors**; failed hydrolysis where **plasma cholinesterase**, or pseudocholinesterase, is deficient; and ryanodine-receptor triggering in **malignant hyperthermia**. One hyperkalaemia story across all three prepares the wrong emergency.

MECHANISM	WHAT GOES WRONG	CONDITIONS NAMED, WHICH IS NOT A CLOSED LIST	THE EMERGENCY
POTASSIUM EFFLUX FROM ABNORMAL MUSCLE	Receptors up-regulate after denervation or disuse, so depolarisation releases potassium from a greatly enlarged population; in an undiagnosed myopathy the muscle is abnormal from the outset	Major burns, multiple trauma, extensive denervation of skeletal muscle, upper motor neuron injury; the label adds chronic abdominal infection, subarachnoid haemorrhage, degeneration of the nervous systems	Severe hyperkalaemia which may result in cardiac arrest
FAILURE OF HYDROLYSIS	Plasma cholinesterase activity is diminished, the drug is not broken down, and sensitivity rises	Genetic abnormalities of plasma cholinesterase; pregnancy, severe liver or kidney disease, malignancy, infection, burns, anaemia, myxoedema; oral contraceptives, glucocorticoids, certain monoamine oxidase inhibitors, organophosphate insecticides	Prolonged block with apnoea. No potassium is involved, and no grounded source here states the management, so none is printed
TRIGGERING AT THE RYANODINE RECEPTOR	A susceptible patient is triggered by the drug	Personal or familial history of malignant hyperthermia, skeletal muscle myopathies, known hypersensitivity	Masseter spasm progressing to generalised rigidity, increased oxygen demand, tachycardia, tachypnoea and profound hyperpyrexia, treated with intravenous dantrolene sodium as an adjunct to supportive measures

The potassium mechanism reaches a patient by more than one route, and their timing differs. After denervation or disuse the receptors must change first, so a window opens and widens. In an undiagnosed myopathy there is none: the boxed warning records rare reports of acute rhabdomyolysis with hyperkalaemia, ventricular dysrhythmias, cardiac arrest and death after succinylcholine in apparently healthy paediatric patients later found to have undiagnosed skeletal muscle myopathy, most frequently Duchenne muscular dystrophy, which bears on the adolescent treated under the statutory gate for minors that the Mental health legislation and forensic psychiatry notes carry. Where potassium is already high no receptor change is needed: the label directs great caution in electrolyte abnormalities and digitalis toxicity, because succinylcholine may then cause cardiac arrest due to hyperkalaemia.

RULE **The hyperkalaemic danger of a denervating insult RISES over time as receptors up-regulate, so recency is not the marker of safety.** Note what the label does NOT say: it does not certify the early period as low risk. It requires GREAT CAUTION during the acute phase and contraindicates the agent after it, so both phases carry an instruction and neither is a clearance. The label states the direction plainly: the risk increases over time and usually peaks at 7 to 10 days after the injury, depends on its extent and location, and its precise onset and duration are not known. The contraindication matches, applying after the acute phase of injury. Since the duration is unknown, this account teaches direction and peak and prints no date of safety.

TRAP **Reading a hemiplegic patient's blockade risk off the recency of the stroke inverts the window and clears exactly the wrong patient.** The recency wording attached to a cerebrovascular or cardiac event in this chapter's account of the risk profile concerns the risk of the induced seizure, highest close to the event. Blockade risk runs the other way. Letting one set the other refuses the relaxant before the receptors have up-regulated, where the label still requires great caution and prints no interval of safety, and hands a depolarising agent to the established hemiplegic in whom danger has built for weeks.

MNEMONIC

POTASSIUM, PROLONGED, PYREXIA

One P for each mechanism, and none shares an emergency with another. **Potassium** is the denervation, disuse and abnormal-muscle route, ending in hyperkalaemic arrest. **Prolonged** is the cholinesterase route, where the emergency is prolonged apnoea and no source here states its management. **Pyrexia** is malignant hyperthermia, treated with dantrolene. The mechanisms are the whole set; the conditions inside each are not, so a patient absent from a list has not been cleared.

IN INDIA

The cholinesterase mechanism has an Indian limb, and is the one place recency is the right marker. Where a patient referred for ECT has a suspected or known suicide attempt within 4 weeks, organophosphorus poisoning should be suspected; prolonged apnoea is reported even after 4 weeks; the pseudocholinesterase level may be assessed in doubt or a non-depolarising agent used; routine determination is not recommended. In renal disease, non-depolarising agents can be preferred, and in haemodialysis patients potassium should be done within 24 hours before ECT. Note the strength difference. the guideline frames its list, adding severe widespread burns, hypercalcaemia and severe neuromuscular disease, as situations in which non-depolarising relaxants may be considered, while the label states several as contraindications.

6.8 Neuromuscular blockade, part three: the alternatives and their reversal

A contraindication is useful only with a replacement, and here the replacement is a class rather than a name. The **non-depolarising blockers** named for ECT are atracurium, mivacurium and rocuronium, whose shared cost is that their relatively prolonged action will need continued anaesthesia, active reversal after treatment, or both. What the guideline settles is stock and not selection: at least one non-depolarising agent such as atracurium or rocuronium must be available alongside succinylcholine.

TRAP **Answering "which agent replaces succinylcholine" with a single drug name.** The three barring mechanisms are not one list, and no source carried in this chapter matches a particular alternative to a particular mechanism, so no one agent can be the answer to all three. The honest answer names the class, says at least one member must be stocked, and leaves the choice within it to the anaesthetist on the mechanism in front of them. Naming one agent silently answers the cholinesterase-deficient patient too, which is the limb where a wrong choice reproduces the prolonged block the substitution exists to avoid.

One agent is reported as the usual practical choice, and it is named as that rather than as the answer to a mechanism. Mivacurium is the drug most often administered as an alternative to succinylcholine during ECT, which is a statement about ECT practice in general and not about which mechanism has barred the depolarising agent in the patient in front of you. Its figures follow for that reason alone. Seizure modification is inadequate at 0.08 mg/kg of mivacurium for relaxation in ECT, owned by the anaesthetist and quoted only for what follows. At least 0.15 mg/kg should be used for relaxation in ECT, owned by the anaesthetist, because a dose trimmed to shorten the block buys an unmodified convulsion under a patient who looks paralysed. A second source disagrees with that figure rather than supporting it, and the disagreement is the honest state: only a full intubating dose, 0.2 mg/kg, gave effective relaxation during ECT, and higher doses caused histamine release and hypotension. Read the two together as a range of opinion on what is adequate, never as one figure evidencing the other. Atracurium at 0.5 mg/kg needed more time to reach a satisfactory train-of-four ratio.

Reversal changed that. Rocuronium and vecuronium were generally given only as precurarising agents, but could theoretically be used with sugammadex. **Sugammadex** encapsulates the relaxant rather than inhibiting an enzyme: 16 mg/kg is recommended where blockade must be reversed soon, approximately 3 minutes after a single 1.2 mg/kg dose of rocuronium, owned by the anaesthetist, and that timing is what makes the alternative usable. Its one reported use in ECT is a single case, never a schedule: the train-of-four ratio recovered to 0.9 within 2 minutes and the first breath within 3 minutes. The conventional REVERSAL pair, which is not a relaxant, is neostigmine with glycopyrrolate, whose only grounded figures are a trial comparator and are not printed. Read the next sentence against that distinction, because the preference belongs to the relaxant and not to the reversal: after neuroleptic malignant syndrome it is rocuronium with sugammadex that is called the more promising method of muscle relaxation, although the same source states that evidence is lacking for a shared pathophysiology with malignant hyperthermia and that succinylcholine given for ECT in it is safe: caution, not a proven contraindication.

GOOD TO REMEMBER

Ask the relaxant question before the list is fasted, not at the trolley. Is there a denervating or disuse lesion, and how long ago was the insult, remembering that risk RISES toward the peak this page states in the rule above and that the acute phase carries its own caution, so recency is not safety in either direction? What may have lowered cholinesterase: organophosphate, liver or renal disease, pregnancy, a family history of prolonged apnoea? Has anyone in the family had malignant hyperthermia?

6.9 The anticholinergic question

Two separate routes slow the heart here and the sources attach them to different things. The first is the stimulus: an anaesthesia review names an initial parasympathetic discharge of ECT as what an anticholinergic is given to antagonise. The second belongs to the drug, not to the stimulus: the incidence of bradycardia, which may progress to asystole, is higher after a second dose of succinylcholine; in adults it is seen only after repeated exposure; and pretreatment with an anticholinergic such as atropine may reduce bradyarrhythmias. Read the second as a dose effect, because that is what the label describes: it says nothing about a second electrical stimulus. A restimulation may carry both, a further discharge and, where a second dose of relaxant is given with it, the label's raised bradycardia risk, and that is why the session that goes to restimulation is a cardiac event as well as an electrical one.

The guideline settles it against routine use. Routine prophylactic use of anticholinergics such as atropine or glycopyrrolate, and of beta blockers, calcium channel blockers, nitrates, hydralazine and ganglionic blockers for cardiovascular stability, is not recommended, and wherever used the rationale should be noted. An anaesthesia review is permissive: glycopyrrolate at 0.01 mg/kg is preferred to antagonise the initial parasympathetic discharge of ECT, intramuscularly at least 3 minutes before the procedure or intravenously just before induction, owned by the anaesthetist and named only so the psychiatrist recognises what is given. It benefits patients on beta blockers and those whose threshold is unset.

6.10 The tourniquet method, recovery and monitoring

The **Hamilton cuff method** exists because the relaxant that protects the patient from a convulsion also hides it. It monitors motor seizures by isolating the ipsilateral limb from muscle relaxants, the right limb in right unilateral ECT, and is preferred in modified ECT because it indicates generalisation. The inflation pressure and its timing are in no grounded source.

Recovery is physiological, not timed. Left lateral positioning and monitoring in the suite continue under an anaesthetist until spontaneous breathing is gained, and only then is the patient shifted to the recovery room. There, pulse rate, blood pressure and oxygen saturation are monitored, with the electrocardiogram in high-risk patients, the example being cardiac disease. Postictal confusion is measured, not waited out: orientation and gait are assessed for recovery to baseline or near baseline before shifting the patient out.

Recovery is a staffed competency, not a room. The guideline specifies a recovery nurse trained in post-anaesthesia care, able to monitor vital signs, pulse oximetry and electrocardiogram, give oxygen, fluids and suction, and support disorientation, delirium or agitation.

The evaluation is done as close to the course as possible and is examination-led: fundoscopy, dental, cardiovascular, neurological and pulmonary examination are mandatory, the blood panel is not, imaging and cardiac tests are indication driven. Fasting is graded at 2, 6 and 8 hours. The induction agent is chosen against separate rankings, ketamine proconvulsant but worst for rate, pressure and emergence. Succinylcholine is the usual relaxant, barred by separate mechanisms whose emergencies differ: potassium efflux from abnormal muscle, failed cholinesterase hydrolysis producing apnoea, and ryanodine triggering treated with dantrolene. Denervation risk RISES over time, with the onset and the duration expressly unknown and great caution required in the acute phase too, and peaks at 7 to 10 days, so recency is never the marker there. A non-depolarising agent must be stocked and never under-dosed.

7 · ECT technique and electrode placement (bilateral, right unilateral, bifrontal)

Two things are decided before any current flows in electroconvulsive therapy: where on the head the two electrodes sit, and what shape of electricity passes between them. The placement is fixed entirely by bony landmarks; the waveform and the electrical parameters fix what the stimulus contains; the impedance test says whether the circuit on the head is the one intended, and the monitoring says what the brain did with it.

Owned here in full and defined nowhere else: each electrode placement with its landmarks and geometry, the stimulus waveform, the electrical parameters that define a stimulus, impedance testing, and the monitoring of the induced seizure. Named and never re-derived, each with its own account elsewhere in this chapter: the comparison between the placements, a dose-dependent trade-off, in the notes on bilateral against right unilateral treatment; the threshold, titration and the adequate-seizure criteria, in the notes on stimulus dosing; the amnesias, in the notes on cognitive side effects; the anaesthetic conduct, in the notes on the pre-ECT workup.

three	four	100 to 3000
MEMBERS OF THE BILATERAL CLASS IN THE CURRENT INDIAN GUIDELINE	ELECTRICAL PARAMETERS THAT TOGETHER DEFINE A STIMULUS	OHMS: THE STATIC IMPEDANCE WINDOW ON ONE NAMED DEVICE

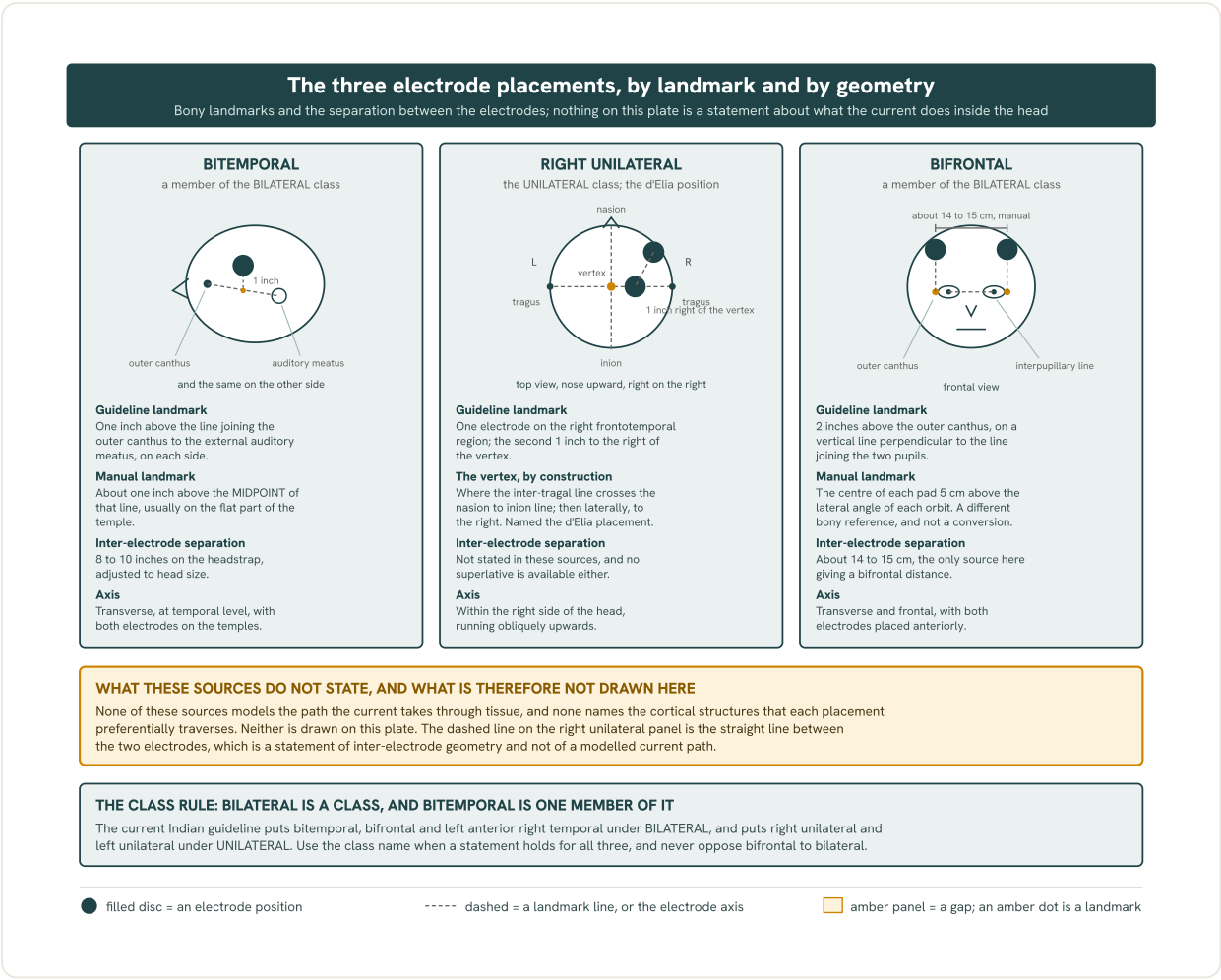


Fig 27.5. The three placements drawn by their bony landmarks and by the separation between their electrodes: bitemporal one inch above the line from outer canthus to external auditory meatus, and above the midpoint of that line in the device manual, with the electrodes eight to ten inches apart on the headstrap; right unilateral in the d'Elia position, one electrode right frontotemporal and the second one inch to the right of the vertex, the vertex itself constructed from the inter-tragal line crossing the nasion to inion line; and bifrontal two inches above the outer canthus in the guideline, five centimetres above the lateral angle of the orbit in the manual and about fourteen to fifteen centimetres apart, two measurements against two bony references rather than one figure converted, with bitemporal and bifrontal both members of the bilateral class, and no current path or traversed cortical structure drawn because no source here states one.

7.1 Bilateral is a class of placements, not one placement

The taxonomy must be right before any landmark is learnt, because every later comparison is phrased in it. The current Indian guideline classifies placements under two headings, Bilateral and Unilateral. Bilateral has three members: bitemporal, bifrontal and left anterior right temporal. Unilateral has two: right unilateral and left unilateral. **Bilateral** therefore names a class; **bitemporal** is one member of it, not a synonym for it.

The consequence is what gets examined. **Bifrontal** is a bilateral placement, not a third option outside the bilateral class. Written the other way it makes the placement comparison unreadable, since a sentence contrasting bilateral with bifrontal contrasts a class with one of its members. That comparison belongs to the notes on bilateral against right unilateral treatment; this fragment answers only the prior question, which is what each placement physically is.

RULE **Bilateral is the class; bitemporal, bifrontal and left anterior right temporal are its members.** Use the class name when a statement holds for all three and the member name when it does not, and never oppose bifrontal to bilateral. The guideline's numbered headings are the authority, not usage.

7.2 Bitemporal: the classical placement

The Indian guideline of 2023 calls bitemporal the classical method and defines it by one line on the face: an electrode in the frontotemporal region, one inch above the imaginary line joining the outer canthus and the external auditory meatus, and the same on the other side.

The guideline does not say which point on that line is meant. The manufacturer's manual does, and adds the separation: about one inch above the mid-point of that line, usually on the flat part of the temple, with the electrodes **8 to 10 inches** apart on the perforated headstrap, by head size. The guideline gives the height above the line, the manual the point on it and the distance; neither is complete alone.

7.3 Right unilateral in the d'Elia position

The right unilateral montage is two points far apart, the second defined by a construction rather than by a bone. One electrode sits on the right frontotemporal region and another 1 inch to the right of the vertex, the vertex being where two imaginary lines cross, one joining the two tragi and the other joining nasion to inion. The construction is examinable in itself: inter-tragal line, nasion to inion line, their crossing, then laterally to the right.

The manual states the eponym: for right unilateral placement the d'Elia placement is recommended. The same manual carries the conduct, which is not incidental: the skin over the right temple is cleaned and dried and the pad applied to bare skin, then the hair near the vertex is parted and the scalp moistened thoroughly with saline. Hair lies under one of these electrodes and not the other, which is why the saline step attaches to the vertex electrode and why that is the one that misbehaves on the impedance check.

7.4 Bifrontal, and two landmarks that must not be merged

Bifrontal puts both electrodes over the frontal regions, and two independent statements of where are carried here. The Indian guideline puts them 2 inches above the outer canthus, on an imaginary vertical line perpendicular to the line joining the two pupils. The device manual puts the centre of each pad 5 cm above the lateral angle of each orbit, about 14 to 15 cm apart, the only source here giving a bifrontal inter-electrode distance.

These are two measurements, not one measurement in two dresses. The units differ and the bony reference differs with them: the outer canthus is the soft-tissue corner of the eye, the lateral angle of the orbit a point on bone. They land close together on a real head and remain separate quotations from separate documents. Nothing here converts one into the other, because a converted figure is an arithmetic step by the author rather than a quotation, putting on the page a number that neither source states.

PITFALL

Converting the bifrontal landmark out of one source's units into the other's and reporting the result as though a guideline had said it. Both are printed against their own bony references, so the reader can quote whichever document a question asks about. The same discipline governs every parameter here: quote the source, name it, and neither average nor convert between sources.

7.5 What the landmarks fix, and what they do not

Geometry is what a landmark buys. Bitemporal puts both electrodes on the temples, so the axis between them runs transversely at temporal level; bifrontal puts both anteriorly, so the axis is transverse and frontal; right unilateral puts one at the right temple and the other just right of the vertex, so the axis lies within the right side of the head and runs obliquely upwards, and the wide separation is what makes this the long-distance montage over the non-dominant side.

What these sources do not state matters as much, because the temptation is to supply it from memory: none models the path the current takes through tissue, and none names the cortical structures each placement preferentially traverses.

AXIS	BITEMPORAL	RIGHT UNILATERAL	BIFRONTAL
CLASS	Bilateral	Unilateral	Bilateral
GUIDELINE LANDMARK	One inch above the line from outer canthus to external auditory meatus, each side	Right frontotemporal, plus 1 inch right of the vertex	2 inches above the outer canthus, on the perpendicular to the interpupillary line
MANUAL LANDMARK	About one inch above the mid-point of that line	Named as the d'Elia placement; vertex scalp moistened	Centre of each pad 5 cm above the lateral angle of the orbit
SEPARATION CARRIED HERE	8 to 10 inches	Not stated in these sources, and no superlative is available either: both separations that ARE stated, in the bitemporal and bifrontal columns of this row, exceed the span from a right temple electrode to a point one inch right of the vertex	About 14 to 15 cm

7.6 Waveform: sine wave, brief pulse, ultrabrief pulse

The **stimulus waveform** is the shape of the electricity in time. The original sine-wave stimulus had a long pulse width of 8.3 ms and was inefficient, using more energy than neurons require in order to discharge; the square-wave brief pulse of 0.5 to 1.5 ms that replaced it reduced cognitive side effects while keeping efficacy. The Indian guideline states the modern position as a recommendation, not as history: a brief or ultrabrief pulse is strongly recommended and should

be given with a constant current device, and sine-wave and constant voltage systems are not recommended in modern practice, on safety grounds.

The bands are where the sources disagree, so each is stated with its body. The Indian guideline of 2023 defines brief pulse as 0.5 to 2 ms and ultrabrief as 0.2 to 0.4 ms. The Australian and New Zealand college guideline of 2019 defines brief pulse as 0.5 to 2.0 ms and ultrabrief as 0.25 to 0.3 ms. The two GUIDELINE brief-pulse bands agree, as the two quoted sentences above show, and it is that agreement and no wider one that may be claimed: a third and narrower brief-pulse upper bound, from a meta-analysis, appears elsewhere in this chapter, so a candidate who memorises the narrowest answers wrong against both guidelines. The ultrabrief bands do not agree at all. An answer giving any one band as the definition without naming its body has adopted a source silently.

The guideline attaches a relation to pulse width, and it holds for pulse width only. Pulse width is likely to have a linear effect on cognitive adverse effects, broader widths being associated with worse ones; an ultrabrief pulse of 0.3 ms has a cognitive advantage over brief pulses with right unilateral placement in depressive disorders, though antidepressant efficacy may be compromised with it; and a lower brief-pulse range of 0.5 to 1 ms may be considered optimal for a rapid clinical effect. The college guideline bounds where that has been shown: for ultrabrief bilateral treatment the published evidence is very limited and efficacy is lower than with brief-pulse bilateral treatment. Both are statements about pulse width inside a placement, and neither licenses carrying this direction across to another parameter. No comparison between placements is made here.

7.7 The four parameters, and why charge alone is not the dose

A stimulus is a tuple, not a number. The arithmetic comes first: electrical charge = current intensity x pulse width x pulse frequency x train duration. The guideline then warns against the natural misreading of its own equation. Charge is generally treated as a linear measure and the chief parameter of dosing, but that approach is faulty: the four parameters in combination, along with electrode placement and the frequency and duration of sessions, are what should be considered in choosing a protocol. Two stimuli of equal charge built from different parameters are different stimuli.

Current intensity is the parameter that is not moved. Historically 500 to 1000 mA has been used, most devices default to 800 to 900 mA, and current correlates linearly with tolerability, cognitive effects and seizure quality, but is held constant during dose incrementation. Low-amplitude stimulation is named in the same paragraph as investigational, its clinical utility yet to be understood; that status is the teaching point and no current band for it is carried.

Pulse frequency is the counterintuitive one. It is the number of biphasic pulses per second, the inverse of the interpulse interval, ranging from 20 to 240 pulses per second, that is 10 to 120 Hz of bidirectional pulse pairs. Lower frequencies are more efficient: a seizure is elicited at a lesser charge with a lower frequency when all else is held constant. That sits awkwardly beside how

devices behave: many increment the dose by raising frequency, so the default increment works against efficiency in the patient whose threshold is already high.

Train duration is the parameter that is moved. It is the most commonly modified parameter for setting the dose; most devices offer 0.2 to 8 s and some up to 16 s; no limit has been examined or recommended for the highest duration; and charge is raised by lengthening the train until the device's limit is reached. What multiple of the measured threshold that charge should be belongs to the notes on stimulus dosing.

MNEMONIC

CURRENT, WIDTH, RATE, TRAIN

The four parameters in the order of the charge equation. **Current** is held constant. **Width** names the waveform brief or ultrabrief. **Rate** is more efficient when lower, the opposite of the intuition. **Train** is the one moved to change the dose.

7.8 One device's specification, read as a device's

Hardware bounds the tuple. One widely used constant-current device specifies a 0.9 A constant current limited to 450 V; 10 to 70 Hz in 10 Hz increments, extending to 140 Hz in the low pulse-width programs; a pulse width of 0.25 to 1.5 ms in 0.25 ms increments, with 0.3 ms available; a train duration of 0.08 to 8.00 s; and a standard maximum output across 220 ohms impedance of 504 mC, that is 99.8 joules.

TRAP **Quoting a device's maximum output, or any of its bounds, as a universal figure.** Every value above belongs to one named device at one revision of its manual; another device has other bounds. The guideline bands for pulse width are the general statement; a specification is not. Write the device's name beside its numbers, or do not write them.

7.9 Impedance: static, dynamic, and why the check is not housekeeping

Two impedances are measured and they are not the same measurement. Stimulation has to overcome the combined resistivity of scalp, skull and other tissues. Static impedances are measured before stimulation using high-frequency alternating pulses, dynamic impedances during passage of the stimulation current, and static impedance can partly be influenced by skin preparation. **Static impedance** is thus the pre-stimulus check the operator can act on, **dynamic impedance** a property of the treatment itself.

The static test has a window and an interpretation at each end. It checks the quality of the skin-to-electrode contact; on one named device the reading should be greater than 100 ohms and less than 3000 ohms before the stimulus is administered, and a reading below 100 ohms suggests a short circuit, probably in the stimulus cable. The ends fail differently: too low says the current has found a path that is not the patient, too high that it will struggle to find one.

The check earns its place because contact quality reaches the seizure. In a single-centre retrospective analysis of 78 patients across 1757 bifrontal sessions from 2012 to 2020, static and dynamic impedance were strongly correlated, and dynamic impedance was significantly related

to Maximum Sustained Power and to the Average Seizure Energy Index but to no other seizure quality criterion; aiming for low static impedance might reduce dynamic impedance, so good skin preparation is recommended. The design bounds the claim: retrospective, one centre, bifrontal treatment only.

GOOD TO REMEMBER

A high static reading calls for a sequence, not a shrug.

- Press each electrode firmly against the skin again, especially the vertex electrode in unilateral treatment.
- Moisten the hair and scalp under the vertex electrode with saline.
- Recheck the skin preparation steps.
- Only then replace the pads, the lead-wires and the stimulus cable, in that order.

7.10 Monitoring the induced seizure

Three records are taken of one event: cortical, muscular and cardiac. What the guideline requires at the station is specific. Vitals monitoring calls for a sphygmomanometer, a reflex hammer, oxygen saturation and ECG, alongside the apparatus with its bite block and the electroencephalogram and ECG monitors.

For the cortical record the montage is specified. The electroencephalogram is a direct measure of seizure activity and is recommended wherever available; bilateral recording from at least two channels, FP1 and FP2, referenced to the ipsilateral mastoid is preferred, and where only one channel is available a contralateral channel is preferred. The fallback is contralateral because a channel over the non-stimulated side is the one that can show generalisation.

The motor record is neither optional nor a substitute. Electroencephalogram recording can carry artefacts from muscle movement and from technical problems, so motor monitoring should always supplement it. The instrument for seeing a motor seizure in a relaxed patient is the isolated-limb method: the Hamilton cuff method is recommended for monitoring motor seizures by isolating the limb ipsilateral to the stimulated hemisphere, the right limb in right unilateral treatment, from the muscle relaxant. The side is the point of the method, not a detail of it: a convulsion in the limb ipsilateral to the stimulated hemisphere is evidence that the seizure crossed to the other hemisphere and generalised. The cuff as anaesthetic conduct belongs to the notes on the pre-ECT workup; only its use as an instrument for reading the seizure is taught here.

A modern device consolidates this onto one trace, with four recording channels: 1 and 2 for the electroencephalogram, 3 for the electromyogram and 4 for the ECG. From those it computes named indices, the postictal suppression index with a range of 0 to 100 per cent, the average seizure energy index, maximum sustained electroencephalogram power and coherence with the time to peak of each, the Duke University measures, power spectral analysis, and peak heart rate. The range of the suppression index is grounded; no threshold on it is, and none is invented. What counts as an adequate seizure belongs to the notes on stimulus dosing.

IN INDIA

The current Indian Psychiatric Society guideline, published in 2023, settles four choices on this page: a brief or ultrabrief pulse from a constant current device; the placement landmarks quoted above, bifrontal among them in inches; a two-channel electroencephalogram referenced to the ipsilateral mastoid; and motor monitoring always supplementing it. Where a device manual or a college guideline states a different figure, name the document beside it.

Bilateral is a class containing bitemporal, bifrontal and left anterior right temporal; unilateral contains right unilateral and left unilateral. Bitemporal sits one inch above the line from outer canthus to external auditory meatus, above its mid-point in the manual; right unilateral is the d'Elia position, right frontotemporal plus one inch right of the vertex; bifrontal is two inches above the outer canthus in the guideline and five centimetres above the lateral angle of the orbit in the manual, two statements and not a conversion. A stimulus is current intensity, pulse width, pulse frequency and train duration together, and the seizure is read on the electroencephalogram, on an isolated limb and on the ECG at once.

8 · Bilateral versus right unilateral ECT (efficacy versus cognitive trade-off)

The choice between a bilateral placement and right unilateral placement is the decision in a course of electroconvulsive therapy most often carried in memory as a fixed ranking, and a fixed ranking is the one thing it is not. Whether right unilateral treatment is as good as bilateral treatment has no answer until the dose is named, because the two have different dose-response relations. Written as a ranking the comparison is memorable and clinically false; written as a function of the multiple of seizure threshold it is correct and usable.

Owned here in full: the trade-off between efficacy and cognitive cost across the placements, the evidence base behind it, where bifrontal placement sits within it, and how the choice is made for a given patient. Named here and not re-derived, each with its own account in this chapter: what each placement is, with its landmarks, geometry and current path; what the seizure threshold is and how it is titrated; and the amnesias themselves, their course and their monitoring. This account states only how placement and dose move that cognitive cost, never what the cost consists of.

17% against 65%	792	6 times
RESPONSE NEAR THRESHOLD, RIGHT UNILATERAL AGAINST BILATERAL, IN THE TRIAL THAT SEPARATED DOSE FROM PLACEMENT	PATIENTS IN THE POOLED COMPARISON FINDING NO DIFFERENCE ONCE THE MULTIPLE IS HIGH	THRESHOLD, ABOVE WHICH RIGHT UNILATERAL PLACEMENT NO LONGER HOLDS A COGNITIVE ADVANTAGE OVER BITEMPORAL

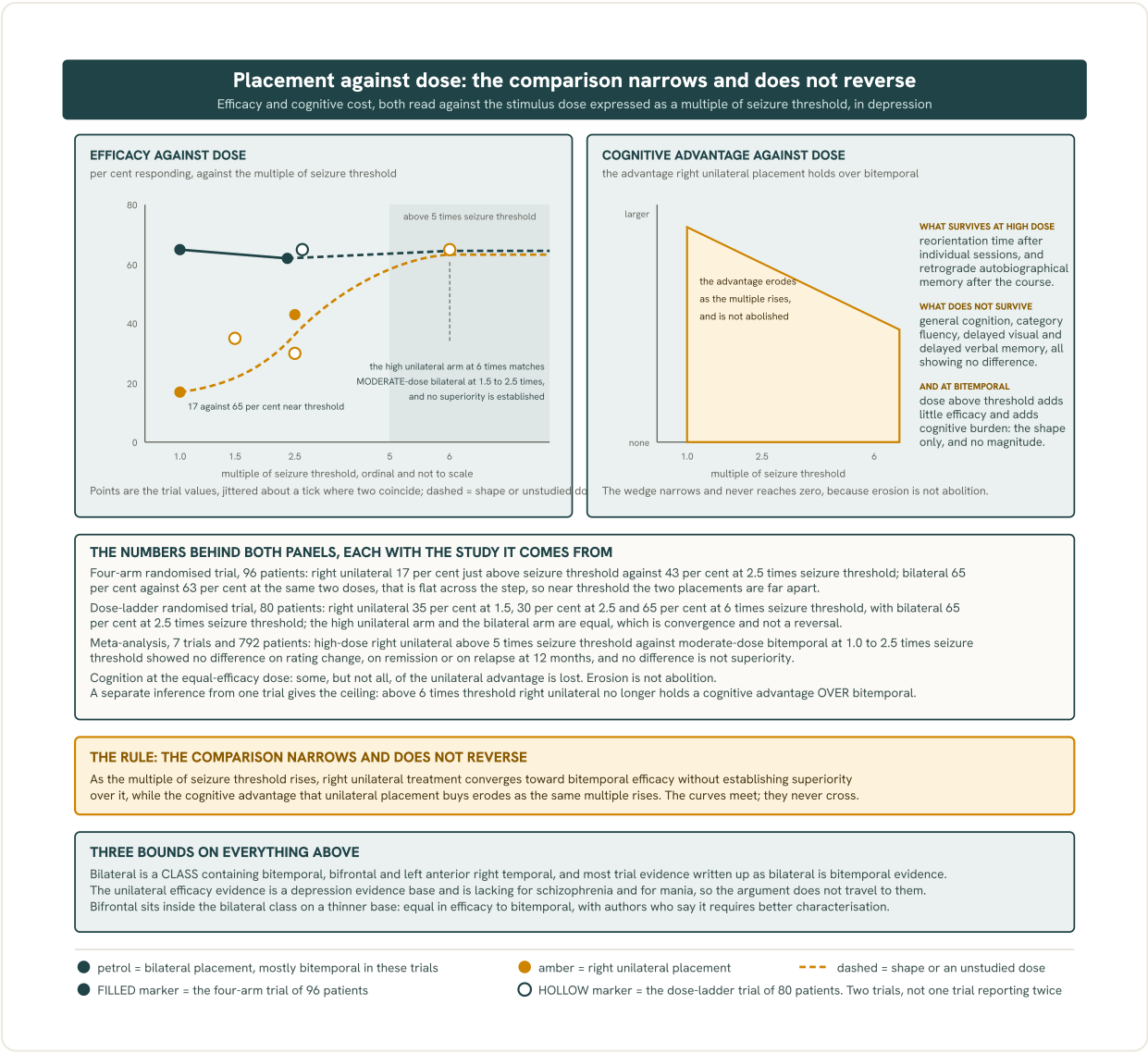


Fig 27.6. Efficacy and cognitive cost read as functions of the stimulus dose expressed as a multiple of seizure threshold, rather than as a fixed ranking of the placements. On the left, bilateral response is already near its maximum at a low multiple and stays flat, while right unilateral response is markedly inferior near threshold and rises to meet it at six times seizure threshold, so the two lines converge and never cross and no superiority of right unilateral over bitemporal placement is established. Read the meeting point precisely, because the geometry invites a stronger claim than the evidence carries: no bilateral arm in any trial here was dosed above two and a half times threshold, so the bilateral line is drawn solid only across the doses actually studied and dashed beyond them, and the match at six times is high-dose right unilateral against MODERATE-dose bilateral rather than two arms compared at the same dose. On the right, the cognitive advantage that unilateral placement buys narrows as the same multiple rises but is not abolished, surviving in reorientation time and in retrograde autobiographical memory and not in general cognition, category fluency or delayed visual and verbal memory. Both halves are stated together, and all of it is a depression evidence base.

8.1 Bilateral is a class of placements, not a placement

The terminology has to be settled before the comparison can be read, because two words are used across this literature as though they were interchangeable and they are not. **Bilateral** is a class of placements, and it has three members, not two: the current Indian guideline lists **bitemporal**, **bifrontal** and left anterior right temporal placement under the bilateral heading. Bitemporal is one member of it. Bifrontal is another member of the same class, so it is not a third option sitting outside a bilateral pair, and the word pair is itself the error, because the class is a set of three. Left anterior right temporal placement is the member most often dropped from an answer, and

dropping it is how a candidate ends up with a two-member class. What each placement is, where its electrodes sit and which way the current runs, is taught in this chapter's account of the placements themselves and is not restated here.

The distinction earns its place because of what it does to the numbers. Most of the trial evidence written up as bilateral is bitemporal evidence: the programmes used the standard bitemporal montage and their reports call it bilateral. A figure carried under the class word is a class-level figure and stays one. Every claim below names which of the two its evidence rests on; evidence about bilateral electroconvulsive therapy is never read here as evidence about bitemporal placement; and no class-level effect size is attached to bifrontal or to bitemporal individually. Where a source pooled the class and never reported the split, the within-class breakdown is not carried here and is recorded as a declared gap, because an inferred breakdown would be indistinguishable on the page from a measured one.

EVIDENCE	LABEL THE SOURCE USES	WHAT THE CLAIM RESTS ON
THE FOUR-ARM DOSE-BY-PLACEMENT TRIAL	Bilateral	Bitemporal, since that programme used the standard bitemporal montage: bitemporal evidence under the class word
THE TRIAL OF HIGH-DOSE RIGHT UNILATERAL AGAINST A ROBUST BILATERAL COURSE	Bilateral	The class; the comparator is not separated into its members, so no figure from it is narrowed here
THE 2017 META-ANALYSIS OF CONVERGENCE AND RESIDUAL COGNITIVE ADVANTAGE	Bitemporal	Bitemporal, named as such by its authors, which makes it the cleanest source here
THE 2003 META-ANALYSIS OF BILATERAL AGAINST UNILATERAL	Bilateral	The class, pooled across doses: class-level and dose-mixed on both axes
THE BIFRONTAL META-ANALYSIS	Bifrontal, bitemporal and right unilateral, separately	The three named separately, which is why it is used here for bifrontal

8.2 Near threshold the two placements are far apart

The bottom of the dose range is anchored by one double-blind randomised trial, and its design is what makes it decisive. 96 depressed patients were assigned to right unilateral or bilateral treatment at either a low dose, just above the seizure threshold, or a high dose at 2.5 times the threshold. What the multiple of threshold is, and how the threshold beneath it is measured, belongs to this chapter's account of stimulus dosing and is taken as read.

The results are two relations and not one, which is the teaching point. For right unilateral placement the **dose-response relation** was steep: **17 per cent responded at the low dose against 43 per cent at the high dose**. For bilateral placement it was flat across the same step: **65 per cent at the low dose and 63 per cent at the high dose**. Near threshold, right unilateral treatment is markedly inferior and bilateral efficacy is already near its maximum. A later meta-

analysis states the settlement plainly: the effectiveness of right unilateral ECT depends on the strength of the electrical dose above the seizure threshold, and it is relatively ineffective at the doses nearer threshold, 1.0 to 2.5 times the seizure threshold, used in bitemporal ECT.

The asymmetry is stated per placement, and per placement is the only way it is true: with right unilateral placement, stimuli much higher than the seizure threshold are necessary for effective treatment, while in bilateral ECT they add little to efficacy while increasing the cognitive burden of the treatment. Two cautions travel with that sentence. It is an introductory assertion in a paper about thresholds, not the finding of a trial that randomised dose and measured cognition at bitemporal placement, so its shape may be taught and no magnitude attached to it. And its direction holds for this parameter only; no general law about work and cost is inherited from it anywhere in this chapter.

RULE **As the multiple of threshold rises, right unilateral treatment converges toward bitemporal efficacy without establishing superiority over it, while the cognitive advantage that unilateral placement buys erodes as the same multiple rises.** Both halves are said together or neither is said usefully. The first alone sells a high unilateral dose as free, which it is not, since the cognitive advantage is what is spent to buy the efficacy. The second alone sells it as pointless, which it is not, since a clinically meaningful advantage survives at the top of the range. The comparison narrows and does not reverse.

8.3 At a high multiple the comparison narrows

The convergence was established by a second double-blind randomised trial with a wider dose ladder. 80 depressed patients were randomised to right unilateral treatment at 50, 150 or 500 per cent above the seizure threshold, or to bilateral treatment at 150 per cent above it. Convert those before reading on, because the rest of this chapter speaks in multiples and the commonest error here is to read the largest of them as five times threshold rather than six: 50, 150 and 500 per cent above threshold are 1.5, 2.5 and 6 times threshold, and the bilateral arm at 150 per cent above threshold is 2.5 times it. **High-dose right unilateral and bilateral treatment were equal in response rate at 65 per cent, and roughly twice as effective as the low-dose arm at 35 per cent or the moderate-dose arm at 30 per cent**. That is convergence and not a reversal: the two placements meet, and no superiority of right unilateral treatment over bilateral treatment is established anywhere in it.

The pooled position agrees, and it is also the source that names its comparator properly. Seven trials and 792 patients compared high-dose right unilateral treatment, above 5 times the seizure threshold, with moderate-dose bitemporal treatment at 1.0 to 2.5 times threshold, and found no difference on depression rating change, with a Hedges's g of -0.03 and a 95 per cent confidence interval of -0.17 to 0.11; on remission, with a risk ratio of 1.06 and an interval of 0.93 to 1.20; or on relapse at 12 months, with a risk ratio of 1.42 and an interval of 0.90 to 2.23.

Two readings of that are wrong in opposite directions. No difference is not superiority: every interval straddles the null, so the wording is **convergence without established superiority**, and calling high-dose unilateral treatment better than bitemporal treatment goes past the evidence.

Nor is no difference an argument for a low unilateral dose, since the result is conditional on the high multiple.

8.4 What the higher multiple costs, and what it does not

The cognitive advantage is not free of the dose either, and the same meta-analysis states the erosion and its limit in one sentence, which is the form to reproduce: some, but not all, of the cognitive advantage of right unilateral ECT is lost when it is given at a dose sufficient to achieve equal clinical efficacy with bitemporal ECT. Erosion is not abolition, and that is why the placement is still chosen at a high multiple rather than abandoned once the efficacy gap closes.

What survives is domain-specific, and it is grounded in two domains and no others. Right unilateral treatment kept an advantage on reorientation time after individual sessions, a mean difference of -8.28 minutes with a 95 per cent confidence interval of -12.86 to -3.70, and on retrograde autobiographical memory after the completed course, a Hedges's g of -0.46 with an interval of -0.87 to -0.04, while there were no differences for general cognition, category fluency, or delayed visual and verbal memory. A claim of a broad cognitive advantage at high dose is therefore not available; a claim of a real one in named domains is.

The trial evidence carries the counterweight most directly. In the week after the randomised phase, bilateral treatment produced greater impairment than any dosage of unilateral treatment on several measures of anterograde and retrograde memory, and at two months the retrograde deficits were greatest among the patients treated bilaterally. Greater than any dosage means the advantage was not abolished even at the top of the ladder, and the later assessment means it was not merely a same-week effect. What those impairments are and how they are tracked belongs to this chapter's account of the cognitive cost; only their movement with placement and dose is settled here.

The ladder has a ceiling on this axis, drawn from a single trial rather than a pooled estimate. The only included study that favoured bitemporal placement on autobiographical memory, and not significantly, had dosed right unilateral treatment at 8 times the seizure threshold, from which the authors infer that there is no cognitive advantage in going beyond 6 times threshold for right unilateral ECT. It is their inference from one trial, and it gives the practical top of the useful range.

TRAP **Answering which placement is better without being told the dose.** The question is under-specified and the examiner usually knows it. Near threshold the bilateral placements are clearly better; at a high multiple the efficacy difference is not demonstrable and part of the cognitive advantage has gone with it. Naming the multiple before answering is most of the examinable content.

8.5 Why an older pooled estimate says bilateral is better

A resident who has read the older literature meets an apparently contradictory finding, and it is not a contradiction. The 2003 systematic review and meta-analysis found bilateral treatment more effective than unilateral treatment across 22 trials and 1408 participants, with a standardised effect size of -0.32 and a 95 per cent confidence interval of -0.46 to -0.19, that is a

small to moderate advantage for bilateral placement. Its trials did not separate high-dose unilateral treatment from low-dose and moderate-dose unilateral treatment. This chapter's reading of that is that the review pooled across the very variable the comparison turns on, so a pooled estimate over a mixture of doses answers a different question from a comparison at a stated multiple; the reading is stated as a reading, because the review does not describe its own placement analysis in those terms. What the review does say about dose it says separately, and it points the same way: high dose ECT is more effective than low dose. The older figure is cited for what it established when it was published and never as current practice.

8.6 Where bifrontal placement sits, and how thin its evidence base is

Because bifrontal placement is a member of the bilateral class, the question is not whether it beats the class but where it sits inside it. A meta-analysis comparing bifrontal with bitemporal and with right unilateral treatment found efficacy equal between bifrontal and bitemporal, with a Hedges's g of 0.102 and a confidence interval of -0.110 to 0.313, and equal between bifrontal and right unilateral, with a standardised mean difference of -0.12 and an interval of -0.378 to 0.139; post-treatment decline on the Mini-Mental State Examination was less for bifrontal than bitemporal, with a g of 0.89 and an interval of 0.054 to 1.724, but not less than for right unilateral; and the authors conclude that bifrontal treatment is not more effective than the other two and, given the longer experience with them, requires better characterisation. That last clause answers how the evidence bases differ in size: the bifrontal one is younger and thinner, not contrary.

The current Indian guideline takes a stronger position, recorded beside the meta-analysis rather than merged with it. Clinical trials have shown that bifrontal is equally if not more effective than bitemporal placement, but with lesser cognitive effects, in patients with mania, depression as well as schizophrenia. Under the precedence this series declares, the guideline controls Indian practice questions; it does not dissolve a meta-analytic finding that bifrontal treatment is not more effective and needs better characterisation. The disagreement is named and deliberately not resolved.

GOOD TO REMEMBER

Do not let the class word do the arguing. If a source says bilateral, ask which member it used; if it says bitemporal, that is a claim about one member only; and if a figure was pooled over the class, it stays a class figure and may not be handed to bifrontal or to bitemporal alone.

8.7 How the choice is actually made for a patient

The dose-dependent comparison is the second question at the bedside and not the first, because it is an argument about one diagnosis. The evidence for the efficacy of unilateral ECT is available only for depression, and it is lacking for the other common indications, namely schizophrenia and mania. Every trial and meta-analysis above is a depression study, so carrying the convergence argument to a patient treated for schizophrenia or mania exceeds the evidence, and in Indian practice, where schizophrenia is a common indication, that limit does real work.

Where the indication permits the choice, the working rule is that **dose is matched to placement** rather than set once for the machine. A current college guideline states that bitemporal and unilateral treatment are both effective when dosed adequately, that cognitive side-effects are greater with bitemporal placement and with higher stimulus doses, and that doses up to 6 times threshold can maximise the efficacy of unilateral treatment while doses at 1.5 times threshold are usually sufficient for bilateral treatment. Read the unilateral figure as an upper bound and not as a range, because an upper bound is the only form this passage of the college guideline gives. The same guideline states that multiple a second time, in its dosing footnote, in a form that carries a FLOOR as well as a ceiling, and it is the floor that disagrees with the current Indian guideline. Both college forms and the Indian figure, with the disagreement between them, belong to this chapter's account of stimulus dosing and are printed there with their bodies and years. What must not happen here is what the open-ended form invites: quoting it alone reads as agreement and dissolves a disagreement that is real. The multiples themselves and how the threshold beneath them is titrated belong to this chapter's account of stimulus dosing, where those figures are taught with their sources and their disagreements.

Selection is not only cognitive. One grounded non-cognitive reason decides real cases: bifrontal treatment has less impact on cardiac rhythm during the stimulus than the other forms, so it is worth considering in a patient at risk of an arrhythmia induced by the treatment.

- **Indication first.** Depression admits the whole argument. Schizophrenia and mania do not, on the unilateral evidence base as it stands.
- **Then the cognitive stake.** The higher it is for this patient, the stronger the case for right unilateral placement dosed properly rather than dosed near threshold.
- **Then the medical constraint.** An arrhythmia risk is a positive reason to consider bifrontal placement, independent of any cognitive argument.

IN INDIA

Two facts shape this choice here more than the trial literature does. The unilateral evidence base is a depression evidence base, and schizophrenia is a common indication, so a bilateral placement is the default for many patients on grounds of evidence rather than habit. And the current Indian guideline, published in 2023, is more favourable to bifrontal placement than the meta-analytic literature is; quote both, with the body and the year attached to each.

MNEMONIC

CLASS, MULTIPLE, INDICATION

Before comparing placements, name the **class**: bilateral is a class of three, bitemporal, bifrontal and left anterior right temporal, so say which of them the evidence used. Before ranking them, name the **multiple** of seizure threshold, because the ranking is a function of it and has no fixed answer without it. Before carrying the ranking to the patient in front of you, name the **indication**, because the unilateral evidence base is a depression evidence base.

PITFALL

Teaching the comparison as bilateral works better, unilateral is kinder. It is compact, it is what most candidates say, and it is wrong twice over: true only near threshold on the efficacy half, and overstated on the cognitive half at the doses right unilateral treatment is actually given at. Give the dose-dependent form, with both halves stated together.

Bilateral is a class of three placements, bitemporal, bifrontal and left anterior right temporal, and most trial evidence written as bilateral is bitemporal evidence. Near threshold, right unilateral treatment is markedly inferior while bilateral efficacy is already near maximal. As the multiple of threshold rises, right unilateral treatment converges toward bitemporal efficacy without established superiority over it, while the cognitive advantage that unilateral placement buys erodes without being abolished, surviving in reorientation time and retrograde autobiographical memory. The comparison narrows and does not reverse. Bifrontal placement sits inside the bilateral class on a thinner evidence base, and the choice is made on the indication first, then the cognitive stake, then the medical constraints.

9 · Stimulus dosing, seizure threshold and adequate seizure criteria

Almost every decision in a course of electroconvulsive therapy is expressed in units of one measured quantity, the electrical dose at which this patient's brain will seize. The seizure threshold is neither a device setting nor a property of the diagnosis. It is measured in the individual patient, differs enormously between patients, moves within the same patient across a course, and the treatment dose is written as a multiple of it. A resident who can define it, set it, dose against it and judge the seizure that followed can run a course.

Owned here in full: the determinants of the threshold, how the stimulus is set, the dose as a multiple of threshold, the rise across a course, the restimulation rules and the criteria for an adequate seizure. Named here and not re-derived, each with its own account in this chapter: what each placement is and the parameters that define a stimulus; the efficacy against cognition comparison between placements; the amnesias and their monitoring; which drugs move the threshold; and why an induced seizure helps.

12-fold	1.5 to 6	47%
RANGE IN THRESHOLD BETWEEN PATIENTS IN THE TRIAL THAT QUANTIFIED IT	TIMES THRESHOLD: THE DOSE IS A MULTIPLE, SET BY PLACEMENT AND PULSE WIDTH	AVERAGE RISE IN THRESHOLD BY THE SIXTH TREATMENT WHEN IT WAS RE-TITRATED



Fig 27.7. The seizure threshold is measured by titration, varies enormously between patients and rises across a course in only some of them; the dose is a multiple of it, sized by placement and pulse width and not by one rule; a formula estimates only the first dose and its two reported errors are measured against two different benchmarks rather than running in two directions; and adequacy is judged by generalisation, electroencephalographic morphology and postictal suppression, the classical duration criteria being retired with no replacement figure offered.

9.1 A measured quantity, not a machine setting

The **seizure threshold** is the lowest electrical dose which elicits seizure activity, as seen on the electroencephalogram, that is slow wave activity, and/or visible motor movement. The disjunction is load-bearing and is quoted as the guideline writes it: either sign will do, and reading it as a requirement for BOTH would make the ordinary case of an electroencephalographic seizure without visible movement under adequate relaxation look subthreshold, which is exactly the reading the missed-seizure rule below contradicts. The definition has consequences. Being defined by a response, it can only be found by producing one, so it is measured rather than looked up. Being defined against a particular stimulus, it is a property of the patient and the waveform together, so a threshold obtained at one pulse width does not transfer to another. And being a floor rather than a treatment, it produces a seizure without necessarily treating the illness, which is why the prescription is a multiple of it.

Stimulus titration turns the definition into a number: a low stimulus is given, and if no seizure follows the dose is stepped up within the same session until one does. That dose is this patient's threshold for this session.

9.2 What determines the threshold, and how wide the range is

The examinable fact is that the threshold does not behave like a constant. The randomised trial that quantified it titrated 52 depressed patients to just above threshold and found a **12-fold** range in the minimum intensity needed to produce a seizure, with sex, age, placement and cumulative treatments each associated with it. A later summary of the wider literature puts the reported variation, between individuals and within one individual across conditions, at **as wide as 40-fold**, which is an upper bound and not a point value. One is the spread inside a single trial, the other across a literature; neither is the number.

In 402 patients receiving bilateral treatment with dose titration, threshold correlated with age and was slightly higher in men, while race, severity of illness, psychosis and psychotropic use did not affect it.

DETERMINANT	WHAT THE EVIDENCE SAYS, AND HOW IT BEARS ON DOSING
AGE	Positively correlated; a principal factor, and what the formulae are built on
ELECTRODE PLACEMENT	A principal factor; bilateral carried a higher initial threshold and a greater cumulative rise, so a threshold measured under one placement does not carry to another
SEX	Associated but weakly; men slightly higher. Enters some formulae, never sufficient alone
CUMULATIVE TREATMENTS	Associated; threshold rises across the course, forcing mid-course review of the multiple
MEDICATION AND ELECTRICAL PARAMETERS	Each contributes; pulse width, frequency and stimulus duration matter, so a formula holds only in the parameter set it came from
RACE, SEVERITY, PSYCHOSIS, PSYCHOTROPIC USE	Did not affect threshold in the large titrated cohort; never a reason to start higher

9.3 Setting the stimulus: the methods and their ranking

The current Indian Psychiatric Society practice guideline, published in 2023, names four ways of arriving at a dose and ranks them. **Stimulus titration** is recommended in regular practice, at the cost of a first session spent finding it. **Formula-based methods**, built on age and sex, guide the first dose of that titration rather than substitute for it. **Fixed high dosing** is reserved for patients with serious medical comorbidity in whom avoiding a subconvulsive stimulus is the priority. **Dosing from benchmark**, a high first dose then down-titration to the lowest dose still producing the same peak heart rate and tonic-clonic convulsion, is the better alternative where fixed high dosing is used.

The titration runs at different speeds: larger increments in elderly patients, smaller in adolescents and younger adults.

RULE **A formula estimates the first dose of a titration; it does not replace the titration.** A formula approximates across a population a quantity that is measured in an individual, which is why the guideline says to use it for the first dose and then titrate.

9.4 The half-age formula, and the size of its error

The **half-age method** estimates the stimulating dose from the patient's chronological age, using half that age as the percentage of charge on one device family, or the equivalent in millicoulombs on another, as the starting point at the first session. The device clause is part of the formula: the same numeral means a different delivered charge on a different device. The standing criticism is that it is a gross estimate, unaccommodating to individual variation in threshold and less sensitive than dose titration.

That criticism has been measured. In the large titrated cohort, half-age estimates averaged **18%** above the empirically determined threshold; the titrated treatment level and half-age agreed

exactly in only 14.7% of measurements; and titration gave a level more than 10 units higher than half-age in 33% of patients. Those two figures are measured against two different quantities, and reading them as two directions of one error is the mistake this paragraph exists to prevent. The overshoot of **18%** is half-age measured against the bare THRESHOLD, and it is the authors' own summary figure, taken from a passage this claim row does not quote. The shortfall in 33% of patients is measured against the titrated TREATMENT dose of 1.5 times threshold, with more than 10 units the margin that counts as a shortfall. A figure a fifth above the threshold is by construction below a level set at half again as much, so the two are one direction read off two benchmarks. What follows for practice is only that the formula overshoots the threshold on average, which is its residual use, since fewer stimulations are then needed.

The Indian guideline attaches a validity condition to every age-based or sex-based formula. The stimulus must have the same amplitude, pulse width, pulse frequency and electrode location as the derivation it came from, all four together, and medications and anaesthetic agents move the threshold in any case.

TRAP **Quoting a formula as though its number carried across devices, waveforms and drug regimens.** A formula is valid only inside the parameter set it was derived in, and the commonest error is to treat half the age as a universal constant rather than a percentage of charge on a named device family at a named pulse width.

9.5 Dose as a multiple of threshold, and why the multiple differs

The therapeutic dose is prescribed as a multiple of the measured threshold, and that multiple is not the same number everywhere. It is set by placement and pulse width, because the dose-response relation differs between them. In right unilateral treatment that relation is steep: stimulation at threshold has relatively low efficacy, and markedly suprathreshold dosing is what achieves efficacy over a course. In bilateral placements efficacy is already obtained marginally above threshold, which is where the guidelines set it, so raising a bilateral dose further is not where bilateral efficacy comes from; what a higher dose does to cognition belongs to this chapter's account of the cognitive cost. Carrying one direction across the placements, either way round, is how this topic is taught wrongly.

PLACEMENT AND PULSE WIDTH	INDIAN PSYCHIATRIC SOCIETY GUIDELINE (2023)	AUSTRALIAN AND NEW ZEALAND COLLEGE GUIDELINE (2019)
BILATERAL, BRIEF PULSE OF 1 MS OR MORE	Marginally suprathreshold, 1.5 times threshold	Effective at that level or just above, 1.5 times threshold
BILATERAL, LOWER BRIEF PULSE OF 0.5 MS	Some evidence suggests 2.5 times threshold	Not carried here
RIGHT UNILATERAL, BRIEF PULSE	4 to 6 times threshold	five to six times threshold, with a footnote recording 3 times as lower but still clinically meaningful
RIGHT UNILATERAL, ULTRABRIEF 0.3 MS	Strong evidence for 6 times threshold, chiefly in depression	Not carried here
BILATERAL, ULTRABRIEF 0.3 MS	Less effective and not advisable on existing evidence	Not carried here

The right unilateral brief-pulse cell is where the sources disagree. The Indian guideline of 2023 gives 4 to 6 times the threshold; the college guideline of 2019 gives five to six times, its footnote recording 3 times threshold as lower in efficacy but still clinically meaningful. One structural difference travels with that disagreement and the table above cannot show it: the Indian figures are stated under pulse-width conditions, while the college's two multiples are given in sentences that attach no pulse-width condition at all, so filing them in a pulse-width row is this chapter's arrangement and not the college's. Each belongs in an answer with its body and year attached. Averaging them would invent a figure neither states, and the lower bound stops markedly suprathreshold dosing being read as the only thing that works. One caution travels with the Indian multiples: the increment ought to be made at fixed amplitude, pulse frequency and pulse width, but most devices cap train duration and raise pulse frequency instead, so the guideline says its figures should be read with that in mind.

9.6 The titration session and its narrow exception

The guideline is explicit that markedly suprathreshold stimulation is not necessary on the first day of titration even for unilateral treatment, and might be considered only in extremely rare life-saving conditions such as neuroleptic malignant syndrome or intractable seizures. The shape of the exception matters: a life-threatening presentation can justify abandoning stepwise measurement for a dose that will certainly work.

A consistency note is owed. The Other psychotic disorders, delusional syndromes and catatonia notes have already told the reader that malignant catatonia is treated with liberal stimulus dosing to induce well-generalised seizures. The guideline sentence is consistent with that in shape, carving out the life-threatening case, but it names neuroleptic malignant syndrome and intractable seizures and not malignant catatonia. The earlier statement is therefore supported by analogy rather than by naming.

9.7 The threshold rises, and the dose can fall behind it

The threshold is not fixed even within one patient. The study that measured this re-titrated at the first and the sixth treatments in 62 depressed patients treated at moderately suprathreshold intensity. The threshold rose by approximately **47%** on average, but only 56% of patients showed any rise at all. Each part matters: the average rise is large enough to count, and nearly half showed none, so a rise is checked for rather than assumed. There it went with increasing age and not with gender, electrode placement or initial threshold, which sits in tension with the earlier trial's finding that bilateral treatment carried the greater cumulative rise. The tension is recorded, not resolved.

The clinical consequence is what gets examined. Therapeutic effect tracks the degree to which the stimulus exceeds the threshold, so if the threshold climbs while the delivered charge stays where the original titration set it, the relative stimulus intensity quietly falls. In a separate cohort, falls in relative stimulus intensity between the first and the sixth treatments were associated with diminished response at the sixth in unilateral treatment, which is where the steep unilateral dose-response predicts the drift will bite.

> **GOOD TO REMEMBER**
>
> If a responding patient stalls mid-course, ask whether the seizure is still adequate and whether the charge is still the multiple it was meant to be. Frequency of treatment is a separate lever: it buys speed of response at a cognitive price, with equivalent efficacy at the end of the course, and belongs to this chapter's account of the acute course and its schedules.

9.8 Restimulation: the missed seizure and the inadequate seizure

Failure at the trolley must be separated by kind, because each kind has its own rule. A **missed seizure** is no seizure at all: if there is no motor and no electroencephalographic seizure even after **20** seconds from completion of the stimulus, restimulation may be attempted at an increased dose, and a delayed-onset seizure should be watched for.

An **inadequate seizure** is a seizure that happened but was not good enough: a low-quality electroencephalographic seizure, or a motor seizure that did not generalise, limited to one side of the body or to the face. The Indian guideline directs restimulation at a higher dose after **45** seconds, and bounds the attempt as follows.

- **How many.** Restimulation may be attempted while the patient remains under the anaesthetic and the muscle relaxant, generally four to five times.
- **The cost of more time.** A top-up dose of anaesthetic can be requested to extend the window, but top-up doses reduce the quality of the seizure obtained.
- **When not to chase.** If the seizure is very brief, of the order of 5 to 10 seconds, and the patient is showing the expected clinical response, restimulation need not be administered.

The delay before the second stimulus is the second disagreement, and each source is stated as it stands. The Indian guideline of 2023 gives 45 seconds. The college guideline of 2019 offers 60 to 90 seconds as an example rather than a rule, and gives the reason the delay is bounded above as

well as below: if it is too long a top-up dose becomes necessary, and the resulting seizure could be poor, negating the benefit while potentially worsening cognition. Neither figure is preferred here.

The sources agree at the other end: restimulation on the same day after an **adequate seizure**, which is multiple monitored electroconvulsive therapy, is not recommended by the Indian guideline, and the college guideline holds that delivering more than one adequate seizure per treatment session is not acceptable practice except in a session where threshold is being determined.

IN INDIA

Indian practice is governed by the Indian Psychiatric Society's current guideline, published in 2023: titration is recommended, the formula is only a starting point for it, quality outranks duration, and the classical numeric criteria are named as retired. Where a college guideline gives a different figure, as for the right unilateral multiple and the restimulation delay, name both with their bodies and years.

9.9 The adequate seizure, and the limits of duration

Adequacy is judged on the motor seizure, the electroencephalographic seizure and what follows, with duration the weakest signal rather than the definition. **Motor seizure duration** is timed by an explicit convention: the time from the beginning of the stimulus until the end of the last clonus in any part of the body, which usually ends in the limbs. A good-quality motor seizure moves through induction during or soon after stimulation, tonic contraction, bilateral convulsions, a gradual ending, a comatose stage, then return of consciousness.

The ictal electroencephalogram runs in numbered phases, and it is morphology rather than length that carries the information.

- Phase 1, recruitment: high-frequency waves of gradually increasing amplitude.
- Phase 2: hypersynchronous high-amplitude polyspike bursts at around 10 Hz lasting 10 to 20 seconds, corresponding to tonic contraction.
- Phase 3: hypersynchronous polyspikes intermixed with slow waves for 20 to 40 seconds, the tonic-clonic phase.
- Phase 4, **postictal suppression**: a flat isoelectric line, whose onset heralds the end of the seizure.
- Phase 5, recovery: delta to theta to alpha waveform.

Generalisation is read off the same trace: a good-quality seizure shows activity in the contralateral channels, which is the criterion the restimulation rules turn on.

MNEMONIC

Generalised, Morphology, Suppression, then Duration

Judge the seizure in that order: whether it **Generalised**, in the limbs and the contralateral channels; whether the **Morphology** moved through its phases; whether **Suppression** followed, the flat isoelectric phase whose onset marks the end; and only then how long it lasted, because **Duration** is the weakest signal.

The limit on duration is stated by the guideline itself. A good quality seizure should be given more importance than any specific duration, and a good quality seizure even of shorter duration has been demonstrated to be efficacious in elderly depressed patients. The classical numeric definition, still a common exam key, is named and retired in one breath: the earlier definition of 15 seconds of motor seizure and 25 seconds of electroencephalographic seizure is found to be of little clinical or prognostic benefit. No replacement figure is offered and none is invented here: the question has been demoted, not re-answered.

PITFALL

Reciting the retired numeric criteria as the current definition of an adequate seizure. They are worth knowing because they are still asked, but the guideline finds them of little clinical or prognostic benefit. Write them as the earlier definition, say that quality rather than a new number replaced them, and invent no replacement.

TRAP **Treating a generalised seizure as proof that the treatment was adequate, and any seizure as proof that the dose was right.** A generalised seizure is necessary for a therapeutic effect and not sufficient for one, which is why the dose is a multiple of threshold rather than whatever charge produced a convulsion. The paired error is to restimulate a patient who has already had an adequate seizure to chase a longer one.

The seizure threshold is the lowest electrical dose that produces a seizure in this patient at this session; it varies enormously between patients, rises across a course in only some of them, is best measured by titration with a formula used only to pick the first dose, and carries a treatment dose set as a multiple sized by placement and pulse width rather than by one rule. Adequacy is judged by generalisation, electroencephalographic morphology and postictal suppression, duration being the weakest signal.

10 · Modified versus unmodified (direct) ECT

Modified electroconvulsive therapy is the treatment given under anaesthesia with a muscle relaxant, and in India it is the only lawful form of it. The contrast with the unmodified or direct technique is therefore not a preference between two ways of doing the same thing. It is the difference between current practice and a practice the statute has closed, and a candidate who treats it as a matter of resources or of taste has misread the strongest legal statement in this chapter. What is examinable is the mechanical reason modification exists, what the unmodified

technique cost the patients who received it, and the fact that the bar on it is legal rather than advisory.

Owned here: the technique contrast, why the unmodified form was once used, what it cost in injury and in the patient's experience of being treated, what modification does and does not abolish, and the settled Indian legal position. Named but not re-derived: the anaesthetic agents, the muscle relaxant, the airway and the equipment of the room, which belong to this chapter's account of the pre-ECT workup and anaesthesia; the introduction of anaesthesia and muscle relaxation as historical events, to its account of the history and evolution of the treatment; the seizure threshold, the stimulus dose and every criterion of seizure adequacy, to its account of stimulus dosing; consent as it is actually taken, to its account of consent and the legal aspects; and the prohibiting provision itself, with its number, its wording and its classification, to the Mental health legislation and forensic psychiatry notes.

2 per 100,000 FRACTURE RISK PER ECT UNDER MODIFIED TECHNIQUE	**Unlawful** THE STANDING OF UNMODIFIED ECT IN INDIA, NOT MERELY INADVISABLE	**86 against 50** PER CENT CONSENTING TO A FUTURE COURSE, MODIFIED AGAINST UNMODIFIED

10.1 What modification actually modifies

Modification adds two things, an anaesthetic and a muscle relaxant, and of the two it is the relaxant whose target is the convulsion rather than the treatment. Both are required, and the statutory bar admits no reading on which relaxant alone would count as modified. The rationale runs in three steps. First, during the motor seizure the muscles of the trunk and limbs contract forcefully and repetitively, predisposing to injuries to muscles, joints, teeth and bones. Second, that motor seizure is irrelevant to the therapeutic action of the treatment. Third, it is therefore abolished by an intravenous muscle relaxant given after the anaesthesia in the ECT premedication. The middle step carries the whole argument: nothing therapeutic is surrendered when the convulsion is removed, so modification is not a concession to comfort or to appearances. It deletes the mechanism of injury and leaves the mechanism of benefit standing.

Two consequences are worth holding apart. The first is anatomical. What the relaxant abolishes is the **motor seizure**, the visible convulsion of trunk and limb; the cerebral seizure the stimulus induces is untouched by it, and a patient whose limbs are still is still having the seizure that treats them. That is why modification does not weaken the treatment, and why an isolated-limb tourniquet exists as a way of watching a convulsion that has been abolished everywhere else. The second is procedural. The relaxant follows the anaesthetic rather than preceding it, and the agents on that trolley, their doses, their contraindications and the airway conduct around them belong to this chapter's account of the pre-ECT workup and anaesthesia, which owns the tourniquet method too and which names the same relaxant this account's source names, succinylcholine.

10.2 What the unmodified technique was, from an Indian record of it

Unmodified, or direct, electroconvulsive therapy is the same stimulus given with neither anaesthesia nor a relaxant, so the generalised convulsion runs its course in the patient's body. It

appears in this chapter in two capacities only, as a historical practice and as an unlawful one, and it is described here rather than gestured at because a description written from the modern side alone softens into a list of theoretical hazards. The record used is a retrospective review of **637 patients treated between 1990 and 1995** at an Indian institute. Nothing in the ledger establishes that its author worked that service, and the receipt in fact shows the cohort reported from a different institution than the one where the patients were treated, so the record's authority rests on what it documents rather than on the author's proximity to it.

The technique is recorded plainly. All the patients were given ECT with an unmodified, bitemporal, sine wave device; there were two doctors, a junior and a senior resident, with multiple nursing and ward staff, but no anaesthetist; Boyle's apparatus was used for mandatory pre-ECT oxygenation and after care; and careful manual restraining of patients during the convulsion was done at all vulnerable joints by multiple ward staff and trainee nurses. The last of those is the sentence to remember. **Manual restraint** at the vulnerable joints was the physical substitute for a muscle relaxant, and it is the reason joint and bone injury is the characteristic harm of the technique. Bone rather than long bone: the injuries this record actually names are a cervical vertebral compression fracture, a dental extraction, a jaw dislocation and back muscle spasm, and not one of them is a long bone. Force applied across a joint against a muscle contracting maximally is not only a safeguard; it is a second path by which load reaches the same bone.

Why the technique was used at all is answered by the arrangement that record describes rather than by any argument advanced in its favour. The treatment came first and its modification came later, a chronology this chapter's account of the history and evolution of electroconvulsive therapy carries; until an anaesthetist could be put in the room, what remained available was the original technique. The series shows that economy exactly: a separate ECT station in each ward, a spacious room holding one ECT table, the ECT machine, Boyle's apparatus and an attached recovery space, staffed by residents, nurses and ward staff. Treatment could be given at ward level, in volume, without competing for anaesthetic manpower or theatre time. No row in this claim list states that rationale in so many words, so it is read off the arrangement and named as an inference rather than asserted as a finding.

10.3 What it cost: the injuries, case by case

The same series lists the injuries that ended a course, and they fall into four families that map exactly onto the tissues the rationale above named.

- **Bone.** Surgical emphysema developed in a 26-year-old woman in whom the radiograph taken after ECT revealed a compression fracture of the fourth cervical vertebra, managed conservatively with a cervical collar.
- **Tooth.** A traumatic dental extraction occurred at the first ECT session in a 45-year-old woman with an acute psychosis.
- **Joint.** Recurrent jaw dislocation occurred in a 28-year-old woman with recurrent mania, who had a past history of safe treatment with ECT.

- **Muscle.** Severe back muscle spasm, with no fracture on radiography, was the reason ECT was discontinued in two more young male patients.

The list teaches more than the injuries themselves. The patient with recurrent jaw dislocation had been treated safely in the past, which disposes of the intuition that a course completed without incident predicts the next one: this risk is per session and mechanical, not a settled property of the person. The dental extraction happened at the very first session, so no accumulation of treatments was needed to produce it. And the back-spasm cases carried no radiological fracture at all, which is the reminder that a normal film does not mean an uninjured patient.

10.4 The rate that series reported, and the caveat that never leaves it

The same paper reports its overall outcome, and the two halves of that report are quoted together or not at all. **637 patients, 6.94 per cent of total admissions, received ECT with meticulous standard of care except the provision of anaesthesia; satisfactory improvement was noted in 95.45 per cent of patients, with no noticeable or reported complication in 89.05 per cent, and premature termination of ECT for complications in 2.19 per cent**. Read on its own, that is the most misusable paragraph in this chapter.

Its author does not leave it on its own. Regarding complications, he writes, there must be under-reporting for not having any screening tool, only the noticeable side effects having got recorded in the files; even so, the serious complications were much less than those reported prior to the 1960s. The consequence is exact rather than rhetorical. **Under-reporting** of that kind makes a complication-free figure an upper bound on safety and not a measurement of it, because the numerator was assembled from whatever happened to be noticed by a busy ward. Musculoskeletal complications were recorded in ten patients, of whom five were severe enough to stop the course, and no film was taken in the rest, which limits the fracture count in the same direction. The series is admitted here for what it establishes about a historical practice. It is not evidence about current practice and it does not qualify the statutory bar by any degree.

TRAP **Quoting the complication-free figure from this series as though it showed the unmodified technique to be acceptably safe.** The author says in the next breath that complications were necessarily under-reported, so the figure bounds what was noticed rather than what occurred. And a perfectly measured figure would settle nothing here, because the technique is barred by statute: its measured harm rate is a fact about history, never an argument about what to do on Monday.

10.5 What it was like for the patient

Injury counts are not the whole cost, and the comparison that reaches the rest was made prospectively in 100 euthymic bipolar patients, 50 of them treated with modified and 50 with unmodified ECT. The majority of all patients, 78 per cent, were satisfied with treatment and 88 per cent with the outcome, without significant differences between the modified and unmodified groups; forgetfulness at 70 per cent and headaches at 57 per cent occurred in all groups, the only significant difference in forgetfulness being that it was reported by more manic patients treated with unmodified ECT. Stop before that final clause and the difference between the techniques

looks like a matter of decorum, which is why the clause is carried: the one adverse-effect difference the source reports runs against the unlawful technique.

The difference sits in what the patients feared and in what they would agree to again. Depressive and manic patients treated with unmodified ECT reported concerns of brain damage and of physical harm significantly more frequently, and **while 86 per cent of patients treated with modified ECT consented to a future treatment, this fell significantly, to 50 per cent, after unmodified ECT**. That is the shape of the finding and it should be kept: satisfaction did not separate the groups, willingness to go through it again did, and by a wide margin. The cohort is Turkish, from a country that mandated modified ECT in 2005, so it is carried for the direction of its finding rather than as an Indian figure.

10.6 What modification buys, and the one place it does not reach

Well-modified treatment produces markedly diminished skeletal muscle contraction and therefore minimal skeletal and dental risk, and the population figures are small enough to be worth quoting exactly. **The fracture risk with modified ECT is 2 events per 100,000 ECTs, and on recent data alone the risk may be as low as 0.36 events per 100,000 ECTs; the dental fracture risk with modified ECT is 0.02 per cent per ECT and 0.17 per cent per ECT course**. Those figures are what make the older injury reports legible. They describe the same treatment with the motor seizure taken out of it.

The residual risk is not spread evenly and part of it is in the operator's hands. Pre-existing bone and dental disease increase the risk; good seizure modification, proper use of bite blocks and effective jaw immobilisation during ECT reduce it. That the **bite block** and **jaw immobilisation** appear on a list of what protects a patient under modern technique is the tell: the jaw is the one place the relaxant does not reach, because the masseter is driven by direct stimulation of the muscle rather than through the neuromuscular route the relaxant blocks. **Seizure modification** is accordingly a quality with degrees rather than a box that has been ticked, and the equipment and airway conduct that deliver it belong to this chapter's account of the pre-ECT workup and anaesthesia.

> **GOOD TO REMEMBER**
>
> Quote the fracture figures as what they are, population rates under well-conducted modified technique. They do not transfer to a patient with known bone or dental disease, in whom the same review states the risk is higher, and the assessment that finds that disease before a course belongs to the pre-ECT workup rather than to this contrast.

10.7 The Indian position: unlawful, not inadvisable

In India the question of modified against unmodified treatment was settled by Parliament rather than by preference. The statute prohibits electroconvulsive therapy given without muscle relaxants and without anaesthesia. The provision that says so, its number, its text and its place in the scheme of prohibited and restricted procedures belong to the Mental health legislation and forensic psychiatry notes, and are named here rather than renumbered, because a chapter that

reprints a section number it did not itself ground is how a legal fiction enters a syllabus. What this account states is the consequence for technique, and it is blunt. Direct treatment is not a rougher version of the same thing that a hard-pressed unit falls back on; it is unlawful. The bar the legislation notes carry admits no exception, so no degree of urgency turns the direct technique into something a clinician is entitled to weigh.

IN INDIA

The working form of that rule in a district hospital is a timetabling rule and not a clinical one. Where no anaesthetist is available today, what changes is the day of the treatment and never the technique of it. The statutory conditions attached to consent for ECT, including who consents for whom, sit with this chapter's account of consent and the legal aspects and with the Mental health legislation and forensic psychiatry notes. What belongs here is the narrower point that the law fixes the technique itself, not merely the paperwork around it.

RULE **In India the modified technique is not the better of two ways to give electroconvulsive therapy; it is the only lawful one.** Everything the unmodified technique is taught for is historical: what it was, what it injured, and what it felt like to receive. Write it in the past tense, name the statutory bar in words and attribute it to the Mental health legislation and forensic psychiatry notes, and never write a sentence in which a clinician is choosing between the two.

AXIS	MODIFIED ECT	UNMODIFIED (DIRECT) ECT: HISTORICAL, AND UNLAWFUL IN INDIA
STANDING TODAY	The lawful form of the treatment	Prohibited by statute, per the Mental health legislation and forensic psychiatry notes
WHAT HOLDS THE BODY	An intravenous muscle relaxant, given after the anaesthesia	Manual restraint at all vulnerable joints by ward staff
MOTOR SEIZURE	Abolished; the cerebral seizure is untouched	Ran to completion in the trunk and limbs
SKELETAL AND DENTAL COST	2 skeletal fractures per 100,000 ECTs, with dental fracture a separate and much commoner cost at 0.02 per cent per ECT, which is an order of magnitude above the skeletal figure and is the one to compare against the opposite cell	Cervical compression fracture, dental extraction, jaw dislocation, back spasm
WOULD ACCEPT IT AGAIN	86 per cent	50 per cent

MNEMONIC

SLEEP, SLACK, SHIELD

Sleep is the anaesthetic and **slack** the muscle relaxant that abolishes the motor seizure; the statute requires them, and their absence is precisely what the word unmodified meant. **Shield** is the bite block with effective jaw immobilisation, which good practice adds because the masseter contraction is driven directly by the stimulus and not through the junction the relaxant blocks. The agents behind the first pair belong to the pre-ECT workup and anaesthesia account, never to this one.

Modification abolishes the motor seizure and nothing therapeutic: the relaxant follows the anaesthetic, the cerebral seizure is untouched, and the injuries of the unmodified technique were mechanical, running from cervical spine to tooth to jaw to back muscle. In India the modified form is not the safer choice but the only lawful one, and the provision that says so is named in words, never renumbered, from the Mental health legislation and forensic psychiatry notes.

Electroconvulsive therapy: effects, consent and course

11 · Cognitive side effects of ECT (anterograde and retrograde amnesia)

The cognitive cost of electroconvulsive therapy is the adverse effect that decides whether a patient consents, whether a course is finished and whether the treatment keeps its place in practice, and it is not one deficit but several running on different clocks. Merging them is the commonest error here: the evidence that one recovers comes from a corpus that could not measure another, so a candidate who has fused them will quote a real finding about the wrong phenomenon. This account holds them apart, gives the modifiers and the monitoring, and reports the persistence question as contested.

Owned here is the cognitive cost in full: the three phenomena, patient-reported memory loss, the modifiers, the monitoring, and the contested status of persistent retrograde deficit. Named and not re-taught, each owned by another account in this chapter: what each placement is and waveform, by technique and electrode placement; the efficacy-against-cognition trade, by the placement comparison; the threshold and stimulus dose, by stimulus dosing; frequency and the schedule, by the acute course; a confusional state past its window, by complications; concurrent drugs, by drug interactions.

5 to 45 minutes	29 percent to 55 percent	6 to 12 months
ORIENTATION GENERALLY RETURNS WITHIN THIS WINDOW AFTER AN ADEQUATE SEIZURE	PATIENTS REPORTING PERSISTENT MEMORY LOSS ON THE STRICT DEFINITION	FOLLOW-UP OVER WHICH AUTOBIOGRAPHICAL LOSS STAYED STABLE, NOT RECOVERING

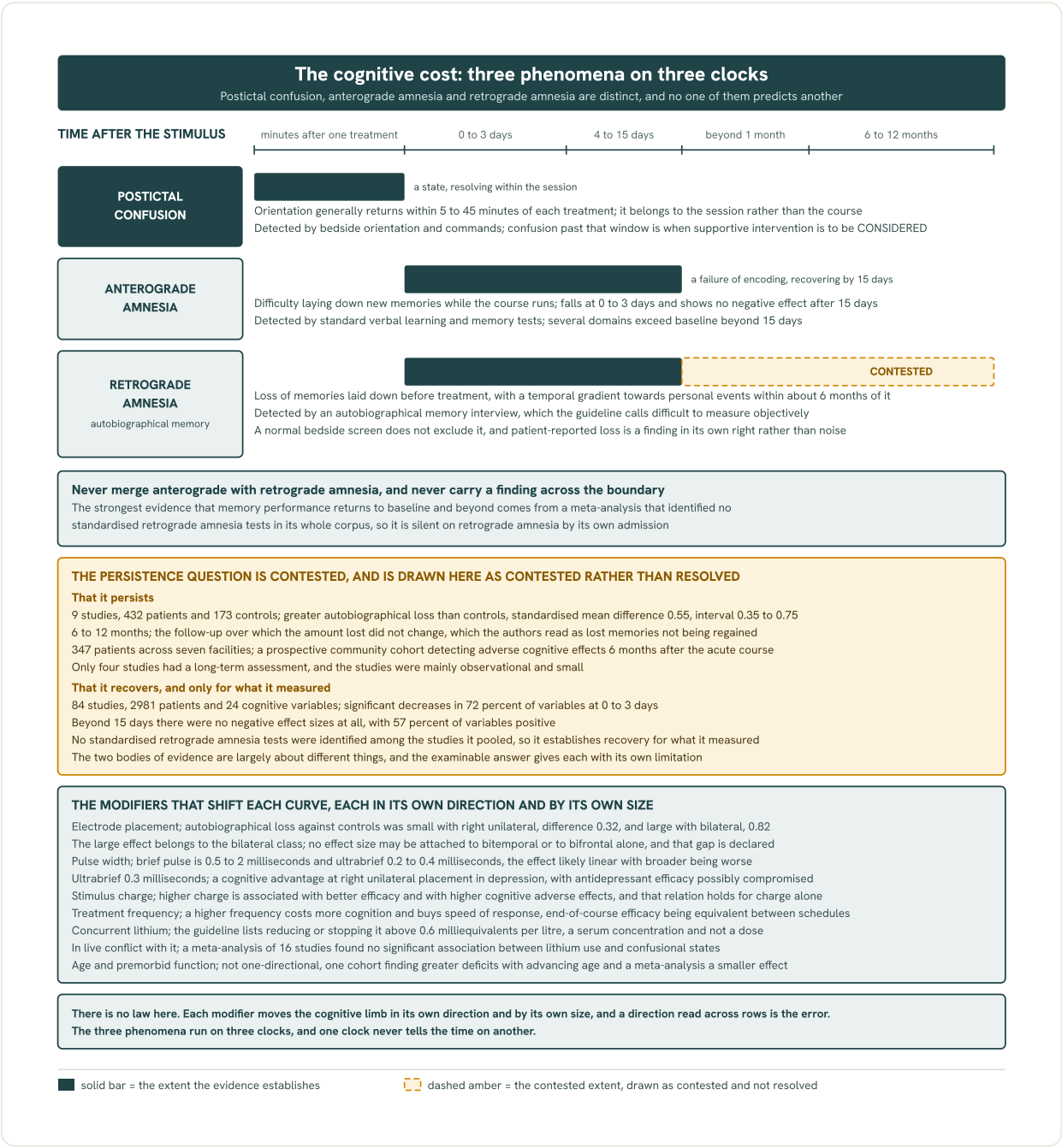


Fig 27.8. The cognitive cost is three phenomena on a common time axis and not one deficit: postictal confusion, a state resolving within minutes of each treatment; anterograde amnesia, a failure of encoding that falls in the first days after a course and recovers with several domains exceeding baseline; and retrograde amnesia, a loss of what was already encoded, with a gradient towards events near the treatment, hard to measure, and of contested persistence, the plate marking that persistence as contested rather than settling it, while each modifier shifts its own limb in its own direction and by its own size.

11.1 Three phenomena, not one deficit

They separate once asked what was lost and when it was laid down. **Postictal confusion** is the disorientation of the minutes after each induced seizure, expected after every treatment and belonging to the session rather than the course. **Anterograde amnesia** is difficulty laying down new memories while the course runs, so the treatment period itself becomes patchy. **Retrograde amnesia** is loss of memories laid down before treatment, and its most studied form is **autobiographical memory**, the record of the patient's own life events. A state, a failure of

encoding, and a loss of what was already encoded: no one predicts another, and a patient can pass a bedside screen and still describe a hole in the year before treatment.

RULE **Never merge anterograde with retrograde amnesia, and never carry a finding across the boundary.** The strongest evidence that memory performance returns to baseline and beyond comes from a meta-analysis that identified no standardised retrograde amnesia tests in its whole corpus. It is silent on retrograde amnesia by its own admission, and quoting its headline as reassurance about lost life memories is the most examinable misuse of evidence here.

AXIS	POSTICTAL CONFUSION	ANTEROGRADE AMNESIA	RETROGRADE AMNESIA
TIME COURSE	Orientation generally returns within 5 to 45 minutes of each treatment	Falls at 0 to 3 days, no negative effect after 15 days	Measurable from the end of the course, with a gradient towards recent events
HOW IT IS DETECTED	Bedside orientation and commands	Standard verbal learning and memory tests	An autobiographical memory interview; the guideline calls it difficult to measure using objective cognitive tests
PROGNOSIS	Resolves within the session	Recovers, several domains exceeding baseline	Contested, and reported as contested

11.2 Postictal confusion, and where it stops being normal

Confusion after an induced seizure is part of the procedure until it outlasts its window. The current Indian guideline states that patients generally regain orientation within 5 to 45 minutes after an adequate seizure, and that confusion with poor response to commands, with or without behavioural agitation, continuing past that period or very severe within it, is when supportive intervention is to be CONSIDERED, which is the guideline's own strength and not a requirement. The window is both reassurance and alarm; the intervention belongs to the account of complications.

Confusion is not one state either. The cleanest separation comes from a prospective cohort of 145 adults aged 55 or over treated for a major depressive episode; the population is part of the number, and these are not general adult figures. At least one subtype occurred in **55.9 percent**. Postictal agitation, at 29.5 percent, and **prolonged time to reorientation**, at 28.3 percent, were common; postictal delirium, at 5.9 percent, and **interictal delirium**, arising between treatments rather than after one, at 4.2 percent, were not. Recurrence separated them further, which changes the plan for the next treatment: 66.7 percent for interictal delirium and 50.0 percent for prolonged reorientation, against 12.5 percent for postictal delirium. The authors note that their instruments were not designed to measure subtypes during a course.

11.3 Anterograde amnesia: the deficit that recovers

This limb has the strongest evidence and the most reassuring answer, and it reassures only about itself. A meta-analysis of 24 cognitive variables across 84 studies and 2981 patients found

significant decreases in **72 percent** of variables at 0 to 3 days. From 4 to 15 days all but one confidence interval included zero or showed a positive effect, and beyond 15 days there were no negative effect sizes at all, with 57 percent of variables positive; processing speed, working memory, anterograde memory and some executive functions improved beyond baseline.

A domain map of 91 studies and 3762 individuals across 11 cognitive domains gives the same shape with the domains named: no significant change in global cognition on a bedside screen within 24 hours of the last treatment, small to medium decreases at 1 to 28 days in autobiographical memory, verbal fluency and verbal memory, and improvement on almost all domains beyond 1 month. The deficit is therefore domain-specific rather than global, and a bedside screen is the wrong instrument for finding it.

11.4 Retrograde amnesia: the distinct phenomenon

This is the limb patients care about and tests are worst at. The Indian guideline says both halves in one sentence: it is among the most distressing adverse effects, and difficult to measure using objective cognitive tests. A normal bedside screen therefore does not exclude it.

Its shape is not random. A systematic review across 15 studies established a **temporal gradient**: the memories predominantly affected are of personal events near the treatment, within about 6 months of it, rather than of the distant past. The same review names three technique features that reduce autobiographical loss, each taught elsewhere in this chapter: a brief pulse rather than a sine wave stimulus, unilateral rather than bilateral placement, and titration of the current against the patient's own seizure threshold.

The reviewers add a confound that travels with the whole literature: the direct effect of the depressive state on memory is not controlled for, a depressed patient recalling their own life poorly before any treatment. That is why the size of the effect is hard to pin down, and it is not a reason to discount a patient's account.

11.5 The persistence question, which stays contested

Whether retrograde deficit persists must not be answered crisply. Two bodies of evidence point in opposite directions, each honest about its limits, and both are set out rather than adjudicated.

That it persists. A meta-analysis of 9 studies, 432 patients and 173 controls found greater autobiographical loss in treated patients than controls, a moderate pooled standardised mean difference of 0.55, 95 percent confidence interval 0.35 to 0.75. The decisive finding is stability, not size: the amount lost did not change between the end of treatment and follow-up at 6 to 12 months, which the authors read as lost memories not being regained. Their limits travel with it: only four studies had a long-term assessment, so none was possible on long-term effects, and the studies were mainly observational and small. The second pillar is a prospective community cohort of 347 patients across seven facilities: it detected adverse cognitive effects 6 months after the acute course, with bilateral placement giving more severe and persisting retrograde amnesia than right unilateral.

That it recovers. The recovery meta-analysis is the strongest statement on the other side, and its bearing here is bounded by a sentence in its own results: no standardised retrograde amnesia tests were identified among the studies it pooled. It establishes recovery for what it measured, not for a variable nobody measured. The two bodies of evidence are largely about different things.

TRAP **Answering "does memory loss after electroconvulsive therapy persist?" with a single word.** "No" quotes a recovery result from a corpus with no retrograde instrument in it. "Yes" states as settled a finding resting on a handful of long-term assessments in observational studies. The examinable answer gives each side with its limitation and says the question is open.

11.6 Patient-reported memory loss, a finding and not noise

Patient-reported memory loss diverges from measured loss, and the divergence is itself what must be taught. In the review above, objective measures placed autobiographical loss under 6 months after treatment while subjective accounts placed it beyond 6 months. A descriptive systematic review of 35 studies of patients' own views found at least one third reporting persistent memory loss, rates on the strict definition running from 29 percent to 55 percent, and the rate did not depend on whether the study was clinician-led or patient-led. It concluded that the professional statement for patients then current, holding that over 80 percent were satisfied and that memory loss was not clinically important, was unfounded. That review is dated 2003 and its target is a statement of that period, not a current college position.

The Indian guideline gives the practice rule directly: subjective assessment of memory impairment is to be given due importance alongside objective assessment and to be part of routine cognitive assessment. Its explanation of why report exceeds measurement is a caution about instruments, not about patients: some cognitive domains are difficult to assess reliably and sensitively with standard tools, which can lead to higher subjective reporting. It names four contributors: that insensitivity; functional impact larger than measured severity; decline belonging to the illness and becoming visible once insight returns; and preoccupation with psychopathology.

RULE **Where patient report and objective testing diverge, the divergence is the finding, not a reason to discount the report.** The guideline offers its contributors as explanations of the gap and never as grounds for dismissal, and the first of them says the tests are insensitive. An answer that files persistent memory complaints under "subjective" and moves on has failed the item.

11.7 The modifiers that shift each curve

Each modifier moves the cognitive limb in its own direction and by its own size. The relation established for one parameter is the relation for that parameter, and reading a direction across rows is the error this chapter prevents.

MODIFIER	WHAT IS GROUNDED ABOUT ITS COGNITIVE EFFECT	REST OF IT TAUGHT BY
ELECTRODE PLACEMENT	Against controls, autobiographical loss was small with right unilateral, standardised mean difference 0.32, interval 0.06 to 0.57, and large with bilateral, **0.82, interval 0.49 to 1.15**. Bilateral is the source's own word and names a class. The Indian guideline separately holds bifrontal to have lesser cognitive effects than bitemporal in mania, depression and schizophrenia, and right unilateral lesser cognitive adversities	Technique; the placement comparison
PULSE WIDTH	Brief pulse is 0.5 to 2 milliseconds, **ultrabrief pulse** 0.2 to 0.4 milliseconds; the effect is likely linear, broader being worse. An ultrabrief pulse of 0.3 milliseconds has a cognitive advantage over brief pulses with right unilateral placement in depressive disorders, the guideline adding in the same breath that antidepressant efficacy may be compromised	Technique and waveform
STIMULUS CHARGE	Higher charge is associated with better efficacy and higher cognitive adverse effects, with the guideline's caution that charge is not a linear measure but a combination of several electrical parameters. The relation holds for charge alone	Stimulus dosing
TREATMENT FREQUENCY	A higher frequency costs more cognition. It buys speed of response and is not an efficacy lever, end-of-course efficacy being equivalent between the schedules	The acute course
CONCURRENT LITHIUM	The guideline lists reduction or stopping of drugs such as lithium, at serum levels above 0.6 milliequivalents per litre, among the steps for cognitive effects. A serum concentration, not an administered dose, and in live conflict with the meta-analysis below	Drug interactions

MODIFIER	WHAT IS GROUNDED ABOUT ITS COGNITIVE EFFECT	REST OF IT TAUGHT BY
AGE AND PREMORBID FUNCTION	Not one-directional. The community cohort found greater deficits with advancing age, lower premorbid intellectual function and female gender; the autobiographical meta-analysis found higher age associated with a smaller effect; the domain map found verbal fluency worse in younger adults	Special populations

The lithium row is a genuine conflict and is written as one. A meta-analysis of 16 studies found no significant association between lithium use and confusional states on its primary model, an odds ratio of 2.09 reaching significance at 2.38 only on a sensitivity model with a different estimator, and no significant differences in global cognition, autobiographical memory or verbal fluency. Its authors state that current evidence does not show a significant increase in cognitive side effects and that methodological limitations preclude definitive conclusions. Both are reported.

PITFALL

Narrowing the placement figure from bilateral to bitemporal. The large effect belongs to the bilateral class, and bifrontal is itself a bilateral placement, so the pooled corpus is not bitemporal only. No effect size may be attached to bitemporal or to bifrontal individually from this evidence, and the source reports no within-class breakdown; the gap is declared, not filled. The guideline's separate statement that bifrontal is gentler than bitemporal neither corrects nor is corrected by a meta-analysis that never separated them.

11.8 How cognition is monitored across a course

Monitoring starts before the first treatment. The Indian guideline requires a **baseline cognitive assessment**, so later change can be attributed, then sorts instruments by what they see. The Hindi Mental Status Examination and the Mini Mental Status Examination are named as simple tools not sensitive to the subtle change associated with treatment; the Montreal Cognitive Assessment battery and the Brief ECT Cognitive Screen as used internationally; the Battery for ECT-Related Cognitive Deficits as validated in the Indian population and recommended at initiation and across the course. Instruments are named only; no content, scoring or cut-off is reproduced.

The cadence is stated: progress in clinical symptoms and in cognitive and other adverse effects is monitored at least once a week while the course runs. A second, differently sourced standard is a United Kingdom technology appraisal of 2003 requiring cognitive function to be monitored on an ongoing basis and at a minimum at the end of each course. Two provenance facts travel with it: its recommendations on depression were replaced in 2009 by the national depression

guideline, while those on catatonia, prolonged or severe manic episodes and schizophrenia have not changed.

What is monitored matters as much as how often: the domain map states that a bedside global screen has extremely limited utility for tracking change, and recommends that assessment cover autobiographical memory, verbal fluency and verbal memory.

IN INDIA

The Indian position is more specific in two examinable ways. It names a battery validated in the Indian population and recommends it at initiation and through the course, rather than leaving instrument choice open. And it directs that patient-reported impairment be given due importance alongside objective testing, making the patient's account a required part of the record.

11.9 What is done when cognitive effects appear

The Indian guideline gives a seven-step menu for cognitive adverse effects during a course, used singly or in combination. Each step is a cognitive-management decision here; the parameter behind it is taught by the account named above.

1. Space the sessions out.
2. Switch to unilateral placement.
3. Switch to a briefer pulse width.
4. Reduce the stimulus dose.
5. Reduce or stop drugs known to affect cognition, such as lithium above 0.6 milliequivalents per litre.
6. Reduce the anaesthetic where it has been given high.
7. Terminate the course where the risks outweigh the benefits.

The guideline attaches a caution that makes this a judgement rather than a checklist: the decision is balanced against the possible reduction in effectiveness these steps carry. A patient made cognitively comfortable and left depressed has not been helped.

IN PRACTICE

Take a baseline before the first treatment, so a later complaint has something to be compared against. Ask about life memories specifically, not only orientation and word lists: the instrument that finds retrograde loss is a conversation about the patient's own past. And when a patient reports a hole the tests do not show, record it as a finding.

MNEMONIC

THREE CLOCKS

Minutes for postictal confusion, orientation generally back within 5 to 45 minutes. **Days to weeks** for anterograde amnesia, worst at 0 to 3 days and recovered beyond 15 days. **Months, and contested** for retrograde amnesia. One clock never tells the time on another.

The cognitive cost is three phenomena on three clocks: postictal confusion, resolving within minutes of each treatment and a complication only when it outlasts its window; anterograde amnesia, measurable in the days after a course and recovering to baseline and beyond; and retrograde amnesia, predominantly for events near the treatment, hard to measure, and of contested persistence, the two sides being a stable autobiographical loss at long-term follow-up and a recovery meta-analysis with no retrograde instrument. Patient-reported loss is a finding in its own right, assessed routinely and weighed alongside testing. The risk is shifted by placement, pulse width, charge, frequency, concurrent medication, age and baseline function, each in its own direction. Cognition is assessed at baseline and watched at least weekly with a sensitive instrument, and the seven-step menu is what is done when the cost appears.

12 · Other complications and adverse effects of ECT (cardiovascular, prolonged seizure)

The complications of electroconvulsive therapy are the events that occur during and after a treatment that was correctly indicated and correctly given. They are a different object from the conditions that make a patient risky before the first stimulus, and they are examined separately: one question asks what happens in the suite and the recovery room and what is done about it, the other asks whom you should hesitate to treat. This account answers the first.

Owned here: the cardiovascular sequence and its arrhythmia and ischaemia risk, the cardiac-event and mortality estimates, prolonged and tardive seizures, postictal agitation and delirium, headache, muscle soreness and nausea, dental and musculoskeletal injury, anaesthetic complications, and the emergence of mania on treatment. Named here and taught elsewhere in this chapter: the conditions that raise risk beforehand, by contraindications and high-risk conditions for ECT; the induction agents, relaxant and airway, by pre-ECT workup and anaesthesia; the threshold and the stimulus dose, by stimulus dosing, seizure threshold and adequate seizure criteria; the drugs that move the threshold, by drug interactions with ECT; and the whole cognitive limb, anterograde and retrograde amnesia, by cognitive side effects of ECT.

1 in 50	180 seconds	2.1 per 100,000
PATIENTS DEVELOPING A MAJOR ADVERSE CARDIAC EVENT OVER A COURSE	THE INDIAN DEFINITION OF A PROLONGED SEIZURE, WHICH IS NOT THE POINT AT WHICH TERMINATION IS ATTEMPTED	DEATHS ATTRIBUTED TO THE TREATMENT, PER TREATMENT GIVEN, IN THE POOLED INTERNATIONAL REVIEW

12.1 The cardiovascular sequence, in its fixed order

The cardiovascular effect is not one event but a sequence produced by autonomic nervous system activation, and the order is the examinable part. **Parasympathetic discharge** comes first: an initial **10 to 15 seconds** of vagal outflow in the tonic phase, with bradycardia and occasional asystole. The **sympathetic surge** follows in the clonic phase, a pronounced response of hypertension, tachycardia and other arrhythmias, peaking **1 minute** after the stimulus and generally resolving within **5 to 10 minutes**.

Bradycardia in the seconds after the stimulus is expected physiology, not a surprise, which is why antimuscarinic premedication is decided in advance, agent and dose belonging to the workup and anaesthesia account. The same activation raises cerebral metabolic rate, cerebral blood flow and intracranial pressure, and intraocular and intragastric pressure with them; what that means in a patient who already has a space-occupying lesion is the contraindications account's question.

PITFALL

Teaching the sympathetic surge as the whole cardiovascular story. A candidate who names only hypertension and tachycardia has dropped the phase that produces asystole, and with it the reason an antimuscarinic is considered at all. Give both phases, in order.

12.2 Why the same surge lands differently on different hearts

The sequence is the same in every patient and the consequence is not. Major depression may itself alter autonomic nervous system activity and increase the risk of arrhythmia and of sudden cardiac death, so the surge does not land on a neutral baseline. The treatment may also cause an acute rise in **QT dispersion**, which may predispose to arrhythmia in its own right. A third contribution is the workup and anaesthesia account's to teach: all the induction agents used prolong the QT interval. Three contributions, one patient, one moment.

12.3 How often a cardiac event actually happens

The quantified answer comes from a systematic review and meta-analysis of 82 studies covering 106,569 patients and 786,995 ECT treatments. Approximately **1 in 50 patients**, or 2 percent, develop a major adverse cardiac event, and the risk per treatment is approximately **1 event in 200 ECT treatments**. Those two round numbers are the review's own glosses on one pair of model estimates, and the estimates are 25.83 per 1,000 patients and 4.66 per 1,000 treatments on its Poisson model. Read them as the composite and not as a component of it: this is one finding written twice, once as a rate and once in round terms, and a page that prints both as though the precise pair were a sub-rate of the round pair states a part larger than its whole. The review's two estimators disagree substantially on the per-treatment figure, so a printed number owes the reader its model.

Both denominators travel together. The per-patient figure is proportionally higher because most patients receive a whole series, and because a course is usually stopped once a serious event has occurred.

GOOD TO REMEMBER

A rate quoted without its denominator is not a small imprecision, it is a different fact. Told it is two percent, a patient hears the risk of the whole course; told one in two hundred, they hear tomorrow morning. Say which you are answering.

12.4 Mortality: two estimates measuring two different things

There is no single mortality figure, and a resident who has memorised one has usually memorised it without its construct. The two pooled estimates carried here are not interchangeable.

AXIS	ALL-CAUSE DEATH AFTER TREATMENT	DEATH ATTRIBUTED TO TREATMENT
WHAT IS COUNTED	Any death within 30 days, whatever its cause; cardiac death was 29 percent of deaths	Deaths judged related to treatment, which is an act of attribution
CORPUS	41 studies, within the meta-analysis that produced the cardiac-event figures	15 studies, 32 countries, 766,180 ECT treatments, 16 ECT-related deaths
ESTIMATE	**0.42 per 1,000 patients** and **0.06 per 1,000 treatments**, Poisson model	**2.1 per 100,000 treatments**, 95 percent interval 1.2 to 3.4
WHAT TRAVELS WITH IT	Dropping the studies that reported only that no adverse events occurred raised it to 0.75 per 1,000 patients	In the 9 studies published after 2001, covering 414,747 treatments, there was 1 reported ECT-related death
ITS LIMIT	The authors state a death may follow treatment without being caused by it	A death not judged related never enters the numerator

The comparator the second review offers is what a frightened patient is asking about: it sets its figure beside a reported general-anaesthesia mortality of 3.4 per 100,000 for surgical procedures, and concludes that death caused by this treatment is extremely rare. One gap is declared rather than filled: the older textbook estimate expressed per patient rests on a task-force document this account could not open, and is not reproduced here.

TRAP **Quoting one figure as the mortality of electroconvulsive therapy.** The two differ by an order of magnitude and neither is wrong: one counts every death in a fixed window, the other only deaths judged caused by treatment, and the denominators differ too. State the construct and the denominator with the number, or do not state the number.

12.5 The prolonged seizure: two numbers doing two different jobs

This is the most commonly mistaught parameter in the chapter, and the error is always that the two figures are collapsed into one. The Indian guideline defines a **prolonged seizure** as a seizure longer than **180 seconds**. In the same sentence it states that termination can be attempted if a seizure extends beyond **120 seconds**, and that conditional wording is the guideline's own. One figure answers what counts as prolonged and is a definition; the other answers when you may act and is a permission.

RULE **The definitional threshold and the action threshold are separate numbers doing separate jobs, and the action threshold sits earlier.** A seizure is called prolonged past one figure; termination may be attempted past a shorter one. Collapse them, in either direction, and you have taught either a delay in acting or an intervention on a seizure that has not met the definition.

Before any drug, airway and respiration are monitored closely until the seizure has completely stopped. The guideline then names the classes that may terminate it: a benzodiazepine,

phenytoin, valproate, or a barbiturate, which is usually the anaesthetic agent already used for induction. That last detail is the practical one, because the drug is already drawn up. Its next steps are to stop theophylline or lithium temporarily or consider alternatives, and to consider shifting to an anticonvulsant anaesthetic agent; which drug moves the threshold in which direction belongs to the drug interactions account.

No dose is supplied by the guideline for any terminating agent, and none is supplied here: that gap is declared rather than filled from a remembered emergency protocol. Where prolonged seizures recur across a course the guideline suggests considering a reduction in stimulus dose, with its own caution attached: seizure duration is highest near the threshold dose, so raising the dose may in some situations shorten the seizure rather than lengthen it. The threshold itself belongs to the stimulus dosing account. **Status epilepticus** is named by the guideline in its emergency drug tray and among the differential diagnoses for recurrent postictal delirium, alongside the non-convulsive prolonged seizure, which is a seizure continuing without motor accompaniment and is therefore found by suspicion in a patient who does not reorient. No treatment-specific protocol and no frequency is given for either, and none is invented here.

IN INDIA

These thresholds are the Indian guideline's and are the ones to give in an Indian examination. They are a guideline convention rather than a physiological constant, and definitions differ between traditions. The other traditions' documents could not be opened here, so this account states the Indian figures with their attribution and claims nothing about what replaces them elsewhere.

12.6 Tardive seizure, and the seizure with nothing to see

A **tardive seizure** is a seizure happening within hours of an ECT session, after the patient has recovered from the treatment itself. Other causes of a seizure must be investigated rather than assumed away: a patient having a course can also have hypoglycaemia, an electrolyte disturbance or an intracranial event. The manifestations may be non-motor, which is the point residents miss, so a nurse watching for a convulsion will not see it. The seizures need to be aborted with anticonvulsant medicines, with no agent and no dose named by the source.

12.7 Postictal agitation and delirium

Postictal agitation is the acute agitated confusional state on emergence. A systematic review and meta-analysis of 21 articles and 66,047 patients, whose corpus was restricted to patients aged 18 or over and so is an all-adult figure rather than a late-life one, found a mean incidence of **13.9 percent** at the patient level and **12.4 percent** at the session level, rising to 18.0 percent and 16.5 percent once studies at high risk of bias in outcome measurement are removed, which are the figures its authors recommend using. Older reports range from about 8 percent to 65 percent, a spread produced by differing definitions, procedures and populations. Of the 7 prognostic factors meta-analysed none reached significance, so there is no validated predictor, and about 34 percent of those who have had it may have it again. One cross-reference belongs with these figures, because the chapter carries a higher one and a reader meeting both should not read a contradiction: this chapter's account of the cognitive side effects reports postictal agitation

at a substantially higher rate in a cohort restricted to later life, and the difference between the two is the population and not the phenomenon.

The Indian guideline's management sequence runs in this order, and the order is the answer.

1. Secure a safe environment, maintain airway, breathing and circulation, and protect the intravenous line.
2. Reduce environmental stimuli, with gentle, temporary physical restraint only where needed to prevent self-harm.
3. Give an intravenous benzodiazepine or an anaesthetic agent where agitation is persistent or severe. No agent is named and no dose given for this step.
4. Use a low-dose antipsychotic where the benzodiazepines have been ineffective.
5. In recurrent cases evaluate physical causes: electrolyte imbalance and infection are the two named.
6. Consider a non-convulsive prolonged seizure, status epilepticus or a tardive seizure as differentials.
7. For recurrence, a prophylactic higher dose of anaesthetic or benzodiazepine may be considered.

One prescribing number appears in that ladder and it is the only one this account carries. The guideline's worked example of the low-dose antipsychotic step is haloperidol given intramuscularly at **2 to 5 mg**, a prescribing decision owned by the treating team and not taught here, with a repeated dose when necessary, for postictal delirium with agitation where benzodiazepines have been ineffective. It is printed so a psychiatrist recognises what has been given and why, not as an instruction: that prescribing decision is the anaesthetist's, and the agents used earlier in the ladder are the workup and anaesthesia account's. Two flags travel with it. Its receipt is a professional body's guideline and not a drug regulator's product label, which was not read here. And the guideline states no starting dose, no ceiling and no minimum interval between repeats, so the tuple is incomplete, and an incompleteness left visible is safer than one closed from memory. Postictal confusion shades into the cognitive effects proper, and that limb, anterograde and retrograde amnesia, is the cognitive side effects account's.

12.8 Pain: headache, muscle soreness and nausea

These are the complications the patient actually reports, grouped by the guideline as pain appearing as headache, muscle soreness or joint pain. **Headache** is generally mild and treated with simple analgesia if severe, paracetamol, aspirin and the non-steroidal anti-inflammatory drugs being named without a dose attached to any of them. **Muscle soreness** has a pattern worth knowing: it is intense after the first session and would not be much with later sessions, so a patient warned in advance is not frightened by it. Intense fasciculation from the depolarising relaxant contributes, and a reduction of relaxant dose or a change of relaxant may be considered, which belongs to the workup and anaesthesia account. Nausea and vomiting generally travel with the headache and answer the same analgesia, with antiemetic options named where severe. No

incidence figure for any of the three is available here; that absence is declared rather than filled with a remembered percentage.

12.9 The injury that survives modification

Modification abolishes almost all of the musculoskeletal injury of the convulsion, and residents conclude from that it abolishes all of it. **Temporomandibular joint dislocation** follows uncoordinated contraction of the temporalis, pterygoid and masseter muscles, and happens with modified ECT because those muscles are stimulated directly by the current. Direct stimulation does not travel through the neuromuscular junction the relaxant is blocking, so the masticatory muscles are not protected by the blockade protecting everything else. Prevention is mechanical: firm holding of the jaws during the stimulus. A dislocation is examined for and relocated.

The recovery-room evaluation adds tongue bite and mucosal injury, and musculoskeletal injuries including fractures and dislocations; the pre-treatment examination makes dental evaluation for loose or missing teeth mandatory, which belongs to the workup and anaesthesia account. No rate for any of these injuries is given here, and injury under modified against unmodified technique is the modified versus unmodified ECT account's.

12.10 Anaesthetic complications, and what the recovery room watches

Anaesthetic complication is a category in its own right, and the Indian guideline treats it as one twice. It plans for it at the pre-anaesthetic evaluation, where investigations or interventions are arranged for conditions carrying a substantial risk of complications related to general anaesthesia. And it records it afterwards: the procedure record has a remarks row for any complication during or after treatment, and the footnote defining that row opens with prolonged seizures and apnoea as a single paired entry, then bradycardia or tachycardia, hypotension or hypertension, and post-ECT confusion or delirium, with a requirement to write how each was handled. **Apnoea** is the anaesthetic limb of that list and the recovery-room list adds aspiration. Why apnoea happens and in whom is the workup and anaesthesia account's.

No frequency exists for any anaesthesia-specific complication here, and none may be inferred from a guideline asking for the event to be documented: a documentation requirement is evidence that the event happens, not an incidence. In the recovery room, pulse, blood pressure and oxygen saturation continue to be monitored, electrocardiography in patients with a history of cardiac disease, feeding is avoided until full consciousness returns, and orientation and gait are assessed before the patient is shifted out.

MNEMONIC

AAA, then TEN

The recovery room watches for Agitation or delirium, Aspiration and Arrhythmia, then Tardive seizure, Enuresis or encopresis, and Neuroleptic syndrome. The three A's need action within minutes; the last three are what a tired team stops looking for once the patient is quiet.

12.11 Treatment-emergent mania

The emergence of mania during a course is a recognised adverse effect, and the whole of the current Indian guideline's direction on it is one clause: ECT may be continued or the frequency reduced based on clinical needs. There is no stopping rule, no threshold and no incidence figure, and what a reduction in frequency costs belongs to the course, frequency and number of ECT sessions account.

The published evidence behind any more confident answer is small. A systematic review searching from 1980 to August 2020 screened 1,662 articles and found 12 articles describing the treatment course of emergent hypomania or mania, in **17 patients** altogether. Nine of those patients had no known history of a manic or hypomanic episode and carried a unipolar diagnosis, which is the clinically useful fact in the record: the switch is not confined to patients already known to be bipolar. Four treatment courses were identified, and the authors state the available data are insufficient to support definitive conclusions. The incidence is therefore not established, no percentage is printed here, and antidepressant switch rates are not carried across to a different intervention in a different population.

The complications that occur are a separate object from the conditions that raise risk beforehand: the cardiovascular sequence runs parasympathetic first and sympathetic second within a short window; roughly one patient in fifty has a major adverse cardiac event over a course and roughly one event occurs per two hundred treatments; mortality has two estimates measuring two constructs, neither quotable without its denominator; the prolonged seizure carries a definitional threshold and a separate, earlier action threshold, with terminating agents named by class and no dose; and postictal agitation, tardive seizure, pain, injury under modification, apnoea and aspiration, and emergent mania complete the list, each with a management step and, where no figure is grounded, a declared absence.

13 · Consent for ECT and legal aspects under MHCA

Consent for electroconvulsive therapy in India is not an act of signing but the establishment of a lawful route, and the commonest way a resident goes wrong is to treat it as a form. The Act defines what informed consent means, names who may give it in each situation a ward produces, closes one route absolutely, gates another behind a statutory body, and imposes a duty to follow the instruction a patient wrote before losing capacity.

Owned here is the practice: what the patient is told, how capacity is judged and recorded, who consents when the patient cannot, re-consent, withdrawal, and the emergency distinction. Provisions below are named in words and never renumbered; the numbering, the prohibited-against-restricted classification and the review Board's machinery are the Mental health legislation and forensic psychiatry notes'. Consent as doctrine is the Forensic ethics, consent and legal procedure notes'. The technique contrast is this chapter's account of modified against unmodified (direct) ECT; the clinical modifications its account of ECT in special populations.

seven days
THE INTERVAL AT WHICH THE PATIENT'S CAPACITY IS REVIEWED AGAIN, THE CONSENT ITSELF NOT BEING WHAT THE ACT PUTS ON REVIEW

an application
THE ONLY LAWFUL EXIT FROM A DIRECTIVE THAT REFUSES THIS TREATMENT

ten cases
THE WHOLE INDIAN OPERATIONAL RECORD OF THE BOARD GATE FOR MINORS

13.1 What the Act means by informed consent

The Act carries its own definition of **informed consent**, stricter than ward habit. It is consent given for a specific intervention, without force, undue influence, fraud, threat, mistake or misrepresentation, obtained after disclosing adequate information including the risks and benefits of that intervention and the alternatives to it, in a language and manner the person understands. Four requirements are asked at once, and the first decides this topic: consent is intervention-specific, so a general consent taken at admission is not consent to electroconvulsive therapy, and the alternatives limb is the one most often dropped.

13.2 The emergency, and the authority the Act withholds

Here a resident is most likely to be taught something unlawful. Electroconvulsive therapy can be clinically indicated in an emergency. The Act's provision on emergency treatment cannot authorise it, because that provision expressly excludes electroconvulsive therapy from the treatment it permits. Neither statement qualifies the other. What follows is that a lawful route must be established separately, and that urgency does not cure an incomplete route.

The second half is the one most often lost: treatment authority is not admission authority. A capacitous adult can consent to electroconvulsive therapy with no admission at all, so urgency never converts the question into whether the patient must first be admitted. That is a proposition about what the Act does not require rather than anything it states affirmatively: what could be grounded is only that the Indian guideline's pre-treatment evaluation form asks the clinician to record whether the patient is an outpatient or an inpatient, so outpatient treatment is contemplated in Indian practice. That absence is offered here as the absence it is.

RULE **Urgency changes the timetable, never the route.** A patient who is not eating, catatonic or acutely suicidal earns speed in arranging a lawful route: capacity assessed now, the directive looked for now, the application filed today. Urgency never earns a shorter route.

13.3 The lawful routes, and the limit that travels with each

The Act supplies a small set of routes, each with its own trigger and its own ceiling. A route taught without its limit is how this topic goes wrong.

ROUTE	WHERE THE AUTHORITY COMES FROM	THE LIMIT THAT TRAVELS WITH IT
THE CAPACITOUS ADULT	His own informed consent, capacity deemed present	Neither a proxy nor a past document displaces it; a decision others think unwise is not incapacity
THE ADULT LACKING CAPACITY	The supported-admission provision: directive first, then his own consent with the representative's support	Substituted consent only where nearly total support is needed, only temporarily, recorded, capacity reviewed every seven days
THE ADVANCE DIRECTIVE	His own written instruction, invoked when he ceases to have capacity	Runs in either direction and more often bars than authorises; a valid refusal binds the team, the only exit an application to the Board
THE NOMINATED REPRESENTATIVE	Appointed in writing, or by the Act's deemed order of precedence	He does not displace a capacitous adult's own consent; his duties bind him to the patient's wishes
THE MINOR	The legal guardian, the nominated representative by operation of law	Treatment needs the guardian's informed consent and prior permission of the concerned Board, with no clinician discretion

13.4 The first route: the adult who has capacity

The default route is the patient's own consent, and the Act starts from a presumption. Every person, including a person with mental illness, is deemed to have **capacity** to make decisions regarding his mental healthcare or treatment, the test being functional and disjunctive: to understand the relevant information, or to appreciate any reasonably foreseeable consequence of deciding or not deciding, or to communicate the decision by any means. Named in words here; the statutory definition is the Mental health legislation and forensic psychiatry notes'. And a decision perceived by others as inappropriate or wrong does not, by itself, mean that the person lacks capacity, which forecloses the commonest shortcut of all: reading a refusal as proof that the patient cannot decide.

The limit is stated twice. A patient admitted on his own request is not to be given treatment without his own informed consent, and the establishment may not require a nominated representative's consent or presence in order to admit him; separately, a capacitous decision overrides any advance directive he wrote earlier. Taking the relative's signature as well is a habit, not a requirement. That statement is drafted about the patient admitted on his own request; no provision stating the limit generally for a person not admitted could be grounded, and the gap is declared.

13.5 The second route: the adult who lacks capacity

Where capacity is absent the Act does not simply hand the decision to the family. It routes treatment through its **supported admission** provision, and the order of authority there is fixed:

an advance directive, if any, is taken into account first, and then the informed consent of the patient with the support of his nominated representative. Only where he needs nearly total support in deciding may the representative temporarily consent to the treatment plan on his behalf, and then the consent is recorded in the medical records and his capacity reviewed every seven days.

Substituted consent is therefore a rolling, revisable state: a course over several weeks crosses that review more than once. The provision is drafted for a person admitted under it, and no free-standing treatment-consent provision for an adult lacking capacity and not admitted could be grounded; that too is a declared gap.

13.6 The third route: the advance directive, which more often says no

A resident who meets the **advance directive** only as a way of saying yes will treat a refusal as an obstacle to work around. Every person who is not a minor may make a directive in writing specifying the way he wishes to be treated for a mental illness and the way he wishes not to be; it is invoked only when he ceases to have capacity and lapses when capacity returns; a capacitous decision overrides any directive he wrote earlier; and a directive made contrary to any law in force is void from the beginning. It therefore runs in either direction, and the refusing limb is the commoner one in practice: the instrument working, not a defect. The void limb disposes of a directive purporting to authorise treatment given without anaesthesia and muscle relaxation, prohibited outright: this chapter's account of modified against unmodified (direct) ECT.

Now the provision this topic most needs. It is the duty of every medical officer in charge of a mental health establishment, and of the psychiatrist in charge of a person's treatment, to give treatment in accordance with that person's valid advance directive, subject only to the Board-review provision. That is a duty, not a consideration to be weighed, and it reaches the treating psychiatrist directly. A valid directive refusing electroconvulsive therapy binds the treating team: there is no bedside-override limb in it, no urgency limb and no senior-clinician limb.

Where a mental health professional, a relative or a care-giver desires not to follow a directive, the Act tells him to apply to the concerned Board. The Board gives an OPPORTUNITY OF HEARING to all concerned parties, including the person whose directive is in question, and may uphold, modify, alter or cancel it against five considerations: voluntariness; whether he meant it to apply to circumstances such as these; whether he was sufficiently informed; whether he had capacity then; and whether its content is contrary to other laws OR CONSTITUTIONAL PROVISIONS. Both of those are worth reading exactly. An opportunity of hearing is the natural-justice formula and is not the same as a promise that everyone is heard, and the fifth consideration carries a constitutional limb that a shortened list drops. The intention limb is what an ECT case usually turns on. The route out of a refusal is that application, never a bedside decision to disapply the directive, and urgency creates no exception. No statutory time limit appears on the face of the provision, so how quickly a Board sits is not promised.

The Act does put emergency treatment beyond the reach of a directive; that cannot open a route to electroconvulsive therapy over a refusal, because the emergency-treatment provision itself

forbids its use. The exclusion is real and, as to this treatment, empty. And a practitioner is not liable for unforeseen consequences of following a valid directive, and is not liable for not following one if he was never given a copy: that turns on possession of the document, not on his view of its merits.

TRAP **Meeting the advance directive only as a route to yes.** Its commoner operation is to bar a treatment, and the refusing directive is a form the statute names first. A resident who has met only the authorising face reads a refusal as a clinical problem to solve, and solving it clinically is the unlawful act.

13.7 The fourth route: the nominated representative, and the ceiling on the role

The **nominated representative** may be appointed by the person himself in writing, and where nobody has been appointed the Act supplies a deemed order of precedence: the individual named in the advance directive, a relative, a care-giver, a person appointed by the Board, and finally a state welfare official. What the role obliges is not proxy preference: he must consider the person's current and past wishes, life history, values, cultural background and best interests, and give particular credence to the person's own views.

Authority without a limit teaches a resident to collect the wrong signature.

- A nominated representative does not displace a capacitous adult's own valid consent.
- He substitutes where capacity is absent; he cannot consent over a capacitous patient's refusal.
- Where he consents for a patient who lacks capacity but is audibly protesting, what the patient says must be weighed.

13.8 The fifth route: the minor, and the gate that leaves no discretion

A person aged below eighteen is not considered capable of giving consent for treatment, and consent for admission and for interventions is obtained from the nominated representative. For a minor the Act fixes who that is: the **legal guardian** is the nominated representative, unless the concerned Board orders otherwise on an application showing that he is not acting in the minor's best interests or is otherwise unfit, when it may appoint a suitable willing individual instead.

The gate has limbs that bind together: the informed consent of the legal guardian, and prior permission of the concerned Board. The current Indian recommendations state it and its consequence in one breath: where a psychiatrist recommends this treatment for a minor under specific clinical conditions, guardian consent and prior permission from a Mental Health Review Board are mandatory, and the Act gives the clinician no discretionary powers for using electroconvulsive therapy in minors. They record the worry the gate creates, that delay or disapproval could impede treatment of suicidal risk and catatonia, and answer it with process rather than an exception: the minors-specific instance of the teaching that urgency does not cure an incomplete route.

What the gate costs in practice has been reported once. One tertiary centre described the first six months of operating it: ten cases, a written summary submitted in each, the caregivers' informed consent sought first and permission after that, a response within a few days without inordinate

delay or objection, and permission denied in no case. That is a single-centre series from an institute with its own Board, and not a national expectation.

The Indian guideline adds what the statute does not: oral or verbal **assent** from the minor as the age permits, alongside written informed consent from the parents or nominated representative, and concurrence by two independent psychiatrists, or a psychiatrist and a physician. That concurrence is professional and not statutory, and presenting it as part of the gate misstates the law.

A minor cannot make an advance directive, but the legal guardian can make one for the minor, and since the instrument runs either way it can bar this treatment as readily as request it. If the minor attains the age of eighteen during the hospital stay he is to be classified as an independent patient, that is communicated to him, and treatment continues on that footing or as a patient requiring high support. Where the Board has displaced the guardian, guardian and representative differ, and whose consent the gate then requires is unanswered by the sources.

IN INDIA

A pair of Indian facts is routinely conflated. The statute supplies the gate for a minor: guardian consent and prior permission of the concerned Board. The guideline supplies more: the minor's own assent, and a second psychiatrist's concurrence. Name which is which.

13.9 The consent as it is taken, documented, re-taken and withdrawn

The Indian guideline states the conversation and its documentation. Written informed consent is taken before treatment begins, on the principles of shared decision-making. It covers the anticipated benefits and the possible short-term and long-term adverse effects in this individual, including the risks of the anaesthesia as well as of the treatment, plus the type of treatment, the modification procedure, the electrode placement and expected outcomes. Unless the patient disagrees, caregivers are made part of the process. Where the patient lacks the capacity to consent, that is documented; any advance directive is examined; and, in accordance with that directive, consent MAY be obtained from the nominated representative. The permissive form is the guideline's own: the representative route is conditional on what the directive says, not an automatic next step.

Read the order, because it is the Act's order too: the directive is examined first, and the representative's consent comes after it and in accordance with it, never instead of it.

Re-consent is given as triggers rather than as an interval: consent is taken again when the patient regains the capacity to consent, when a minor attains the age of eighteen, and before continuation or maintenance treatment is initiated, the clinical condition, the purpose and the character of the treatment having all changed by then. That indication itself is this chapter's account of continuation and maintenance ECT.

The guideline states the right in the patient's own terms: he may refuse consent now, and may withdraw consent during the course of treatment, and in either case the best available alternative

treatments will be provided without any prejudice. That second half is the part residents omit, and it makes the right real rather than nominal.

RULE **Record the route, not only the signature.** The note should say which route was used: capacity present and consent taken from the patient; or capacity absent, the directive looked for and recorded, and the representative's consent taken temporarily with the review date entered; or the guardian's consent with the Board's permission on file.

MNEMONIC

ADULT, ABSENT, ADVANCE, AGENT, AGE

Adult: the capacitous adult's own consent, an unwise choice not being incapacity. **Absent**: capacity absent, so the supported route, substituted consent temporary and recorded, with the patient's CAPACITY reviewed every seven days. **Advance**: the directive, which runs either way and binds when it refuses, the exit being an application to the Board. **Agent**: the nominated representative, who never displaces a capacitous adult. **Age**: the minor, needing guardian consent and prior permission of the concerned Board.

PITFALL

Writing the emergency into the answer as though it were a route. It is a clinical urgency, never a source of authority, and the Act's emergency-treatment provision excludes this treatment by name. The paired error runs the other way: assuming that because that route is closed the patient must be admitted first. He need not be.

Consent for this treatment is the establishment of a lawful route, measured against the Act's definition of informed consent: intervention-specific, voluntary, and informed as to risks, benefits and alternatives. The routes are the capacitous adult; the adult lacking capacity, under the supported provision with substituted consent temporary and recorded and the patient's capacity reviewed every seven days; the advance directive; the nominated representative; and the minor, through guardian consent with prior permission of the concerned Board. Each carries a limit: no proxy and no past document displaces a present capacitous decision, and a valid directive refusing this treatment binds the team, the only exit an application to the Board. The emergency provision is not a route to this treatment, a lawful route must be established separately, and urgency does not cure an incomplete one. Consent is written, taken again when capacity returns and before continuation treatment, and may be withdrawn at any point without prejudice.

14 · ECT in special populations (pregnancy, elderly, adolescents)

A special population does not change what electroconvulsive therapy is; it changes the plan built around it, meaning who else evaluates the patient, what is monitored, which ordinary choice is deliberately reversed, and what the conversation before treatment must contain. The four special populations are different problems: pregnancy an evidence problem before a

technique one, old age a selection problem in which the usual instinct runs backwards, the minor a legal problem, comorbidity a co-management one.

Owned here is the clinical modification each population requires, and nothing else. Named below and not re-taught: the anaesthetic conduct being modified, owned by the pre-ECT workup and anaesthesia; the consent practice being varied, owned by consent for ECT and the legal aspects; and likewise the cognitive cost, the conditions raising risk beforehand, concurrent drugs, the titration and the placement comparison. The statutory gate on ECT for a person under eighteen belongs to the Mental health legislation and forensic psychiatry notes; it is named here in words and never renumbered.

20 weeks THE GESTATION PAST WHICH THE SETTING CHANGES, NOT THE TECHNIQUE	**no randomised trials** IN THE LARGEST SYNTHESIS OF ECT IN PREGNANCY	**0.35** OLDER AGE AS A PREDICTOR OF RESPONSE, AS A STANDARDISED MEAN DIFFERENCE

14.1 Pregnancy: the modification, the monitoring and the threshold

The Indian guideline states its own scope before its list. These steps are most applicable in the second and third trimesters, and the risk evaluation is a joint activity of the psychiatrist, the anaesthetist and the obstetrician. That is structural, not courteous: each item follows from a physiological change an obstetrician reads better.

Hydration is maintained through the fast, with intravenous fluid if dehydration is noted. A **left lateral or pelvic tilt** maintains aorto-caval circulation, the supine gravid uterus otherwise cutting venous return just as a surge is induced. Pre-oxygenation is provided but hyperventilation avoided, the instruction most often got backwards: blowing off carbon dioxide favours the seizure in ordinary practice, while here placental oxygen delivery governs. An H2 blocker or antacid is considered against the raised aspiration risk. The anticholinergic line is a trade and not a prohibition: routine anticholinergic premedication is avoided even though the guideline concedes in the same sentence that it would reduce the risk of foetal arrhythmias. Memorise the first half and you have a rule; carry both and you have a decision. The agents and sequence are the workup and anaesthesia account's; no dose appears here.

Monitoring is doubled. Doppler or cardiotocography is used for the foetus before, during and after treatment, alongside maternal vitals. Then the threshold: after 20 weeks of gestation, treatment should preferably be given where obstetric support is available, with **tocolytics** and preferably a **tocodynamometer** to hand against induced labour or abortion. The guideline hedges with preferably twice, and the hedge is carried rather than tightened. Past that gestation what changes is the room, not the treatment.

14.2 The comparative claim, and the three things it does not say

The sentence a resident will meet is that ECT is safe and effective in pregnancy and is a preferred option, given its lesser risk of teratogenicity compared to many psychotropics. It supplies a comparator, a class and not a drug: many psychotropics. A domain: teratogenicity. A direction: lesser, for ECT.

It does not supply the gestational window in which the comparison holds, nor the illness severity at which it holds, nor any graded strength of evidence. It says nothing about outcomes other than teratogenicity, which matters because the register evidence below points the other way on preterm delivery and the newborn's condition at birth. Those absences cannot be filled from another source here, so they are not.

RULE **Never write that ECT is often the safer option in pregnancy without stating what it is safer than, in which period of the pregnancy, for which severity of illness, and at what strength of evidence.** The published sentence is bounded on one axis only, and the bare version silently promises a general safety finding no source here states.

14.3 What the pregnancy evidence base is, and what it cannot settle

The largest and most recent synthesis included 82 reports carrying information on more than 1,300 pregnancies and deliveries, and its composition is the point: 57 were case reports and 14 were case series, the remainder non-randomised or retrospective. There are no randomised trials, so a clinical position rests on the weakest available design.

What it reports is reassuring in shape: the most frequent adverse events were generally mild and transient. But the same synthesis records, across the reviewed reports, 1 placental abruption, 21 premature deliveries, 6 congenital malformations and 4 stillbirths. Then the authors state the limit themselves, in the sentence that must travel with every reassuring one: because information was limited, causal links between modified ECT and many of these adverse events were difficult to establish, and no inference could be drawn about differential effectiveness or safety between important patient subgroups or across variations in technique.

That cuts both ways and residents usually take one. The harms cannot be confidently attributed to the treatment; neither can the reassurance. A literature that cannot establish causation cannot establish its absence, so the reassuring picture and its limit are one object, never taught apart.

14.4 The register cohort, and why the comparator decides the answer

The largest observational study inside that review is a Swedish population-based register cohort of 793, propensity-score matched. On effectiveness it reassures: response rates matched those of non-pregnant women receiving ECT. On obstetric outcome, the control group decides the answer.

Premature delivery ran at 14.4 per cent against 9.0 per cent compared with psychiatrically hospitalised pregnant women not given ECT, an odds ratio of 2.33, confidence interval 1.15 to 4.73, which is significant. Against the second, additional pregnancy control group it ran at 10.9 per cent, odds ratio 2.16, confidence interval 0.75 to 6.22, which does not exclude no effect. A low 5 minute Apgar score behaves identically: 9.3 per cent against 2.1 per cent, odds ratio 3.68, confidence interval 1.58 to 8.55 against the first, and 2.2 per cent, odds ratio 2.53, confidence interval 0.59 to 10.90 against the second. The same table carries the finding that cuts the other way, and it travels with the rest: none of the reported preterm births occurred in close time proximity to treatment, which weakens the causal link. These figures are attributed to the review's own table at the review's year, the primary study not having been opened here.

The reviews then disagree for methodological, not empirical, reasons. An overview identified 9 articles, of which 5 met inclusion criteria, covering 32 to 339 patients, foetal deaths ranging from 2 to 12. 1 of the 5 recommended ECT only as a last resort while the others called it relatively safe, the split attributed to differing inclusion criteria and differing attribution of adverse events, and that is read at abstract level here. Reviewers of overlapping literature reached opposite recommendations because they counted differently: the question is unsettled at the level of method, not of data.

TRAP **Quoting a risk figure without its comparator.** Every significant obstetric signal here loses significance when the control group changes, and every one keeps its point estimate. Carry the preterm figure without its comparator and you report a hazard the study does not establish; carry only the non-significant comparison and you report a safety it does not establish.

14.5 What the uncertainty puts into the consent conversation

The perinatal review draws an explicit consequence from its own uncertainty, and it is a consent instruction rather than a treatment one. Modified ECT is an important option for severe perinatal depression, and informed consent practice should reflect the knowledge gaps identified in the review as well as the well-known side effects, and should be set against the substantial adverse consequences of untreated or undertreated maternal depression. That supplies the honest comparator for the risk discussion and makes the gaps a required disclosure: not a reason to withhold, not a reason to soften the account, but content the woman is entitled to. How consent is taken, meaning capacity, the nominated representative, re-consent and withdrawal, is the consent for ECT and legal aspects account's.

14.6 The elderly: not merely tolerable candidates but preferentially good ones

Start with the effect size, because the order matters. A meta-analysis screened 2193 articles and included 34. Older age predicted remission at a standardised mean difference of 0.26 and response at 0.35, at P less than 0.001. Psychotic features predicted remission at an odds ratio of 1.47 and response at 1.69; severity predicted response at 0.19 but not remission. The authors conclude that ECT is particularly effective in patients with depression with psychotic features and in elderly people with depression, and the Indian guideline agrees from practice: response rates in elderly depression exceed those in mixed-age populations.

Carry that first, and as an effect size rather than an impression, because everything below is a burden to be managed and a resident who meets the burdens first reads this section as reasons to withhold. Age is a positive predictor; the burdens are separate facts about the same patient, not the price of the benefit.

High seizure threshold, missed seizures, medical comorbidity and postictal delirium are all commoner in old age. The cognitive concern is located rather than generalised, which is the guideline's most careful sentence here: cognitive adverse effects are of greater concern primarily when ECT is indicated for dementia-related behavioural disturbance. That is a statement about one indication, not a blanket caution about age. The amnesias and how cognition is tracked are the cognitive side effects account's.

The modifications follow, each owned elsewhere. Propofol may be avoided and anticonvulsants minimised, the agents being the workup and anaesthesia account's. Non-convulsive shocks are minimised, for which a higher initial dose and faster increments may be considered; the titration is the stimulus dosing account's, and increments means the electrical dose, never the number of treatments in a week. Right unilateral or bifrontal placement is preferred to bitemporal, a preference inside a comparison the bilateral versus right unilateral account owns, and age does not reverse it. A lower range of brief or ultrabrief pulse may be considered. One number sits alongside: concomitant lithium above 0.6 is avoided for the risk of **postictal delirium**. The unit is a separate point and gets its own sentence rather than an interruption inside that clause. The guideline prints it here as 'mq/l', which is a typographical error for milliequivalents per litre; the error is reproduced rather than silently repaired, because substituting the unit one believes was meant is how a wrong number enters a chapter, and the same guideline spells the same unit 'meq/l' at the second place the same figure appears, which this chapter's account of drug interactions with electroconvulsive therapy carries.

14.7 Adolescents and minors: the gate first, then the four headings

For a person under eighteen the statutory question is answered before any clinical one. That gate, its prohibition and the narrow exception that opens it, are taught in full by this chapter's own account of consent for electroconvulsive therapy and the legal aspects, which is where to go for them; the Mental health legislation and forensic psychiatry notes own only the numbering and the wider classification of the provisions. The gate is named here rather than restated, and is never renumbered. Nothing below creates an exception to it: the criteria decide whether ECT is the right treatment, never whether it may lawfully be given.

The clinical frame sets out four headings, all of which must be satisfied.

- Heading A is specific clinical features: high risk to self from active suicidal ideas or behaviours, high risk to others from severe repetitive aggression, catatonic features, or long illness with inadequate response.
- Heading B is the adequacy of pharmacological or psychotherapeutic intervention.
- Heading C is that alternatives, including other medications, other routes and other brain stimulation methods, were considered and not found suitable.
- Heading D is that medical contraindications for modified ECT have been ruled out by pre-anaesthetic check-ups, where the word modified is doing real work, modified being the only lawful form in India.

Heading B is the one to learn, because the urgent case is answered there. It is satisfied when recommended treatments have been used in adequate dose and duration with inadequate change; or when recommended treatments have been initiated but would take time to be effective, during which time the clinical features might pose a significant risk to the patient; or when the child is not tolerating the up-titration required to reach recommended doses. The middle limb answers the objection that a child cannot wait for a drug to work: the waiting itself can be the risk, stated as a clinical criterion inside the gate, not a shortcut around it.

TRAP **Reading urgency as a legal exception.** Heading B answers the urgent case clinically and leaves the statutory position untouched; a fast clinical justification is not an authority to treat. The reverse error is as common: treating the gate as a reason to conclude that ECT is unavailable to a young person in a dangerous state.

One thing genuinely reverses here, on a single physiological fact. **Prolonged seizures** are commoner in this age group, so proconvulsant or seizure-neutral induction agents such as etomidate and ketamine are best avoided. Elsewhere those agents are reached for precisely to protect a poor seizure; here that virtue is the hazard. Their comparative effects belong to the workup and anaesthesia account, and no dose is stated. The same guideline records that the literature is limited, that no differential efficacy or safety has been shown, and that a second psychiatrist's opinion is useful.

IN INDIA

The published Indian experience is uncontrolled tertiary-centre case series, which is what it must be called before its rates are read. The first reported 25 patients below the age of 18 over 12 years, with catatonia the commonest indication at 68 per cent, and after a mean of 10 treatments the response rate was 76 per cent. A later series from the same group covered 51 individuals up to 19 years of age over 7 years, a mean of 8 treatments producing significant improvement in 86 per cent. At a third centre, twenty-two children treated over a ten-year period, the youngest aged 10, had prolonged seizure as the commonest side effect at close to 35 per cent.

The international series agree on the shape and the mechanism. Fifty-one adolescents of mean age around 16, in a United States series covering 1991 to 2013, had bitemporal treatment over a mean of 9 sessions with clinically significant improvement in 77 per cent, and it records that prolonged seizure duration is common in young people and declines with age across adolescence. That age gradient is why the anaesthetic choice changes. A Swedish national register covering 118 young people under 19, from 2012 to 2016 used right unilateral placement over a mean of 7 sessions, and fifty-seven per cent responded adequately, adverse effects in 25 per cent being minor. The placements differ between the series and that difference is not a recommendation.

14.8 Physical comorbidity: co-management, organ by organ

Here the population is an organ, and the modification takes one form: optimise it before, protect it during, share the decision with its specialist. The conditions raising risk beforehand, and the interval after a recent infarction, are the contraindications account's.

SYSTEM	WHAT IS MODIFIED	WHY
CARDIAC	Blood pressure optimally controlled, with short-acting antihypertensives during treatment if needed; anticoagulation considered in valvular disease; multiple restimulations avoided in congestive heart failure, with functional capacity assessed by 2D echocardiogram and treadmill test; short-acting parenteral antihypertensives in aneurysm; conduction defects and pacemakers generally safe, given only in consultation with cardiologists	A brief surge is met with agents of equally brief effect
CEREBRAL	Postponement after recent ischaemic or haemorrhagic stroke and with a symptomatic intracranial mass until the risk of complications is minimised; patency of a ventriculo-peritoneal shunt confirmed; after craniotomy, electrodes placed away from the site to avoid a very high charge density; depolarising muscle relaxants avoided in multiple sclerosis; cerebral implants and foreign bodies not an absolute contraindication, treatment given in consultation with neurologists	An opened skull conducts differently, and a defect concentrates charge
RENAL	Adequate muscle relaxation, with non-depolarising agents preferred; in haemodialysis patients, serum potassium measured within 24 h before treatment	A depolarising relaxant releases potassium a failing kidney cannot excrete
RESPIRATORY	Hypoventilation avoided, since it leads to respiratory acidosis, hyperkalaemia and further acid-base imbalance; theophylline discontinued 24 h before treatment, for the risk of prolonged seizures and status epilepticus	Ventilation is supplied, not spontaneous, so its adequacy is a decision

PITFALL

Reading the two relaxant lines as one rule. One avoids a depolarising agent in multiple sclerosis, the other prefers a non-depolarising agent in renal disease: two members of an open group reached by different routes, not one instruction generalised.

MNEMONIC

Room, rate, reversal, ruling, review

Room: past the stated gestation, pregnancy changes where treatment happens, not how. **Rate**: in the elderly the response rate leads. **Reversal**: in minors the agents chosen elsewhere to protect a seizure are avoided. **Ruling**: the gate is statutory and comes first. **Review**: comorbidity is co-managed with the organ's specialist.

The examinable core is a habit, not a list: say what the plan changes, name who else is in the decision, and state the evidence at the strength it has. The comparative claim in pregnancy never travels without the three things it does not say; the elderly get their effect size first and their burdens second; the minor gets the gate named, then the four headings.

15 · Course, frequency and number of ECT sessions

An acute course of electroconvulsive therapy is a schedule rather than a single decision, and the schedule settles how fast the patient recovers, what that costs in memory and when treatment stops. How often treatments are given is the parameter most often remembered as an efficacy lever, and it is not an efficacy lever. A much faster schedule exists for presentations that kill within days, bought with mortality rather than impatience.

Owned here in full: the acute course and every schedule on it, treatment frequency and the twice-weekly against thrice-weekly comparison, the accelerated schedule for life-threatening presentations, the number of treatments by indication, judging response, the mid-course decision and stopping, with the malignant catatonia protocol assembled end to end. Named and not re-derived, each with its own account here: why catatonia and its malignant form earn the treatment; the seizure threshold, its titration and the adequate-seizure criteria; and the cognitive cost. Continuation and maintenance treatment begin where this ends. The catatonia entity, the lorazepam challenge, the malignant catatonia against neuroleptic malignant syndrome differential and the rating scale are the Other psychotic disorders, delusional syndromes and catatonia notes'.

2 to 3 per week	8 to 12	3 to 5 days
THE INDIAN ACUTE-COURSE FREQUENCY, A RANGE AND NOT ONE SCHEDULE	TREATMENTS BEFORE AN INDIAN COURSE MAY BE CALLED A FAILURE	OF DAILY OR TWICE-DAILY BILATERAL TREATMENT OPENING A MALIGNANT CATATONIA COURSE

15.1 The acute course, and what treatment frequency means

The acute course is the block of treatments that brings the present episode to remission, described by three things: how often treatments are given, how many, and what stops them. **Treatment frequency** is the first, and it is a construct rather than a schedule, because the guidelines state it as a range. The Indian guideline gives the acute course as **2 to 3 sessions per week**, and its consent supplements state what the patient is told, ECT on alternate days or twice a week for about 2 to 3 weeks.

A policy guideline adds the spacing rule, that ECT is usually given two or three times per week in an acute course, spaced as evenly as possible across the week. Two schedules dominate within it, the **twice-weekly schedule** and the **thrice-weekly schedule**, and what separates them is not how well the course works.

15.2 What the faster schedule buys, and what it does not

Two double-blind randomised trials carry the comparison, each holding anaesthetics constant with a weekly simulated treatment in the slower arm. The first found that both schedules significantly improved depression scores, with no difference in outcome either 1 week or 1 month after the series ended, while response to ECT three times a week was significantly faster and cognitive effects more prominent. The replication found no significant difference in antidepressant outcome, with significantly more rapid response on the thrice-weekly schedule, associated with more severe memory impairment.

Their joint synthesis adds what decides the teaching: the antidepressant effect of the two schedules at the end of the course was equal, while the thrice-weekly schedule induced more severe memory impairment even when the number of treatments in the series did not differ significantly. The cost belongs to the spacing, not to receiving more treatments, so it cannot be argued away by pointing at the total. The reviewers give the rule with its premise: when rapid improvement is clinically warranted, thrice-weekly ECT may be preferred, given that both schedules are equally efficacious.

RULE **Treatment frequency buys SPEED of response, and it is not an efficacy lever: both schedules reach equivalent efficacy at the end of the course, and the faster one reaches response sooner at a higher cognitive price.** Written the other way round it teaches a resident to impose extra cognitive injury for a gain that does not exist. **Speed of response** is a real good where the illness is racing; where it is not, the price is paid for nothing.

15.3 How thin the evidence beneath that rule is

The only meta-analysis confirms the efficacy half and prices the slower schedule in days: twice-weekly ECT produced a similar change in depression score, with a statistically significant longer duration of treatment of roughly 4.8 to 6.5 days. It fixes the lower bound too, since thrice-weekly ECT was significantly more efficacious than once-weekly ECT, and reports no cognitive endpoint, corroborating the efficacy and duration halves only.

Outside depression the trade is half established. The one comparison in schizophrenia, a retrospective series of 43 patients, found more rapid response on the thrice-weekly schedule, equal effectiveness, and no significant difference in cognitive impairment at the end of the study: the speed finding reproduces, the cognitive one does not. Nothing newer exists, since a review of trials to March 2019 found no original research study examining the issue of frequency of ECT sessions among its 30 studies. The rule rests on trials from the 1990s and on consensus, and saying so is part of teaching it.

15.4 Which schedule each tradition actually recommends

The shorthand is a two-camp story, the United Kingdom twice weekly and North America thrice weekly, and the sources do not support it. What is grounded is practice: in the United States and Israel thrice-weekly ECT is a common practice, while in the United Kingdom twice-weekly sessions are commonplace. Practice is not recommendation, and the American position is itself a range: the task force recommends twice or thrice weekly sessions, cautioning that more frequent sessions could result in higher cognitive deficits and that frequency should be reduced if cognitive effects are of serious concern. The United Kingdom limb ties higher frequency to lethality, not impatience: the college handbook recommends twice weekly for bilateral and for unilateral ECT, with thrice-weekly bilateral reserved for life-threatening illness and only while the threat is high.

One exception inverts that and stops the twice-weekly preference reading as a blanket law: given the slower speed of response already seen in ultrabrief ECT, that form should routinely be given three times per week. Where no source settles a jurisdiction's recommendation the gap is declared: none states thrice weekly as a North American recommendation.

15.5 The accelerated schedule, and what buys it

Twice against thrice weekly is not the whole of frequency, and what it omits matters when the clock is short. In malignant catatonia the course opens on an **accelerated schedule**: in view of the life-threatening potential of malignant catatonia, bilateral treatments daily or twice daily for 3 to 5 days are often required to achieve a rapid result, followed by ECT at conventional frequencies until complete resolution. Why the presentation earns treatment is this chapter's account of the indications, and the entity and its differential are those catatonia notes'. The schedule is assembled here and nowhere else.

The same guideline grades it, with trigger, endpoint and step-down: if there is partial or no response to lorazepam within 48 to 72 hours, institute bilateral ECT once or twice daily for up to 5 days until malignant catatonia abates, followed by ECT three times per week until sustained improvement, usually 5 to 20 treatments in total. Its grade is D, expert consensus and not trial evidence, part of the teaching. A second jurisdiction corroborates it, introducing ECT with closer spacing, that is successive days, for rare severe and life-threatening states such as malignant catatonia, then resuming at 2 or 3 per week, but not twice-daily treatment, which rests on the consensus guideline alone.

The comparable emergency is not handled identically: in neuroleptic malignant syndrome, if clinical features persist, consider bilateral ECT three times weekly or, in severe cases, once or twice daily until the syndrome abates, then three times per week until sustained improvement, to a total of 5 to 20 treatments, graded C. Accelerated dosing opens the malignant catatonia course and is held in reserve here.

What buys the extra cognitive cost is mortality, and the evidence is survival data. Across five series, 51 of 54 patients treated with ECT survived, whereas only 6 of 14 given antipsychotics and supportive care recovered, and timing decided the rest: **16 of 19 patients receiving ECT**

within 5 days of onset survived, while none of 14 starting beyond that 5-day point recovered. These are historical series inside a current guideline, cited for what they established, not as a practice parameter. The trade is the chapter's usual one, priced differently: where the competing risk is death within days, speed outweighs the cognitive cost, and that cost is this chapter's account of the cognitive effects.

STEP	MALIGNANT CATATONIA	NEUROLEPTIC MALIGNANT SYNDROME
TRIGGER TO TREAT	Partial or no response to lorazepam within 48 to 72 hours	Clinical features persist
PLACEMENT AND STIMULUS	Bilateral, the stimulus dosed liberally to secure a well-generalised seizure; threshold, titration and adequacy are this chapter's dosing account	Bilateral, under the same account
OPENING FREQUENCY	Daily or twice daily, for 3 to 5 days	Three times weekly by default, once or twice daily in severe cases
ENDPOINT OF THAT PHASE	Until malignant catatonia abates	Until the syndrome abates
STEP-DOWN	**Step-down** to three times per week until sustained improvement	Three times per week until sustained improvement
TOTAL AND GRADE	Usually 5 to 20 treatments, grade D	A total of 5 to 20 treatments, grade C

TRAP **Giving malignant catatonia the ordinary twice-weekly or thrice-weekly course.** The decision to treat is right and the schedule too slow, which in a presentation whose survival turns on how early treatment starts is a wrong answer with a right diagnosis attached.

15.6 How many treatments, and by which indication

No source fixes a course length in advance: the number of ECTs should not be predetermined but should be based on the needs of individual patients. Around it sit the examinable figures. The task force gives **6 to 12 sessions for depression**, higher where the protocol changed and in schizophrenia, and practice sits inside it, the mean number of ECT sessions being between 6 and 10 in most studies, which its reviewers say cannot be read without knowing why courses stopped.

In catatonia practice was wider than any recommendation: most studies reporting frequency described ECT three times weekly, ranging between daily and twice weekly, with sessions from 3 to 35 and a mean of 9, and no data on the superiority of these protocols. For non-malignant acute catatonia the guideline sets a floor, that where ECT is administered in acute catatonia it should be given at least two times weekly, tying the count to response, risks and side effects. Direction is grounded where a number is not: in general the response to ECT is seen after more sessions in schizophrenia than in depression and mania.

15.7 Judging response, the mid-course decision, and stopping

Two rhythms of assessment run through a course. The Indian guideline requires a structured, indication-specific symptom rating tool weekly wherever feasible, and a clinical assessment after every ECT, preferably within 24 hours and certainly before the next session, with symptoms and cognitive and other adverse effects monitored at least once a week. A policy guideline sets another cadence, clinical assessments at least twice per week, with structured rating scales before commencing and at the end: the difference is jurisdictional.

The **mid-course review** carries two thresholds, not to be merged. Technique changes first: **switching unilateral to bilateral, or ultrabrief to brief pulse, may be considered after a risk-benefit analysis if improvement is not noted after four to eight sessions**. Failure is declared much later, since a minimum of 8 to 12 ECT sessions should be provided in acute courses before considering failure of response, with continuation beyond 12 sessions decided after a risk-benefit analysis. The United Kingdom handbook is stricter and conflicts with that floor: bilateral ECT for depression may be stopped if there is no improvement at all during the first six treatments, and continued to 12 sessions where there is some. Both are printed and the conflict named, not resolved.

Clinically, ECT may be terminated at any time if complete clinical improvement or remission is achieved, and the course is generally stopped as soon as remission is achieved, or if initial improvement remains unchanged for two additional sessions. Current United Kingdom depression guidance has two limbs: stop ECT for a person with depression immediately if the side effects outweigh the potential benefits, or when stable remission has been achieved. The endpoint is **stable remission** and not response, the wording of the superseded appraisal: assess clinical status after each session and stop when a response has been achieved, or sooner on adverse effects, monitoring cognitive function at a minimum at the end of each course, which stays quotable for catatonia, a prolonged or severe manic episode and schizophrenia only.

- **After every treatment.** A clinical assessment, before the next one.
- **Once a week.** A structured rating tool where feasible, plus adverse effects.
- **At the mid-course review.** If nothing has moved, change technique before changing the verdict.
- **At remission.** Stop; current depression guidance ends on remission, not response.
- **Before calling it a failure.** Count what was given; the Indian floor is high.

> **GOOD TO REMEMBER**
>
> Cognitive concern is a reason to slow a course down, not only to change the placement: both the task force and the policy guideline say to reduce the frequency where cognitive effects are a serious concern. Slowing costs time to remission and not the eventual result.

IN INDIA

Three numbers come from the current guideline, the last two from a single passage: two to three sessions per week, the minimum of eight to twelve sessions before failure is considered, and the four to eight session point at which technique changes instead. The last two are routinely confused, the earlier changing placement or pulse width, the later changing the verdict. Schizophrenia is common here and responds after more sessions, so a long course may be ordinary for it.

MNEMONIC

SOONER NOT STRONGER, DYING BUYS DAILY, COUNT BEFORE YOU CALL IT FAILED

Sooner not stronger is the frequency question: the faster schedule reaches response earlier and costs more memory, and both finish in the same place. **Dying buys daily** is the accelerated regimen: lethality, not impatience, pays for the added cognitive cost, and the schedule steps down once the emergency abates. **Count before you call it failed**: change technique at the earlier threshold, and count what was given before declaring failure.

PITFALL

Writing a faster schedule into an answer as though it made the course more effective, which it never does. The consequence is clinical: the resident who believes it imposes avoidable memory impairment on a patient who would have reached the same end point anyway.

The acute course runs at two to three treatments a week, and treatment frequency buys speed of response rather than efficacy: both schedules reach equivalent efficacy at the end of the course while the faster reaches response sooner and costs more cognitively, established in depression, not schizophrenia. Malignant catatonia and comparable life-threatening presentations open instead on an accelerated schedule of daily or twice-daily bilateral treatment for a few days, triggered by failure of the lorazepam trial, run until the syndrome abates and stepped down to three times a week, because mortality pays for the cognitive cost. No course length is fixed in advance; response is assessed after every treatment and rated weekly; technique changes at the earlier non-response threshold and failure only at the later; the course stops at stable remission.

16 · Continuation and maintenance ECT

An acute course of electroconvulsive therapy buys a remission and does not keep it, and what is put in place in the weeks after the last treatment decides whether the remission survives. A patient left with nothing active after remission is at very high risk of relapse, heaviest in the first weeks. Continuation and maintenance treatment answers that fact: medication continued, or treatment on a lengthening interval, or both. It is examined as a sequence of intervals, written here.

Owned here in full: the relapse problem after a successful course, the continuation pharmacotherapy that must follow it, and continuation and maintenance electroconvulsive therapy itself, meaning its indications, its tapering schedule of intervals, its maintenance interval, its duration and review, its cognitive monitoring, its evidence and the unsettled question of its stimulus dose. Named and not re-derived, each owned elsewhere in this chapter: the acute course and its frequency, which end where this section begins; the seizure threshold and its titration; the amnesias; and the drugs that move the threshold. The choice of continuation drug belongs to the Mood disorders: pharmacotherapy notes.

84%	1 to 12 weeks	3 to 6 months
RELAPSE AFTER A SUCCESSFUL COURSE WHEN NOTHING ACTIVE FOLLOWS IT	THE MAINTENANCE INTERVAL BAND, ONCE THE CONTINUATION PHASE IS OVER	HOW OFTEN COGNITION IS REASSESSED ON A MAINTENANCE COURSE

16.1 The problem this section exists to answer

The trial that defines the problem randomised remitters to placebo, nortriptyline, or nortriptyline with lithium. Over 24 weeks the relapse rate was **84%** on placebo, 60% on nortriptyline alone and 39% on the combination. Two readings follow. Without active treatment virtually every remitter relapses within 6 months of stopping, so no acute course ends without a continuation plan. And the danger is front-loaded: on the combination, all but one relapse occurred within 5 weeks of the end of the course, while on placebo or nortriptyline alone relapse accrued throughout. The weeks straight after the last treatment are the ones that need cover, which is what every schedule below is for.

16.2 Continuation pharmacotherapy, and whether ECT does better

Continuation pharmacotherapy is the default and is not optional. The current United Kingdom depression guideline of 2022 states that if depression has responded to a course, the clinician should start or continue antidepressant medication or a psychological intervention to prevent relapse and to provide ongoing care, and consider lithium augmentation of the antidepressant. Which antidepressant, at what dose, and how the lithium augmentation is set up are the Mood disorders: pharmacotherapy notes'.

Whether continued treatment beats continued drugs was tested head to head: 201 patients who had remitted with bilateral treatment, randomised to continuation electroconvulsive therapy or to continuation lithium with nortriptyline for 6 months. Relapse was 37.1% in the continuation electroconvulsive therapy arm and 31.6% in the drug arm, with no significant difference in overall survival curves or in time to relapse. Both beat a historical placebo control, and both had limited efficacy, with more than half of patients either relapsing or dropping out.

TRAP **Writing continuation electroconvulsive therapy as the stronger option because it is more intensive.** The head-to-head trial found no difference in relapse or in time to relapse against continuation drugs. It is an alternative for the patient in whom drugs have failed, are not tolerated or are refused, not an upgrade for everyone who responded.

16.3 Continuation and maintenance: two phases, one line between them

The two words are not interchangeable, and the boundary between them is a duration. **Continuation electroconvulsive therapy** follows the **index course** and consolidates the improvement obtained; **maintenance electroconvulsive therapy** begins beyond that line and prevents a new episode. The Indian guideline draws the line at 6 months and names the conditions in which either is used: major depressive disorder, bipolar disorder and schizophrenia. Who earns an acute course at all is this chapter's account of the indications. A second guideline draws the same line and adds the operative instruction: if patients remain well during the continuation phase, the interval between treatments should be progressively extended.

AXIS	CONTINUATION PHASE	MAINTENANCE PHASE
WHEN IT RUNS	From the end of the index course, up to 6 months of remission	Beyond 6 months from the treated episode
PURPOSE	Consolidating what the index course obtained	Prophylaxis against a further episode or recurrence
INTERVAL	Weekly to monthly, extended progressively while the patient stays well	**One session every 1 to 12 weeks**, and occasionally less frequently
SETTING	Both are generally administered as outpatient treatments	
CONSENT	Obtained again before either begins, because the clinical condition, the purpose and the character of the treatment have all changed	

The consent row is not a formality: what has changed since the first conversation is the condition, the purpose, now relapse prevention rather than rescue, and the treatment's own character, meaning its frequency and its endpoint. The statutory machinery is this chapter's account of consent and the law.

16.4 The taper: the schedule of intervals, in order

This is the schedule the reader was promised, and it begins where the acute course ends; this chapter's account of the acute course and its frequency stops there. The Indian guideline states the sequence plainly: an effective acute course is gradually tapered down from twice a week to weekly, then fortnightly, then monthly, tailored to the patient's clinical needs. Two instructions travel with it: the course should not be prefixed, and it should be planned dynamically and scheduled on periodic clinical review. Once the continuation phase is over, **maintenance is usually given at a frequency ranging from one session every 1 to 12 weeks**, with pharmacological and psychological alternatives weighed in the risk to benefit analysis while it is planned. Three named schedules fill it in, differing in how much is fixed in advance.

SCHEDULE	THE INTERVALS, IN ORDER	WHAT IT IS FOR
STEP-DOWN ECT	One treatment 1 week after the index course is completed, a second 2 weeks after the first, then a continuation protocol if required	Bridging the end of the index course; relapse rates can be lower than with abrupt cessation
FIXED SLOW TRANSITION	Weekly for 4 weeks, then every 2 weeks for 2 months, then monthly thereafter	The trial-derived fixed taper, and the most concrete sequence available
SYMPTOM-TITRATED SCHEDULE	4 continuation treatments over 1 month, then further treatment only as needed under a symptom-titrated algorithm, alongside continued medication	The flexible alternative; it gave better mood outcomes than medication alone at 24 weeks with no significant difference in global cognitive screening

One indication carries a tighter band. In bipolar disorder, more frequent sessions may be required than for other indications, with inter-treatment intervals as short as one to three weeks. The same guideline settles a question that arises there: mood switches occur at a rate similar to that seen with antidepressant medication, concurrent lithium may reduce the risk, and in general a switch is not an indication to stop treatment.

MNEMONIC

TWICE, WEEKLY, FORTNIGHTLY, MONTHLY

The taper in the order it is asked for: from the acute schedule of **twice** a week, down to **weekly**, then **fortnightly**, then **monthly**, and out into the maintenance band. Each step down is earned at a review rather than reached on a calendar, which is why the course is never prefixed.

16.5 The principle that governs every interval

A resident who memorises the intervals and not the principle will schedule the wrong patient wrongly. The guideline states that the optimal timing and frequency of continuation and maintenance treatment is yet to be determined and probably varies between individuals, and gives the aim that orders the section: to extend the inter-treatment interval and minimise the cumulative number of treatments, in order to reduce the risk of cognitive side-effects. What is managed is cumulative exposure; the **inter-treatment interval** is how. The opposite failure is named too: adhering rigidly to a pre-defined transition schedule is problematic, and pursuing a fixed goal of an inter-treatment interval of one month, especially too quickly, may result in relapse, particularly in patients with rapid-cycling bipolar disorder, or depressive illness with a history of early relapse after treatment.

RULE **The maintenance course is scheduled to the patient, not to a target interval.** Lengthening the interval keeps cumulative treatments low, which is what limits cognitive risk; but a fixed transition pursued too fast precipitates relapse in the patients who needed treatment most. One practice avoids both errors: set the next interval at a review of this patient, not in advance.

16.6 Maintenance ECT is not given alone

Medication continues through the maintenance course rather than being replaced by it. The guideline directs that maintenance treatment should be given in conjunction with an antidepressant, unless there is a specific contraindication such as poor tolerability in an individual patient, and that lithium as a relapse-prevention strategy after a course has a strong evidence base and can be used together with maintenance treatment. The interval does one more job here: lithium is safer during maintenance than across an acute course precisely because treatments are much further apart, fortnightly to monthly rather than two to three times a week. Why lithium is a concern across an acute course, and how the threshold-moving drugs are handled around each treatment, are this chapter's account of concurrent medication and drug interactions.

16.7 The stimulus dose at maintenance: an open question, not a schedule

What the threshold is, and how titration measures it, is this chapter's account of stimulus dosing and the seizure threshold. The question that follows belongs here: when sessions are weeks apart, is the dose set at the original titration still the right dose? It is a real question because the threshold moves. It rises across a course of frequently repeated treatments and returns to baseline 6 months after treatment stops, leaving the continuation and maintenance window, longer than a few days and shorter than 6 months, unexamined. Three pieces of evidence bear on it, and none is a rule.

- **The series that framed it.** A retrospective study of 19 patients whose treatments were separated by at least 14 days found significant changes in seizure duration when treatments were separated by more than 60 days, read by its authors as **a fall in threshold or a loss of anticonvulsant action after 2 months, and as an indication to re-titrate after that time**. Its limits are part of the answer: the measured quantity was seizure duration, not the threshold, and the design was retrospective and small.
- **Lowering the dose on purpose.** A unit whose device was capped at 614 mC, 60% of the maximum possible output, asked whether the lower dose gave its maintenance patients better seizure quality, which is the direction that makes the positive finding an argument for re-titration rather than against it. In 15 patients, lowering the energy delivered increased seizure quality overall, which the authors read as an argument for re-titration during maintenance.
- **What is actually done.** In usual clinical practice a constant stimulus dose is delivered through the maintenance course, and the threshold is not re-titrated even when the interval between two successive sessions is longer. The same report describes a stable long-term maintenance patient whose threshold had fallen, which is why the practice is worth questioning.

Evidence on the effect of continuation and maintenance regimens on the threshold is scant, and there are no clear guidelines on the duration after which it has to be recalculated; the authors suggest periodic re-titration, which would prevent unwarranted high stimulus dosing and thereby reduce the risk of cognitive adverse effects.

TRAP **Quoting a re-titration interval as though a guideline had set one.** The figures come from a small retrospective series and a single-unit report, and the sources state that no clear guideline exists. Answer with the shape of the question: the dose is usually carried forward unchanged, the threshold may have moved by the time it is used, and periodic re-titration is reasonable practice rather than a schedule.

16.8 Cognitive monitoring across a maintenance course

The obligation is stated as an interval, and examined in that form. Patients receiving continuation or maintenance treatment must have regular cognitive assessment with a standardised instrument, every 3 to 6 months, or more frequently if significant or worsening deficits are identified, or if the patient is on lithium. The **standardised cognitive assessment** is the instrument; the interval lever is the intervention. What the deficits are, how anterograde and retrograde amnesia differ in mechanism, course and prognosis, and how far a retrograde deficit persists, are this chapter's account of the cognitive side effects.

The monitoring is required because the evidence does not settle the question. Screening scores remain unchanged or increase during continuation or maintenance treatment. But neuropsychological testing of 11 patients treated for depression for an average of 3 years, with almost 2 months between treatments, identified deficits in learning and frontal function compared with controls, while studies of treatment given across a lifetime have shown no evidence of cumulative cognitive deficit. A screening instrument and a neuropsychological battery do not ask the same question, which is the likeliest reason they disagree, and why the obligation is to measure this patient.

GOOD TO REMEMBER

Three things are checked at every maintenance review: whether the illness is still quiet at the current interval, whether cognition has been formally assessed within the required window, and whether the patient is on lithium, which tightens that window. If the interval has been stretched and symptoms have broken through, shorten it and review again before concluding the treatment has failed.

16.9 How long it runs, and what ends it

No source located sets a fixed duration for maintenance treatment, and inventing one is how this question is usually answered wrongly. What is set instead is review: the need for maintenance treatment must be regularly reviewed and documented, and plans to continue or to cease should be made in consultation with the patient and their family. In that guideline's jurisdiction statutory consent also expires and must be renewed, turning the review into a fixed stop. The Indian position arrives there by another route: consent is taken again before continuation or maintenance begins, and the course is planned dynamically at periodic review rather than prefixed.

IN INDIA

Quote the Indian Psychiatric Society guideline of 2023: continuation and maintenance divided at the six-month line; the taper from twice a week to weekly, to fortnightly, to monthly; maintenance at one session every one to twelve weeks; no prefixed course, planned dynamically at periodic review; and fresh consent before continuation or maintenance begins, because the condition, the purpose and the character of the treatment have all changed.

PITFALL

Carrying a foreign statutory consent expiry into the Indian frame. One guideline's review point is a consent validity period set by that state's own mental health legislation, not an Indian rule. The Indian requirement is fresh consent before continuation or maintenance begins, with no stated expiry.

Relapse is the rule when nothing active follows a successful course, so continuation pharmacotherapy is started or continued in every case, and continuation electroconvulsive therapy is an alternative to it rather than an upgrade. The taper runs from twice a week to weekly, to fortnightly, to monthly, then into a maintenance band of one session every one to twelve weeks, tighter in bipolar illness; it is never prefixed, the aim is to lengthen the interval and minimise cumulative treatments, and the course is held to standardised cognitive assessment every three to six months and to a documented review deciding each time whether it continues.

17 · Drug interactions and concurrent medications with ECT

Concurrent medication is the part of an electroconvulsive therapy course that is settled before any current is set, because almost every patient arrives already taking drugs that change the seizure, change the anaesthetic, or change what the treatment does to them. A drug interaction here is never one fact. It carries a direction on the seizure, a consequence for the patient, and a management step, and the three come apart: a drug can shorten every seizure without harming anyone, and another can be dangerous while leaving the seizure untouched.

Owned here is the interaction: which drug moves the seizure in which direction, what follows, and how each class is managed. Named and not re-derived, each owned elsewhere in this chapter: the threshold itself and how a stimulus is set against it, by the account of stimulus dosing and the seizure threshold; the induction agents, the relaxant and the airway, by the account of the pre-ECT workup and anaesthesia; postictal confusion and amnesia, by the account of the cognitive side effects; and the continuation drug and maintenance schedule, by the account of continuation and maintenance ECT. No titration method and no induction dose appears here.

24 hours	600 mg a day	0.6
THE INDIAN PRE-TREATMENT DISCONTINUATION INTERVAL FOR THEOPHYLLINE	THE CLOZAPINE BAND AT WHICH THE THRESHOLD IS KNOWN TO FALL	THE ELDERLY SERUM LITHIUM LEVEL, IN MILLIEQUIVALENTS PER LITRE, ABOVE WHICH CONCOMITANT LITHIUM IS AVOIDED; THE GUIDELINE'S OWN SPELLING OF THAT UNIT IS DISCUSSED BELOW

17.1 Four classes, four different consequences

The controlling Indian statement asks for the psychotropic drugs that can interfere with anaesthesia and treatment to be evaluated beforehand, and it maps four classes to four distinct consequences:

- anticonvulsants increase the seizure threshold;
- antipsychotics such as chlorpromazine and clozapine are **pro-convulsant**, which is to say they lower it;
- lithium increases the risk of postictal delirium;
- tricyclic antidepressants increase the risk of cardiac adverse events during treatment and anaesthesia.

Read that for its structure, not as a list of drugs. Only the first two entries are threshold statements. The lithium entry is about the brain after the seizure and the tricyclic entry about the heart during the anaesthetic; neither says anything about how easily a seizure is produced.

RULE **State the direction on the seizure and the consequence for the patient as two separate claims, then the management step.** A drug that raises the threshold threatens the treatment: the seizure may be short or absent and the course may fail. A drug that lowers it threatens the patient: the seizure may not stop. A drug that does neither may still be the most dangerous thing on the chart.

17.2 The morning list, one instrument in two halves

The Indian guideline gives the bedside decision as two lists read together. On the **withhold half**, the morning dose of oral hypoglycaemics, lithium, diuretics, anticonvulsants, theophylline and benzodiazepines can be withheld, with the requirement considered on an individual basis. On the continue half, scheduled antihypertensives, thyroxine, antiemetics, antireflux drugs, antianginals, bronchodilators and anticholinergics should not be withheld and can be taken orally 2 hours before the procedure with small sips of water.

The withhold half is not a threshold list: the oral hypoglycaemic is held because the patient is fasting and the diuretic because of volume and electrolytes, neither having anything to do with the seizure. The continue half holds blood pressure, thyroid state, gastric acid, coronary perfusion and airway calibre, whose omission destabilises a patient about to be anaesthetised. Withholding one of those that morning is as much an error as continuing an interacting drug.

17.3 The drugs that raise the threshold

Benzodiazepines increase the seizure threshold, decrease seizure duration, decrease the potential efficacy of treatment and possibly increase cognitive side effects. The third of those matters most:

it threatens whether the course works, not merely seizure length. Where the drug has been taken routinely the impact on adequacy may be smaller; it also adds sedation at induction.

Stop the benzodiazepine before the course where possible; a long-acting one may need some days. Where abrupt cessation risks a withdrawal seizure, taper and cease early in the course; that is safe because treatment is itself anticonvulsant. Non-benzodiazepine hypnotics act the same way close to a treatment. Where the drug cannot be stopped, two workarounds are named: a higher stimulus, whose setting belongs to the account of stimulus dosing and the seizure threshold, or reversal. Flumazenil 0.4 to 1.0 mg intravenously immediately before treatment is that reversal, a prescribing decision owned by the anaesthetist and not taught here, for the patient on high doses or unable to tolerate discontinuation; the figure is the anaesthetist's prescribing decision, printed so the psychiatrist knows what is being reversed, not as an instruction issued here.

Anticonvulsant mood stabilisers behave as their name promises: threshold up, seizure expression and duration down, efficacy possibly reduced. Where the anticonvulsant is only augmenting an antidepressant it should be withdrawn before the course begins. In bipolar disorder that is balanced against the risk of a manic swing. If it is continued, the dose can be reduced to the lower end of the therapeutic range, and it is common practice to withhold the evening dose before each treatment and delay the morning dose until after recovery. The patient taking it for **epilepsy** is handled with the neurologist and carries an instruction that must never be dropped: the dose may fall further as the course proceeds, because treatment is itself potently anticonvulsant, and it must be restored to pre-treatment levels when the course ends.

TRAP **Teaching anticonvulsant reduction as settled practice.** The same guideline that advises withdrawal records the evidence against it: full-dose anticonvulsant through a course of bitemporal treatment gave faster recovery in bipolar disorder without deterioration in seizure quality (Rakesh and colleagues, 2017), and continuing sodium valproate did not affect seizure duration or recovery from mania (Haghighi and colleagues, 2013). The direction of the class effect is not in doubt; the instruction to reduce or withhold is contested for mania and should be written as contested.

17.4 Lithium, the interaction most often examined

No source carried here states that lithium moves the threshold in either direction, which is not the same as a finding that it leaves the threshold alone, and the silence is why it is misfiled so often. Its mechanism sits at the brain: treatment increases the permeability of the blood brain barrier and lets more lithium in at an unchanged serum concentration. With a narrow therapeutic index that is enough to produce toxicity in a patient whose blood level reads as therapeutic, and reports of severe confusion concentrate at the higher end.

The full interaction carries four consequences: delirium, prolonged seizures, toxic lithium levels and **prolonged neuromuscular blockade**. The last is not a psychiatric event; the relaxant and the management of a prolonged block belong to the account of the pre-ECT workup and anaesthesia, and it is named here so it is discussed with the anaesthetist beforehand rather than discovered on the list. The same source states, against a common teaching, that the combination

is not an absolute contraindication and may be considered in certain EMERGENCIES where the benefits clearly outweigh the risks. The emergency restriction is the source's own and is the difference between a narrow carve-out and general permission.

Indian practice attaches a figure to the elderly patient: concomitant high-dose lithium **above 0.6** is avoided because of the higher risk of postictal delirium, and the same figure returns in the management of cognitive adverse effects, as the level above which lithium should be reduced or stopped. Both are population-qualified; neither is a general prohibition. The unit takes one sentence of its own, because the guideline is not consistent about it: it spells the unit 'mq/l' in the elderly bullet and 'meq/l' in the cognitive bullet, both mean milliequivalents per litre, the first is a typographical error, and both renderings are reproduced here rather than replaced by the unit this chapter believes was meant, since silently correcting a source is how a wrong number enters a chapter.

Suspend lithium before the course if clinically safe. If it is maintained, omit the evening dose before each treatment and delay the morning dose until after recovery, or run a lower regular dose at the lower end of the range. In bipolar disorder the risk of a manic swing on withdrawal makes this a weighing rather than a default. Case reports in ELDERLY patients indicate that holding lithium for only 24 hours may still leave a level sufficient to contribute to delirium, and the population is part of the finding rather than a detail, so an omitted dose is not by itself a guarantee.

17.5 The drugs that lower it, and a divergence about theophylline

On the other side of the ledger, theophylline and bupropion can increase seizure duration and should be ceased before the course commences where possible. Theophylline is the more dangerous because of where it is met: a patient treated for asthma or chronic obstructive pulmonary disease, where the drug is doing necessary work and cannot simply be deleted.

Two grounded sources handle it differently and this account states the divergence rather than choosing. The Indian guideline gives a flat interval: theophylline is to be discontinued 24 hours before treatment because of the risk of prolonged seizures and status epilepticus. The second source gives no interval at all, says only that it should be ceased where possible, and names bupropion in the same breath. Carry the Indian interval into an Indian examination, and notice that the other source stops the drug without a clock and adds one the Indian sentence does not.

The interaction literature sharpens this. There are reports of status epilepticus after treatment, with high theophylline levels during treatment associated with status epilepticus, making this level-dependent rather than a presence-or-absence signal. Its management ladder ends somewhere more useful than stopping: taper the theophylline and continue bronchodilators and inhaled glucocorticoids, or reduce to the lowest therapeutic level and monitor closely, treating an exacerbation in the standard way before proceeding. That is substitution rather than withdrawal, and it is the asthmatic patient's answer. One thing must not be merged into it: the Indian guideline names theophylline again after a prolonged seizure has happened, as something to stop temporarily, and that is reactive, not a second version of the prophylactic interval.

17.6 Antipsychotics continued through a course

Antipsychotics are commonly continued and are generally well tolerated. Two hazards are named. Clozapine has many reports of safe use and also a risk of prolonged seizures and confusion, because it lowers the threshold; patients on it may also be at risk of tachycardia and of aspiration from hypersalivation. Typical antipsychotics can produce electrocardiographic abnormalities, QTc prolongation in particular.

The clozapine effect is dose-dependent and the band is stated: the threshold falls **usually at doses above 600 mg a day**, and a dose reduction before treatment may be considered on that basis. That figure is a prescribing decision belonging to the treating team, printed only to locate where the threshold effect becomes real; the indication for combining clozapine with treatment belongs to the account of the indications for ECT.

17.7 Antidepressants continued through a course

Antidepressants have generally been administered safely during treatment, with risks and benefits discussed with the patient; the class differences are what an examination wants. Tricyclic antidepressants carry reports of adverse cardiac effects, specifically QRS prolongation, atrioventricular block and hypotension, together with confusion and excessive sedation. Venlafaxine carries reports of prolonged asystole and hypotension, with a dose signal: a possible increased risk of asystole **at doses above 300 mg a day**, again a prescribing matter for the treating team rather than an instruction here. The counterweight travels with it: a controlled study found nortriptyline and venlafaxine not associated with an increase in cardiac events compared with placebo (Sackeim and colleagues, 2009). Selective serotonin reuptake inhibitors are generally considered safe.

Monoamine oxidase inhibitors carry a rule rather than a caution. Their use with treatment has been associated with blood pressure changes, hyper-reflexia and seizures, and the patient taking one is at increased risk of hypertensive crisis if given a sympathomimetic agent or pethidine, so those agents are avoided during the course. Bupropion belongs with the threshold-lowering drugs above, and status epilepticus has been reported with its concomitant use.

One fact points forward. The majority of patients with a major depressive episode who respond are likely to relapse within six months without continuation pharmacotherapy, and it is standard practice to commence an antidepressant, usually from a different class, during or just before the end of the course. Which drug, on what schedule, and the maintenance course itself belong to the account of continuation and maintenance ECT.

17.8 The anaesthetic interactions

Several interactions above reach into the anaesthetic room: sedation at induction from the benzodiazepine, prolonged action of neuromuscular blocking agents from lithium, cardiac events from the tricyclics and venlafaxine, and two agents barred by the monoamine oxidase inhibitor rule.

The one a resident meets is in an elderly patient on a dementia drug. Acetylcholinesterase inhibitors may potentiate the bradycardia produced by the electrical stimulus and by

suxamethonium, and they potentiate its muscle relaxant effect, particularly rivastigmine, which also inhibits butyrylcholinesterase, the enzyme that metabolises suxamethonium. The relaxant and the management of a prolonged block are the pre-ECT workup and anaesthesia account's in full; what belongs here is naming the drug that causes it, so its continued use is discussed with the anaesthetist rather than assumed. There are numerous case reports of the safe use of donepezil and rivastigmine during a course, and a systematic review indicated rivastigmine may even protect against treatment-induced cognitive impairment (Henstra and colleagues, 2017): an interaction to plan around, not a reason to stop the drug.

17.9 The ledger in one table

DRUG OR CLASS	DIRECTION ON THE SEIZURE	CONSEQUENCE	MANAGEMENT ACROSS A COURSE
BENZODIAZEPINES AND Z-DRUGS	Threshold up, seizure shorter	Course efficacy threatened; possibly more cognitive burden; sedation at induction	Stop early, long-acting ones days ahead; taper if withdrawal seizures threaten; else higher stimulus or flumazenil
ANTICONVULSANT MOOD STABILISERS	Threshold up, expression and duration down	Efficacy possibly reduced	Withdraw if only augmenting; balance against manic relapse; else low in range, doses withheld around treatment; restore at the end in epilepsy
LITHIUM	Not stated by the grounded sources	Postictal delirium, prolonged seizures, toxicity, prolonged neuromuscular blockade	Suspend if safe; else omit the evening dose, delay the morning dose, or run low in range
THEOPHYLLINE	Seizure longer; a threshold direction is not stated by the grounded sources	Prolonged seizure and status epilepticus, level-dependent	Indian interval beforehand, or taper with inhaled therapy substituted; sources diverge
BUPROPION	Seizure longer; a threshold direction is not stated by the grounded sources	Status epilepticus reported	Cease beforehand where possible; no interval carried
ANTIPSYCHOTICS (CLOZAPINE, CHLORPROMAZINE)	Threshold down (chlorpromazine and clozapine)	Prolonged seizures and confusion; tachycardia and aspiration risk with clozapine; QTc prolongation with typical agents	Usually continued; dose reduction considered in the high clozapine band
TRICYCLICS AND VENLAFAXINE	Neither direction	Cardiac events and confusion; asystole reports with venlafaxine, unconfirmed in a controlled study	Continued after weighing risk and benefit
MONOAMINE OXIDASE INHIBITORS	Neither direction	Blood pressure changes, hyper-reflexia, seizures; hypertensive crisis with sympathomimetics or pethidine	Continued, those agents barred
ACETYLCHOLINESTERASE INHIBITORS	Neither direction	Bradycardia potentiated; block prolonged, most with rivastigmine	Continued after discussion with the anaesthetist

MNEMONIC

UP, DOWN, AND NOT THE THRESHOLD AT ALL

Up: benzodiazepines and anticonvulsants, which threaten the course. **Down**: clozapine and chlorpromazine, theophylline and bupropion, which threaten the seizure ending. **Not the threshold at all**: lithium, the tricyclics and venlafaxine, the monoamine oxidase inhibitors and the acetylcholinesterase inhibitors, whose dangers are delirium, the heart, a crisis and a block. That third group is the one candidates lose.

A drug interaction across a course is three statements: a direction on the seizure, a consequence for the patient, and a management step. Benzodiazepines and anticonvulsant mood stabilisers raise the threshold and threaten the efficacy of the course, and are stopped, tapered or timed around each treatment. Clozapine, chlorpromazine, theophylline and bupropion lower it and threaten a seizure that does not stop. Lithium moves it in neither direction and is still the most examined interaction, because treatment lets more of it into the brain at an unchanged serum level, bringing delirium, toxicity and a prolonged block. Antipsychotics and antidepressants are usually continued, each class carrying a hazard rather than a prohibition. The threshold itself and the conduct of the anaesthetic are other accounts' in full.

Other neurostimulation therapies

18 · Repetitive transcranial magnetic stimulation (rTMS): mechanism, indications, parameters

Repetitive transcranial magnetic stimulation puts an electrical current into cortex without touching the skin, and it is dosed so that the current never becomes a seizure. That second half separates it from everything the electroconvulsive therapy half of this chapter has taught. The examinable weight sits in the parameters, because rTMS is prescribed as a set of values that travel together rather than as a dose, and each is referenced to a measurement taken from the patient's own cortex, the motor threshold, which a candidate must define, obtain and keep separate from the threshold this chapter has already taught.

This account owns rTMS and nothing else, from the induction mechanism to the safety limits. It does not grade rTMS against the other devices of this band; that comparison, across invasiveness, target, reversibility, evidence and Indian availability, belongs to this chapter's cross-cutting synthesis and its band grid, and each of the other five items teaches its own device. The seizure threshold and the stimulus dose belong to this chapter's account of stimulus dosing, the seizure threshold and adequate seizure criteria, and why an induced seizure helps to its account of the mechanism of action and neurobiological theories of electroconvulsive therapy; both are named for contrast, neither re-derived.

5 of 10	120%	7 per 100,000
POSITIVE TRIALS THAT DEFINE THE MOTOR THRESHOLD	OF MOTOR THRESHOLD, THE INTENSITY OF THE CLEARED DEPRESSION PROTOCOL	STANDARDISED SEIZURE RISK PER SESSION, PROTOCOLS POOLED

18.1 Electromagnetic induction, and why the magnetic field is not the stimulus

Electromagnetic induction is the principle the technique rests on, and the chain runs in a fixed order. A high-voltage, high-current discharge system drives a transducing coil, and the discharge produces a strong, time-varying magnetic field at right angles to the coil, peaking at 1 to 2.5 Tesla and lasting under about 1 millisecond. With the coil tangential to the head, that field penetrates scalp and skull with minimal attenuation, and in the conductive tissue beneath it induces a secondary eddy current whose electrical field is perpendicular to the magnetic field and opposite in direction to the current in the coil.

The name of the technique misleads, and its defining source says so. The magnetic field does not stimulate cortical neurons; it serves only to induce an electrical current in neural tissue, and it is that induced electrical current that depolarises cortical axons and triggers action potentials at suprathreshold intensities. At the neuronal level the excitation is the same as with conventional electrical stimulation, and induction buys freedom from direct electrical contact with the skin. Because the brain is not homogeneous, tissue conductivity distorts the induced currents, so exact distributions can be modelled but never read off the coil position.

18.2 Sub-convulsive by design: how rTMS differs from ECT

Conventional therapeutic rTMS is **sub-convulsive** by design, not by accident. The current international safety consensus states the contrast in the form this chapter needs: in conventional rTMS the goal is to select parameters that minimise seizure risk, whereas the modality that uses the same induction to treat by producing a seizure selects parameters efficient at inducing one, and that modality is taught in this chapter's account of magnetic seizure therapy and the emerging neuromodulation techniques. In rTMS a seizure is an adverse event, not the therapeutic mechanism.

Two consequences of that absence must be held apart, because collapsing them is the error this chapter's axis rule forbids. The first is **cognitive**: at therapeutic intensity rTMS does not produce the amnestic syndrome that follows a course of electroconvulsive therapy, and the reason is structural: that syndrome follows the induced generalised seizure, and rTMS induces none. The second is **safety**: an unintended seizure can still occur, at a rate quantified below, and the published safety limits exist to bound it. These are different currencies, and the absence of the first neither abolishes nor excuses the second.

18.3 The motor threshold, and how it is determined

The **motor threshold** is what every rTMS intensity is referenced to, and it is measured in this patient rather than looked up. The international practical guide defines it by a relative-frequency criterion, the Rossini criterion: the lowest stimulus intensity, given as a percentage of maximum stimulator output, required to induce a motor evoked potential in **5 out of 10** trials. It is obtained with single-pulse stimulation at the optimal site over the hand area of the primary

motor cortex. Two thresholds are defined and are not interchangeable: the **resting motor threshold**, read with the muscle relaxed, and the **active motor threshold**, read during tonic contraction, each with its own amplitude criterion. Those criteria, the contraction level and the step sizes of the standardised search are not carried here. The Indian guideline states the same construct as an operator uses it, and adds the caveat that matters in a busy clinic: a threshold read by eye rather than by electromyography comes out significantly higher, and an inflated threshold inflates every intensity derived from it.

AXIS	INTERNATIONAL PRACTICAL GUIDE	INDIAN THERAPEUTIC GUIDELINE
CRITERION	Motor evoked potential in 5 of 10 trials	Response in at least 50 percent of stimuli, usually 5 of 10
MUSCLE	A hand muscle at the optimal motor site	Abductor pollicis brevis or first dorsal interosseous, contralateral
DELIVERY	Single pulse, figure-of-eight coil	Single pulse, graded small to high, every 5 seconds

RULE **The rTMS motor threshold and the ECT seizure threshold are different constructs, and neither may be carried across to the other.** The motor threshold is a corticospinal excitability measurement, read from a hand muscle with single pulses over motor cortex, expressed as a percentage of the stimulator's maximum output, and used to set a deliberately sub-convulsive intensity. The seizure threshold is the stimulus dose at which an adequate generalised seizure is produced, and all of it, including dose expressed as a multiple of threshold, belongs to this chapter's account of stimulus dosing, the seizure threshold and adequate seizure criteria. They share a word and nothing else: different physiology, different instrument, opposite purpose.

GOOD TO REMEMBER

Treat the motor threshold as perishable. It would ideally be measured before every session; in practice the value set before the first may carry through that week. Re-determine it if sessions fall more than a week apart, if medication doses change, after heavy alcohol or substance intake, or if the patient reports headache or scalp pain.

18.4 The stimulation parameters, and frequency

The **stimulation parameters** are target, intensity as a percentage of motor threshold, frequency, train duration with its intertrain interval, and total pulses per session and per course. The two protocols cleared for depression share a target and an intensity and agree on nothing else.

PARAMETER	FIGURE-OF-EIGHT COIL PROTOCOL	H1 COIL DEEP TMS PROTOCOL
DEVICES NAMED	Neuronetics NeuroStar, Magstim Rapid2, MagVenture MagVita	Brainsway Deep TMS
TARGET AND INDICATION	Left dorsolateral prefrontal cortex, major depressive disorder in adults	
INTENSITY	120 percent of motor threshold	120 percent of motor threshold
FREQUENCY	10 Hz	18 Hz
TRAIN DURATION	4 seconds	2 seconds
INTERTRAIN INTERVAL	26 seconds	20 seconds
PULSES (PER SESSION)	**3,000**	1,980

Protocols are conventionally divided at 1 Hz, with **high-frequency rTMS** above that figure and **low-frequency rTMS** at or below it. The direction of effect is the teaching point, not any single value: low frequencies reduce cortical excitability and faster frequencies increase it. Both cleared protocols are high-frequency and left-sided. The practice band of intensities around the labelled figure, and the specific low-frequency value, are not carried here. Train structure carries its own safety-relevant direction: shorter trains with longer intertrain intervals are less likely to induce a seizure, so a protocol is never described by its frequency alone.

MNEMONIC

Target, Intensity, Frequency, Train, Total

The five values that define an rTMS prescription: **Target**, by a stated localisation method; **Intensity**, as a percentage of motor threshold; **Frequency**; **Train** duration with its intertrain interval; and **Total** pulses and sessions.

TRAP **Quoting a pulse count from one protocol beside a frequency from the other, or presenting either tuple as the general settings of rTMS.** A candidate who writes one set of standard rTMS parameters has described a protocol no regulator cleared and no trial ran. Name the coil geometry, the device family and the indication whenever a value is quoted.

18.5 Coils, devices, and finding the target

Coil choice is a focality decision. The **figure-of-eight coil** is the most commonly used and the most focal; H coils serve deep TMS; double-cone and round coils are larger and less focal. The safety consequence is that the larger, less focal designs are more efficient at producing a seizure than the focal figure-of-eight, which is why the seizure-inducing modality of this band uses them and conventional rTMS does not. Whether the choice changes outcome is thinner than it looks: efficacy and acceptability across NeuroStar, MagPro and Magstim in depressive disorders did not differ significantly, on response ($p = 0.12$), discontinuation ($p = 0.84$) or remission ($p = 0.07$). H1 against figure-of-eight coils showed a larger reduction in depression severity in the H1 studies

and a trend to higher remission the other way, differences the authors deem not clinically relevant on a low volume of studies with no placebo control.

Targeting the dorsolateral prefrontal cortex uses four methods:

- The **centimetre rule**, placing the coil a fixed scalp distance anterior to the motor cortex, classically 5 cm, with slightly longer distances also used.
- The International 10-20 electroencephalography system, in which F3 is the left dorsolateral prefrontal site.
- Stereotactic frames.
- Neuroimage-guided frameless neuronavigation.

The consensus preference is explicit: the method most practical in time and accuracy is head measurement to identify F3 using 10-20 coordinates. What moved practice off the centimetre rule was error: in a trial informed by magnetic resonance imaging the coil had to be moved forward to the longest quoted distance in a substantial minority, the proportion not carried here. The Indian guideline agrees, calling neuroimaging more accurate and the 10-20 system a cost-effective alternative.

18.6 Theta-burst stimulation

Theta-burst stimulation patterns the pulses instead of spacing them evenly, and the values a candidate is examined on belong to two different protocols. The original neurophysiological parameters, described in 2005 and used by most subsequent work, are 3-pulse bursts at 50 Hz, the bursts repeated at 5 Hz, the theta rhythm the technique is named for, at 80 percent of the active motor threshold, typically over motor cortex. The therapeutic protocol differs: the pivotal depression trial targeted the left dorsolateral prefrontal cortex and referenced its intensity to the resting motor threshold, the safety update recording it as an exception to the usual intensity range, which is not carried here. The Indian guideline names three sub-forms: intermittent, continuous and intermediate theta-burst stimulation.

TRAP **Reading a theta-burst intensity across from the active motor threshold to the resting one, or quoting the original neurophysiological intensity as the depression dose.** The two reference intensity to different thresholds, so an intensity here is meaningless unless its threshold is stated in the same breath. On safety, the honest position is that a seizure has been recorded with the original parameters, and that theta-burst within the usual intensity range can be regarded as safe.

18.7 The acute course, the indication and the evidence

A course of rTMS is measured in weeks of daily attendance, and the whole course is the treatment. The consensus position is that a standard acute course lasting **20 to 30 treatment sessions over 6 weeks** will very likely be needed to achieve results consistent with the published regulatory trials. Two things follow. The full course, not a fraction of it, reproduces the trial result, so a patient stopped early has not had the treatment that was studied; and extension trials indicate that non-responders to a standard 20 to 30 session course may still respond if daily

sessions continue. The weekly cadence, session length and time to early symptom change are not carried here. The indication both grounded protocols carry is major depressive disorder in adults, under a United States clearance rather than an Indian one; the Indian guideline covers therapeutic rTMS across a wider set of neuropsychiatric disorders whose individual indications and evidence are not enumerated here.

18.8 Adverse effects and the unintended seizure

The adverse event that governs how rTMS is regulated is the **unintended seizure**, a different object from the therapeutic seizure of electroconvulsive therapy in every respect that matters: not sought, not dosed, not monitored for adequacy, not the mechanism of any benefit. Until recently it had never been quantified; a large survey now gives standardised risks. Pooled across protocols the risk is **7 seizures per 100,000 sessions**, over more than 300,000 sessions reported for 2012 to 2016, rising to 33 per 100,000 in subjects at elevated risk from medication, brain lesions or epilepsy. Within high-frequency rTMS the split is starker: 67 per 100,000 in those with risk factors against 1 per 100,000 without, while single, paired and low-frequency stimulation together ran at 8 per 100,000. The same document elsewhere puts the risk at under about 0.03 percent, and records that a TMS-induced seizure has never caused permanent damage in current practice and occurs only during stimulation.

One risk factor was new and is directly operational: lack of previous exposure. Over 62 percent of seizures occurred on the first exposure and 75 percent within the first three, and the Indian guideline carries those figures into Indian practice with the instruction to exercise high precaution during the initial sessions. The limitations belong beside the numbers: self-reported survey data, described by their own authors as no more than approximate and possibly biased in reporting, with numbers in low-risk individuals too small for valid comparison.

18.9 The safety limits, and what the most recent update actually published

Here a chapter can print something that does not exist. The original quantitative safety tables of intensity against frequency and train duration are Wassermann's, augmented and improved in later consensus papers. The most recent international update did not replace them: it decided expressly not to provide a formal update of the safety tables and proposed operational guidelines instead, with one hard ceiling. The parameters used to induce a seizure in the seizure-induction modality of this band, taught in this chapter's account of magnetic seizure therapy and the emerging neuromodulation techniques, must not be exceeded. The usual lowest such combination is **100 percent** of maximal stimulator output at 25 Hz in a single train lasting up to 10 seconds, and every combination of intensity, frequency and duration in conventional rTMS must remain well below it. The guidelines tell an investigator who exceeds the earlier limits what to do rather than what number to stop at:

- Balance risk against benefit explicitly, as the responsible investigator.
- Use neurophysiological monitoring, taking motor twitches as a warning of rising cortical excitation.
- Reconsider the protocol if a seizure occurs.

- Alert the scientific community rather than treating the event as local.

One limitation travels with the older tables: they were derived in healthy young subjects, with figure-of-eight coils only, over the primary motor cortex, and were never a universal map of safe parameters for every coil, target and patient.

PITFALL

Printing a quantitative rTMS safety table and attributing it to the most recent international safety update. That update declined to publish new tables, issuing operational guidelines and a single ceiling instead, so a table carrying its name is an artefact the reader cannot find in the source. Attribute the tables to the earlier work, and say plainly that the current position is operational rather than tabular.

18.10 rTMS in India: the suite and the regulatory position

The Indian guideline sets the equipment baseline for an rTMS suite: main unit, coil, cooling unit and control cable, power supply, electromyography machine, coil holder and computer, with provision for emergency seizure management including stored anticonvulsants and immediately available trained physicians. The sample specification is a stimulator of at least 50 Hz capacity with a burst mode, so theta-burst can be delivered. Note what that seizure provision is: an emergency provision, for an event the prescription is designed to avoid.

IN INDIA

The Indian device position is a checked absence and should be stated as one. The national risk-based classification list enumerates 131 devices in the Neurological category, and no entry corresponds to a repetitive transcranial magnetic stimulation system. Searching it for transcranial returns exactly one entry, a transcranial electrical stimulation system at serial 127, Class B, whose device is taught in this chapter's account of transcranial direct current stimulation. Electroconvulsive therapy systems, deep brain stimulators and vagus nerve stimulators are all listed, each taught in its own item; transcranial magnetic stimulation is not. This concerns device classification only. It establishes nothing about Indian product licensing of any named rTMS device, or any indication-specific authorisation, neither of which may be inferred from a foreign clearance.

rTMS induces a current in cortex by electromagnetic induction and is dosed to stay sub-convulsive, so it does not produce the amnestic syndrome of an ECT course, while an unintended seizure remains a rare, separately bounded safety risk; its intensity is a percentage of the motor threshold, a corticospinal measurement that is not the ECT seizure threshold; and its parameters are quoted only as a whole tuple belonging to a named protocol, coil and indication.

19 · Transcranial direct current stimulation (tDCS)

Transcranial direct current stimulation passes a weak, constant current between scalp electrodes and changes how likely cortical neurons are to fire, without ever making them fire.

That is the whole of its mechanism and its separation from the electroconvulsive therapy half of this chapter: no seizure is induced, none is wanted, and nothing is dosed to produce one. The difficulty is that a treatment this simple to deliver has an evidence base that is not simple at all, so a candidate who quotes the milliamps but not the trial result has learned the easy half.

This account owns tDCS and nothing else: the subthreshold mechanism with its qualification, the montage, the milliamps, the session and the course, the evidence and where it is weak, tolerability, and the home-use and regulatory questions. It does not grade tDCS against the other devices of this band; that comparison, across invasiveness, whether a seizure is induced, target, reversibility, evidence and Indian availability, belongs to this chapter's cross-cutting synthesis and its band grid, and every band item teaches its own device. Electromagnetic induction and the motor threshold belong to this chapter's account of repetitive transcranial magnetic stimulation, and the induced therapeutic seizure to this chapter's account of magnetic seizure therapy and the emerging neuromodulation techniques; each is named for contrast, neither re-derived.

2 mA	null	Class B
THE CURRENT OF THE ONE DEPRESSION PROTOCOL CARRIED HERE, NOT A UNIVERSAL SETTING	WHAT THAT TRIAL FOUND FOR ACTIVE STIMULATION AGAINST SHAM ON ITS ANTIDEPRESSANT OUTCOME	THE INDIAN DEVICE CLASSIFICATION, WHICH AUTHORISES NO PSYCHIATRIC INDICATION

19.1 What tDCS is, and the envelope that defines its family

Transcranial direct current stimulation belongs to a family the current safety and application guidelines call **low intensity transcranial electrical stimulation**, defined by a parameter envelope rather than by a mechanism. The envelope is a total intensity of 4 mA or less, for alternating waveforms measured peak to peak, a total stimulation duration of up to 60 minutes per session, electrode sizes of 1 to 100 square centimetres, and frequencies of 0 to 10,000 Hz. Direct current sits at the bottom of that frequency range, which is why it shares an envelope and a safety literature with the alternating and random-noise members.

The envelope carries arithmetic rules that are easy to breach without noticing. With more than one channel, the total intensity is the sum across all electrodes of the same polarity; with more than one session in a day, the total duration is the sum of the sessions. A montage or a schedule can therefore leave the envelope while every reading on the device looks compliant.

RULE **The envelope is a convention, not a safety limit, and its own source says so in terms.** The guideline that defines the family states that its parameter limits rest on convention and do not represent formal safety limits. Printing them as the safe maximum inverts the claim: they mark where the accumulated evidence stops applying, not where injury begins. Quote the envelope with that caveat attached, or do not quote it.

19.2 The subthreshold mechanism, and why polarity is not destiny

Low intensity stimulation modulates brain activity primarily through **subthreshold neuronal polarisation**. The induced electric field shifts membrane potentials without directly causing action potentials, making neurons more or less likely to fire in response to ongoing synaptic input, and through that shift it can influence synaptic efficacy, alter neuronal excitability and shape network-level oscillatory dynamics. Everything the technique does, it does to a cell already

receiving input: there is no step at which the device fires a neuron. That is the structural contrast with electroconvulsive therapy, prescribed precisely to induce a generalised seizure, and with the induction technique of this chapter's account of repetitive transcranial magnetic stimulation, not re-derived here.

The rule every candidate carries, that the anode excites and the cathode inhibits, is a simplification and has to be named as one. The guideline gives the mechanism and its qualification in one passage: tDCS is explained by a sustained polarity-specific change in membrane potential, and the mechanisms of low intensity stimulation vary with the current properties and with electrode placement. tDCS also always has at least one anode and at least one cathode, so every montage is active at each end and inert at neither. Direction and magnitude are therefore modified by montage, current density, duration and the state of the tissue, and the intensity at which the polarity relation stops holding is not carried here.

RULE **Polarity is not destiny: write the anodal and cathodal directions as a first approximation that names its own conditions.** The relation is real, and it is why a montage is described by which pole sits where. But the source states it beside the statement that mechanisms vary with current properties and electrode placement, so a sentence giving the direction without the modifiers has promoted a conditional finding into a law the source does not assert.

19.3 The montage, and the electrode that is not a return

The **montage** is the geometry: which electrode goes where, of what size, and with what current between them. Montage primarily shapes how current flows through the brain, while electrode design primarily affects tolerability, so a change made for comfort is not neutral to physiology. Since the family's electrodes span 1 to 100 square centimetres, and **current density** is current divided by the area it crosses, electrode size is part of the dose and not a detail of the kit: the same milliamps at a smaller electrode is a denser current at the skin and beneath it.

The fully specified montage carried here comes from a named trial, not a textbook default: the anode over F3 and the cathode over F4, a bifrontal arrangement over the prefrontal cortices. Read it as one protocol's geometry, tied to its indication and device, and notice what the **return electrode** is doing. It is not disposing of current at a neutral site; it sits over cortex and it stimulates. That is why the montage rather than the anode gets named, and why moving the second electrode changes the treatment.

19.4 Current, session duration and the course

tDCS is prescribed as a tuple, and the milliamps mean nothing on their own. In the DepressionDC trial, active stimulation was a constant **2 mA** direct current lasting 30 minutes, given as 24 sessions within 6 weeks, in adults with major depressive disorder not responding to a selective serotonin reuptake inhibitor, the stimulation added to that antidepressant rather than replacing it. The trial was triple-blind, randomised and sham-controlled across eight psychiatric centres in Germany, the device a named mobile direct-current stimulator, and the **sham** a ramp-up and ramp-down sequence reproducing the skin sensation without the treatment current.

AXIS	THE DEPRESSIONDC PROTOCOL	THE LOW INTENSITY FAMILY ENVELOPE
CURRENT	Constant 2 mA direct current	Total intensity 4 mA or less
SESSION DURATION	30 minutes	Up to 60 minutes per session
ELECTRODES	Anode F3, cathode F4	Sizes 1 to 100 square centimetres
COURSE	24 sessions within 6 weeks	Not addressed: the envelope bounds a session, never a course
WHAT IT IS	One trial's protocol, one indication, one device	A stated convention, expressly not a formal safety limit

TRAP **Printing one trial's current and session length as the settings for tDCS.** The values in the left column travel together: one protocol, one indication, one device, one montage. Quoted alone they describe a setting rather than the treatment that was tested; quoted beside the right column they invite the reader to treat a convention as a prescribing range. Name the trial, montage, indication and device whenever a milliamp figure is written down.

MNEMONIC

Montage, Milliamps, Minutes, Sessions

The four values that make a tDCS prescription, none meaning anything without the other three and the indication they were tested in: **Montage**, both electrodes named by site; **Milliamps**, with the electrode size that turns current into current density; **Minutes** per session; and **Sessions**, over a stated number of weeks.

19.5 The evidence, and where it is weak

The honest summary is that the evidence for tDCS is heterogeneous, and the trial this account carries in full is a negative one. DepressionDC was a triple-blind, randomised, sham-controlled multicentre trial of tDCS added to a selective serotonin reuptake inhibitor in major depressive disorder, reported in 2023, and its investigators state their own result plainly in a later peer-reviewed report of the same trial: the main trial did not show beneficial antidepressant effects of active tDCS over sham. That is the sentence to carry into an answer: it is the investigators' own and it is not hedged.

The rest of that record matters more than a summary verdict. The same secondary analysis found no group difference in neurocognitive change and no predictive value of baseline cognition, so in this trial the technique bought neither antidepressant benefit nor cognitive cost. That is the actual relation for this parameter in this trial, stated as such, with no general direction imported from elsewhere in this chapter. The blinding problem is structural rather than incidental: electrode designs that produce less sensation blind better, so sham reliability is not comparable across studies using different electrode designs, which means a pooled figure assembled across them pools degrees of blinding as well as montages.

What this account will not do is convert one trial into a verdict on the technique. No pooled estimate and no second trial are carried in the allowed claims, so the wider literature is called heterogeneous rather than summarised with a figure this fragment cannot ground. The primary report is paywalled and was not read at origin, so the quotation above is attributed to the investigators' open-access analysis.

PITFALL

Treating tolerability as evidence of efficacy. tDCS is cheap, portable, painless for most patients and easy to sham, and each is a reason it has been trialled widely rather than a reason it works. An answer that lists the practical advantages and then asserts antidepressant efficacy has skipped the step being tested. Name the negative trial and let the advantages explain the research interest rather than the conclusion.

19.6 Tolerability, and what actually causes a skin lesion

What patients report is local and mild:

- tingling, itching and warmth beneath the electrodes;
- sometimes described as burning or pressure;
- transient, subsiding shortly after stimulation;
- most prominent in the early sessions and habituating with repetition.

Because the sensation is what a patient notices, it is also the main threat to blinding, which is why a sham reproduces the ramp instead of leaving the device off.

The useful claim about skin injury is that it is a technique and equipment problem rather than a current problem. The guideline states that skin lesions are not an expected adverse event when best practices are followed and appropriate equipment is used, and that essentially every case report of a skin lesion during tDCS was correlated with poor technique or poor technology. Framed that way the claim tells a resident what to do, which a bare incidence figure would not.

GOOD TO REMEMBER

The practice points are specific. Prepare the skin by cleaning and drying it, and do not abrade it. Use electrodes verified for the stimulation being delivered, not electroencephalography recording electrodes and not transcutaneous nerve stimulation pads from another trolley. Read impedance as quality control and read it honestly: a low impedance is neither necessary nor sufficient for tolerated stimulation, so it confirms the setup rather than certifying the session.

19.7 Home-based tDCS, and the adjective that carries the safety data

Home use is the first question a patient asks about a portable device, and the answer turns on one adjective. A large-scale tolerability dataset, which the source describes in those terms and not as the largest, is an analysis of **6,779 sessions** of **remotely supervised at-home tDCS** delivered in clinical trial contexts. It reported no serious side effects and a session abort rate of 0.04 percent, the commonest side effect being mild tingling, itching and warmth distributed equally across active and sham. The guideline holds that remotely supervised home-based stimulation is safe

within the standards it sets, under medical prescription or in research settings, and that it buys accessibility and scale rather than any change in effect.

TRAP **Reading the home-use safety data across to an unsupervised consumer device.** Every session in that dataset was remotely supervised and delivered inside a clinical trial, with the equipment, montage, dose and reporting a trial imposes. Direct-to-consumer devices are discussed separately in the same guideline and this dataset is not extended to them. A patient asking whether a headset bought online is safe is not asking a question this evidence answers, and the reply names the supervision condition, not the session count.

19.8 The Indian position: a classification is not an authorisation

India's regulator has classified the device, which is a smaller statement than it appears, so the halves are held apart. The national classification of non-notified medical devices carries an entry for a transcranial electrical stimulation system, continuous current and pulsed current, intended for one or more psychiatric or neurological therapy types, and the intended-use text of that entry names transcranial direct current stimulation and transcranial alternating current stimulation expressly. The tail of the same entry names anxiety, depression, insomnia and addiction among those intended uses and assigns the device a risk class of B.

What that entry establishes is device classification and nothing more. Indication-specific authorisation is recorded separately, and it is recorded as absent: the current international guideline surveyed national regulatory positions and reports that no country in Asia has officially approved transcranial electrical stimulation devices as medical devices, except Singapore and South Korea. In that passage India appears only for its classification system, running from Class A at the lowest risk to Class D at the highest under the Medical Devices Rules of 2017. The absence is stated from a source rather than inferred, and approvals granted elsewhere are never read across to India.

IN INDIA

Write the Indian position as two lines, and never let the first stand in for the second. First: a transcranial electrical stimulation system exists as a classified device category, at a stated risk class, with intended-use text naming direct current stimulation and psychiatric conditions. Second: no indication-specific authorisation for such a device is established in India, and Indian product licensing of any named device is not established by this record either. A classification tells a manufacturer which regulatory pathway a device sits in; it does not tell a psychiatrist that an indication has been authorised.

RULE **A device classification is not an authorisation of an indication, and the two come from different sources.** Classification answers how a device is regulated; authorisation answers whether it may be marketed for a stated clinical use. Print only the classification and the reader supplies the missing half, and the half a reader supplies is always the permissive one.

tDCS shifts membrane potentials below the threshold for firing, so it changes the probability of a response to ongoing input and induces no seizure; its polarity rule is a first approximation conditioned by montage, current density, duration and tissue state, with both electrodes active; its dose is a tuple of montage, milliamps, minutes and sessions belonging to a named trial; its evidence is heterogeneous and the trial carried here is negative; and in India it is a classified device, which is not an authorised indication.

20 · Deep brain stimulation (DBS) in psychiatry

Deep brain stimulation is a permanently implanted electrode left switched on inside the brain, and in psychiatry it is the rung reached only after everything else has failed. The examinable weight sits in three places. The targets have names, and earlier chapters have already promised the reader those names. The sham-controlled depression trials built to settle the depression question did not meet their primary endpoints, which is a fact to be stated rather than managed. And the Indian legal position turns on a question the statute itself leaves open.

This account owns the psychiatric use of deep brain stimulation in full: the implanted system, the circuit rationale, the targets by name, the indications in refractory obsessive-compulsive disorder and refractory depression with the evidence as it reads, the parameters and programming, the adverse effects, and patient selection with the ethical and regulatory frame. It does not grade this device against the others of its band; that comparison belongs to this chapter's cross-cutting synthesis and its band grid. The obsessive-compulsive treatment algorithm that delivers a patient to this rung, and the cortico-striato-thalamo-cortical circuit as obsessive-compulsive neurobiology, are the Anxiety, OCD and trauma-related disorders notes'. The mood, impulse-control and cognitive sequelae of deep brain stimulation given for Parkinson disease are the Neuropsychiatry of systemic and neurological disease notes'. Ablative surgery and its irreversibility belong to this chapter's account of ablative psychosurgery, and the implanted but non-cranial vagus nerve system to its own item. Each is named here; none is re-taught.

VC/VS	8 of 9	130 to 180 Hz
AMONG THE MOST FREQUENTLY INVESTIGATED TARGETS IN OBSESSIVE-COMPULSIVE DISORDER, NAMED JOINTLY WITH THE NUCLEUS ACCUMBENS	GUIDELINES PLACING DBS AFTER ALL ELSE HAS FAILED	STIMULATION FREQUENCY ACROSS THE PSYCHIATRIC LITERATURE

20.1 The implanted system, and why it is no longer called a lesion

Deep brain stimulation is a chain of hardware a candidate should be able to draw. Intracerebral electrodes are placed by stereotactic technique, connected subcutaneously by extension leads, and driven by an **implantable pulse generator** sited in the subclavicular region or the abdomen. Nothing is removed and nothing destroyed: the generator is programmed from outside the skin, its settings revised at any visit, and the intervention ends when the device is switched off.

That is the fact that keeps this item apart from ablation. Stimulation was initially conceptualised as a reversible lesion and is now better described as a **circuit therapy**, and the mechanism as now stated is not inhibition of a target but replacement of a pattern: electrical pulses trigger action

potentials in axons both orthodromically and antidromically, generating new patterns of synaptic activity that overwrite the pathological ones. The history follows the same logic: stimulation entered psychiatry in 1999, when a Belgian group led by Nuttin reported stimulation of the anterior limb of the internal capsule in a patient with refractory obsessive-compulsive disorder, following the 1987 report of thalamic stimulation for parkinsonian tremor.

RULE **Deep brain stimulation and ablative psychosurgery are different kinds of intervention, and the difference is not one of degree.** The published framing of the choice is exactly this trade: the irreversibility and relative simplicity of lesioning against the reversibility, adjustability, and costs associated with stimulation. Ablation destroys tissue, cannot be undone, and carries no device to fail. Stimulation leaves the anatomy intact and can be titrated or stopped, and buys that with a foreign body, a battery, a programming clinic and a failure mode the lesion lacks. Write neither as the more advanced version of the other; the ablative procedures are this chapter's own account of ablative psychosurgery.

20.2 The circuit rationale, and the targets by name

The targets are what earlier chapters have already promised, so they are delivered here in full and split by indication, because the obsessive-compulsive target set and the depression target set are not the same set. For obsessive-compulsive disorder, the ventral capsule and ventral striatum and the nucleus accumbens have emerged as the most frequently investigated loci, with the anterior limb of the internal capsule, the bed nucleus of the stria terminalis and the subthalamic nucleus also receiving substantial attention; less conventional targets include the inferior thalamic peduncle, the caudate nucleus and various thalamic nuclei. For depression, the best studied targets have been the ventral capsule and ventral striatum, the subgenual or subcallosal cingulate cortex (Cg25), and the medial forebrain bundle.

Each target was chosen from a circuit argument, and those arguments explain why one target serves obsessive-compulsive disorder and another depression. The ventral capsule and ventral striatum, including the nucleus accumbens, is a key node of the reward circuit, with afferents from the ventral tegmental area and efferents to multiple limbic structures, and stimulation there was hypothesised to relieve anhedonia. The subcallosal cingulate was proposed as a mood-regulating hub on the basis of functional-imaging hyperactivity in depression. The superolateral medial forebrain bundle connects the ventral tegmental area with ventromedial prefrontal and orbitofrontal cortex, and its fibres course through the anterior limb of the internal capsule. That last detail is why one trajectory recurs under several target names: these are neighbouring parts of one white-matter corridor, not scattered points.

The field has also moved, and the direction is examinable: the preferred stimulation site within the anterior limb of the internal capsule has shifted posteriorly with tractography, and the emphasis has moved from grey-matter nuclei to white-matter convergence zones. A resident who learns a target as a nucleus has learned the older version of the idea.

MNEMONIC

VANS

Ventral capsule and ventral striatum, **A**nterior limb of the internal capsule, **N**ucleus accumbens, **S**ubcallosal cingulate: the psychiatric electrode targets an examiner expects by name. The first three cluster in one ventral corridor; ventral capsule and ventral striatum carries both the obsessive-compulsive work AND one of the two depression trials, and the subcallosal cingulate is the other. Do not let the corridor grouping become an indication grouping: the target sets overlap, which is the point the fragment makes below.

20.3 Refractory obsessive-compulsive disorder: where the guidelines actually put it

The honest answer to whether psychiatric DBS is recommended is a shape, not a yes. A systematic review of the guidelines for obsessive-compulsive disorder found that 9 guidelines were identified, 8 recommended that DBS is used after all other treatment options have failed, and one did not recommend DBS beyond a research setting. The gaps in that literature matter as much as the placement: only one guideline performed a cost-effectiveness analysis, and the other 8 did not provide details on safe or effective DBS protocols. A guideline that endorses a procedure without specifying how to deliver it endorses a direction, not a technique. The neurosurgical consensus is blunter: in its 2021 consensus guidelines the World Society for Stereotactic and Functional Neurosurgery considers DBS an emerging but not yet established treatment, because the current evidence does not meet its standards.

The outcome figures are correspondingly wide, and the width is the teaching point rather than any midpoint a candidate might quote. Response rates for DBS in obsessive-compulsive disorder across studies range from 10 to 61 percent, largely because of differences in electrode placement, electrode type and stimulation. A response rate quoted without its target and its settings therefore tells the reader nothing.

PITFALL

Reading the United States authorisation as evidence of the kind a premarket approval carries, or as an Indian permission. The obsessive-compulsive authorisation is a **Humanitarian Device Exemption, number H050003, decided in 2009**, and a humanitarian exemption is not a premarket approval: it is a small-population route demanding a lower evidentiary showing. Its scope is narrow: bilateral stimulation of the anterior limb of the internal capsule, as an adjunct to medications and as an alternative to anterior capsulotomy, for chronic, severe, treatment-resistant obsessive compulsive disorder in adult patients who have failed at least three selective serotonin reuptake inhibitors. Collapsing that into approved for OCD overstates both the evidentiary standard and the jurisdiction.

20.4 Refractory depression: two pivotal sham-controlled trials, and two different endpoints

Both pivotal sham-controlled trials of deep brain stimulation for depression failed to separate active stimulation from sham on their primary endpoints, and both were stopped for futility. That must be stated plainly: the open-label literature around each is encouraging, and a chapter reporting only the encouraging half is reporting a different field.

The subcallosal cingulate trial, BROADEN, followed encouraging open-label work with reported response rates of 36 to 66 percent. Its endpoint was defined in its own report as frequency of response, defined as a 40 percent or greater reduction in depression severity from baseline, averaged over the fourth to sixth months of the double-blind phase, with a futility analysis performed when approximately half the proposed sample had received implantation and completed the double-blind phase. The result: both groups showed improvement, but there was no statistically significant difference in response during the double-blind, sham-controlled phase, with 12 patients (20 percent) responding in the stimulation group against five patients (17 percent) in the control group. The trial was halted because of a low probability of success on that interim futility analysis.

The ventral capsule and ventral striatum trial, RECLAIM, ran a different design and used a different threshold. Patients were randomised to active versus sham stimulation in a blinded fashion for 16 weeks, followed by an open-label continuation phase, and the primary outcome measure was response, defined as a 50 percent or greater improvement on the Montgomery and Asberg Depression Rating Scale from baseline. The result was again negative: no significant difference in response rates between the active arm (3 of 15 subjects, 20 percent) and the control arm (2 of 14 subjects, 14.3 percent), and no significant difference in the rating-scale change as a continuous measure on completion of the 16-week controlled phase, and the trial was terminated for futility.

RULE **The two trials did not share a primary endpoint, and printing one threshold for both invents a fact.** BROADEN's response threshold was a 40 percent or greater reduction in depression severity, averaged over the fourth to sixth months. RECLAIM's was a 50 percent or greater improvement on the Montgomery and Asberg Depression Rating Scale, assessed at the end of a fixed blinded window. Different thresholds, different scales, different timepoints, different targets. Quote each trial with its own definition, or quote neither.

AXIS	BROADEN	RECLAIM
TARGET	Subcallosal cingulate	Ventral capsule and ventral striatum
RESPONSE DEFINED AS	40 percent or greater reduction in depression severity from baseline	50 percent or greater improvement on the Montgomery and Asberg scale
BLINDED WINDOW	Averaged over the fourth to sixth months of the double-blind phase	16 weeks
RESULT ON THAT ENDPOINT	No significant difference; 12 (20 percent) against five (17 percent)	No significant difference; 3 of 15 (20 percent) against 2 of 14 (14.3 percent)
STOPPED	Halted on interim futility analysis	Terminated for futility

The criticism raised against both is of design rather than of the device, and a candidate should be able to state it without using it to explain the results away. Short blinded phases and limited parameter adjustment likely contributed to the negative findings. Later work is offered against the negative result and should be given as what it is, later and less controlled. Open-label follow-up suggested response rates rising over 1 to 3 years, and tractography work reported response rates above 80 percent when stimulation targeted the convergence zone of forceps minor, cingulum bundle and uncinate fasciculus. And on the same ventral target, another double-blind trial that allowed a one-year optimisation phase before a crossover comparison reported significant differences favouring active stimulation, with response rates of 50 to 60 percent. Two trials on one target reaching opposite conclusions is a live disagreement, not a resolved question, and the difference is largely how long the operator was allowed to programme before the comparison was made.

20.5 Stimulation parameters and programming

Programming does not begin in the operating theatre. Across the psychiatric literature stimulation was mostly bilateral, starting 6 to 8 weeks after surgery, the interval letting the acute surgical effect settle before any setting is judged. The reported settings are **4 to 6 V** of amplitude, 60 to 120 microseconds of pulse width and 130 to 180 Hz of frequency.

What those figures are must travel with them: ranges derived from a systematic review of heterogeneous studies across several targets and electrode types, not a validated protocol and not a manufacturer specification. The same review names variance in target and electrode type as a principal source of the wide response range. Read the trial criticism above against this section and it stops being a statistical complaint: a fixed short blinded window with restricted adjustment means the comparison was made before the operator had finished finding the setting, which is a different objection from an underpowered sample.

20.6 Adverse effects, and the implant effect

The adverse effects divide by source, the division a consent conversation follows. Device-related complications reported across the psychiatric literature included lead damage or malposition, and postoperative infections, some of which necessitated **explantation** or generator replacement. That last clause is the one to hold: a psychiatric implant can end with the hardware coming out. On the surgical side, seizures occurred at a rate consistent with other intracranial procedures, and isolated incidents of intracranial haemorrhage were described. The stimulation-related effects specific to each target and each setting are not carried in this record and are declared as a gap rather than filled from memory.

One phenomenon here explains something about the trials. The reviewers name an **implant effect**, a temporary improvement following electrode placement, possibly linked to microlesioning, and offer it alongside cognitive placebo effects and natural fluctuation as reasons the active and sham arms did not separate. A sham arm in this field is not an untreated arm: it is a patient who has had a craniotomy and an electrode placed, and whose device is off. The mood, impulse-control and cognitive sequelae of stimulation given in a movement-disorder indication

belong to the Neuropsychiatry of systemic and neurological disease notes; they are named here and not taught.

20.7 Patient selection

Selection is where the honest evidence position becomes a clinical rule, and the candidate definitions are strict in both indications.

- Obsessive-compulsive disorder: severe or extreme illness of more than five years' duration, having exhausted first-line and second-line treatments including cognitive behavioural therapy, several selective serotonin reuptake inhibitors, clomipramine and antipsychotic augmentation at adequate doses and durations.
- Depression: severe depression on standardised scales, illness duration beyond two years, marked functional impact, and documented resistance to multiple pharmacological regimens and to electroconvulsive therapy, or a patient for whom electroconvulsive therapy is not appropriate, not acceptable or declined.

Two things follow. Resistance must be documented rather than asserted, so the trials of treatment, their doses and durations are in the record before the question is raised. And electroconvulsive therapy sits upstream of stimulation in the depression pathway, so a referral made without it, or without a stated reason it cannot be used, skips a rung.

20.8 India, the law, and a question the Act does not answer

IN INDIA

The Indian device position is a classification and only a classification. The national risk-based list carries the deep brain electrical stimulation system, designed to apply electrical stimuli to specific areas of the deep brain for the treatment of movement disorders, psychiatric disorders or chronic, severe, intractable pain, in the highest risk class, Class D. Two things follow and neither may be softened. The classification names psychiatric disorders in its own intended-use text, so the category exists in Indian regulatory language. But it establishes nothing about product licensing of any named system in India, and nothing about any indication-specific psychiatric authorisation; neither may be inferred from the classification, and neither from the foreign humanitarian exemption above.

The legal question is unsettled, and this account states it as unsettled rather than answering it. The Mental Healthcare Act restricts psychosurgery, and that restriction, with its dual requirement of informed consent and Board approval, is the Mental health legislation and forensic psychiatry notes' provision; it is named in words here and never renumbered. The question is whether an implanted stimulation system for refractory obsessive-compulsive disorder falls within psychosurgery for the purposes of that restriction, because if it does, the dual requirement attaches. Read against the statute's own text the answer does not arrive: the Act restricts psychosurgery but nowhere defines it, its definitions clause contains no entry for psychosurgery, and the word appears in the Act only in the restricting provision itself. The Act's own definition therefore does not settle the question, and it is recorded here as a declared gap. A

regulation defining the term could settle it; none was located. The practical consequence is caution rather than paralysis: a unit contemplating an implant proceeds as though the stricter reading may apply, and takes the question to its Board and to legal advice. The consent conversation itself, as taken in Indian practice, is this chapter's account of consent for electroconvulsive therapy.

TRAP **Answering the psychosurgery question from memory, in either direction.** Writing that psychiatric deep brain stimulation is psychosurgery under the Act, or that it plainly is not, both assert something the statute does not say. The defensible answer names the restriction in words, says the Act does not define the restricted procedure, and leaves the question open. A second trap sits beside it: quoting the Act's section numbers here. Those belong to the legislation notes, and a chapter that reprints a number it did not itself ground is how a legal fiction enters a syllabus.

Deep brain stimulation is an implanted, adjustable, reversible circuit intervention, not a lesion, and its psychiatric targets are the ventral capsule and ventral striatum, the anterior limb of the internal capsule, the nucleus accumbens and the subcallosal cingulate; most guidelines place it after everything else has failed; both pivotal sham-controlled depression trials, on different targets and with different response thresholds, failed to separate active from sham and were stopped for futility, so the case now rests on later, less controlled work; and whether the Act's psychosurgery restriction reaches an implanted system is a question the statute leaves open.

21 · Vagus nerve stimulation (VNS)

Vagus nerve stimulation treats depression from outside the skull, by driving a peripheral nerve in the neck and letting that nerve carry the signal inward. It is the implanted device of this band whose electrode never enters the brain. Its centre of gravity is not the surgery but the latency: a response, where there is one, emerges over months, and the trial designed to detect it in weeks did not.

This account owns vagus nerve stimulation in full: the implanted left cervical system, the afferent route, the parameters and duty cycle, the regulatory position in both jurisdictions, the evidence with its limits, and the adverse effects. It does not teach the implanted intracranial device, whose surgical placement, psychiatric targets and trial evidence belong to this chapter's account of deep brain stimulation in psychiatry, named here and not restated. Nor does it grade the band's devices against one another: that comparison, across invasiveness, seizure induction, target, reversibility, evidence and Indian availability, belongs to the cross-cutting synthesis and its band grid.

p=0.238	12 months	58.2%
THE ACUTE SHAM-CONTROLLED PRIMARY ENDPOINT, NOT SIGNIFICANT	THE WINDOW OVER WHICH THE EFFECT SUPPORTING APPROVAL WAS MEASURED	VOICE ALTERATION IN THE EARLY MONTHS OF STIMULATION

21.1 The implanted system, and why it is not a cranial implant

The **VNS Therapy System** divides into what is inside the patient and what is not.

- An implanted **pulse generator**, carrying the battery and the programming, sits in a subcutaneous chest pocket.
- A **helical lead** runs from it into the neck and is wound on the cervical vagus nerve, passed between the sites with a tunneler.
- Outside the body are an external **programming wand** and its software, through which every parameter is set without a second operation, and a patient magnet.

The electrode therefore sits on a peripheral nerve, and implantation is a neck and chest procedure carrying the risks of a neck dissection and of a stimulated nerve, not those of an intracranial electrode. Implanted does not mean here what it means for the intracranial device taught elsewhere in this chapter.

21.2 The afferent route, and how far the record goes

Stimulation is delivered to the **left cervical vagus**, and the therapeutic traffic runs centrally rather than out to the viscera. The vagus there is predominantly afferent, so the fibres the electrode recruits carry impulses into the brainstem. The route taught runs from the **nucleus tractus solitarius**, the brainstem terminus of vagal afferents, to the **locus coeruleus** and the raphe nuclei, and on from those monoaminergic nuclei to limbic structures: a peripheral electrode producing a diffuse, slowly accumulating central effect rather than a focal one.

Where the grounding stops must be said. The generator and lead architecture is read from the regulator's own summary of safety and effectiveness data, while the left-sided placement and the brainstem-to-limbic pathway are standard teaching, stated as teaching, not captured verbatim from a primary source by this chapter's grounding, and recorded as a declared gap.

21.3 The stimulation parameters and the duty cycle

Every figure below belongs to one protocol, one indication, one target and one device, and none is a universal setting for vagus nerve stimulation. These are the settings actually reached at 12 months in the pivotal depression cohort, D-02, as printed in the labelling approved for the depression indication on the VNS Therapy System delivered to the left cervical vagus. They are not manufacturer defaults, not a starting prescription, and not transferable to the seizure indication.

PARAMETER	MEDIAN AT 12 MONTHS	RANGE IN THE COHORT
OUTPUT CURRENT (NO UNIT IN THE RECORD)	1.0	0 to 2.25
FREQUENCY	20 Hz	2 to 30 Hz
PULSE WIDTH	500 microseconds	130 to 750 microseconds
ON TIME	30 seconds	7 to 60 seconds
OFF TIME	5 minutes	0.3 to 180 minutes

One row carries no unit in the record it was read from: the labelling table as extracted gives the output current as a bare figure and does not say what it is measured in. No unit is printed here

and none may be supplied from memory, because a plausible unit beside a real number makes the number look grounded when the unit is not.

The **duty cycle** is what the ON and OFF times mean together: the device does not stimulate continuously but delivers current for the programmed ON time, stops for the programmed OFF time, and repeats indefinitely. The duty cycle is therefore not a fixed property of the device but an arithmetic consequence of those two times as programmed, changing the moment either is changed. No duty-cycle percentage is printed here, because computing one is arithmetic rather than a reading from the labelling.

MNEMONIC

SLOW

Subcutaneous generator in the chest. **L**eft cervical vagus carries the lead. **O**n and off, because stimulation is cyclical and never continuous. **W**ait, because the response is measured in months.

21.4 The regulatory position, in its separate parts

The regulatory claims a candidate must keep apart are device **classification**, product **licensing**, **indication-specific authorisation**, and **foreign clearance or approval**. Holding one says nothing about holding another. In the United States the Food and Drug Administration, through its Center for Devices and Radiological Health, approved the VNS Therapy System for depression under premarket approval P970003, supplement S050, decision date 15 July 2005. The authorised indication is precise: adjunctive long-term treatment of chronic or recurrent depression, in patients 18 years of age or older, experiencing a major depressive episode, who have not had an adequate response to four or more adequate antidepressant treatments. Read the wording: adjunctive means added to treatment, and long-term sits in the indication because the evidence is long-term.

IN INDIA

What is established is classification and nothing more. The national risk-based list names the antiseizure and psychiatric-therapy vagus nerve electrical stimulation system in the Neurological category at serial 9, Class D, with its programming system separately at serial 131 in Class C. There the Indian record stops. No Indian product licence for any named device, and no Indian indication-specific authorisation in treatment-resistant depression, was established by this chapter's grounding; both are declared gaps and neither may be inferred. The United States approval above is foreign status: it is not evidence of an Indian one and must never be written so a reader could take it for one. A provenance caution attaches to the list itself, hosted as final while its covering notice circulates the annexed classification for comment.

21.5 The acute randomised trial, which did not separate from sham

The pivotal acute study, **D-02**, is why this modality can be taught honestly at all. Patients with treatment-resistant depression were implanted, then randomised to active stimulation or to sham, the device present in both arms and left off in the sham arm, and the acute phase ran 3

months. The primary endpoint was the proportion of responders, a fall of 50 percent or more in the Hamilton score from baseline to exit.

In the evaluable population 15.3 percent of the active group responded, 17 of 111, against 10.0 percent on sham, 11 of 110. The difference was not statistically significant, p=0.238. Among the secondary measures only the self-rated inventory of depressive symptomatology separated on percent responders, 17.4 against 7.5 percent, p=0.032; the clinical global impression, the observer-rated depression and mania scales and the SF-36 did not.

21.6 The long latency, and what the approval rests on

The device was not approved on that comparison. After the acute analysis was completed, an alternate statistical plan for demonstrating effectiveness was adopted, comparing the 12 month results of the D-02 continuation phase against a separate observational study, D-04. That is the approval basis: a non-randomised comparison of an open-label extension against a different cohort.

The long-term primary analysis was a repeated-measures linear regression on raw Hamilton scores over the first 12 months of stimulation. The slope was minus 0.47 points per month in the completer population, n=177, p less than 0.001, with minus 0.45 in the evaluable population, n=205, and minus 0.40 in the intention-to-treat population, n=231. At 12 months Hamilton response was 29.8 percent and remission 17.1 percent, and sustained response was reached by 27 percent of completers, 47 of 177. Against the observational comparator the average Hamilton change at 12 months was minus 8.2 with the device against minus 4.9 with standard care, p=0.006, with response in 30 against 13 percent, p=0.003.

RULE **The long latency is not a caveat on the evidence; it is the shape of the evidence, and it decides how the device is judged and when.** An effect appearing as a slope over months cannot be detected by a trial reading its endpoint at 3 months, which is why the randomised comparison came out flat and why the authorised indication says long-term. A patient is therefore not a non-responder here at the point at which the same patient would be judged a non-responder to a drug. What is traded is TIME and not cognition; it is this device's own trade, and no direction carries over to any other device in the band.

TRAP **Quoting the long-term response and remission rates as though they came from the randomised sham comparison.** They did not. That comparison failed its primary endpoint; the long-term figures come from an open-label continuation set against a separate observational cohort. An answer giving the second without the first is not partial, it is wrong.

RECOVER is the modern test of the same question, described as the largest neuromodulation study in psychiatry to date: participants randomised to active against sham stimulation for a year, then a prolonged open-label phase. Preliminary reports indicate the primary endpoint was not met, while clinically meaningful improvements were observed in depressive severity, functioning and quality of life. This chapter's grounding read that in a peer-reviewed review and not in the primary report, and the review's own reading, that the device may act as a long-term

progressively acting adjunct whose effect symptom scales do not fully capture, is attributed rather than adopted.

21.7 Adverse effects, and the pattern that matters

The adverse effects divide by cause into what the operation does and what the stimulation does, and the second group carries a pattern worth memorising: the effects involving the voice persist across the first year while most of the rest settle. The frequencies are from the approved labelling for the depression indication.

STIMULATION-RELATED EVENT	0 TO 3 MONTHS (N=232)	9 TO 12 MONTHS (N=209)
VOICE ALTERATION	135 (58.2 percent)	113 (54.1 percent)
INCREASED COUGH	55 (23.7 percent)	13 (6.2 percent)
NECK PAIN	38 (16.4 percent)	27 (12.9 percent)
DYSPNOEA	33 (14.2 percent)	34 (16.3 percent)
DYSPHAGIA	31 (13.4 percent)	9 (4.3 percent)
PARAESTHESIA	26 (11.2 percent)	9 (4.3 percent)
LARYNGISMUS	23 (9.9 percent)	10 (4.8 percent)

The persistent entries are mostly of the voice, the airway and the neck, which is what an electrode wound on a nerve in the neck predicts, and the table above shows why the generalisation cannot be tightened further: dyspnoea persists and rises and is respiratory rather than laryngeal, neck pain persists and is somatic, and laryngismus is laryngeal yet largely settles. and voice alteration is the effect a patient notices and is asked about at every review. Cough, dysphagia and paraesthesia largely settle; dyspnoea does not. Surgery-related events in the acute phase were incision pain in 36 percent, device-site pain in 23 percent, device-site reaction in 14 percent and headache in 8 percent.

The serious events, as the labelling reports them: 30 serious adverse events in 27 subjects in the acute phase, including one death by suicide under active stimulation, with single reports of asystole and of bradycardia in the treatment group; and in the long-term phase worsening depression as 62 events in 31 subjects, 7 suicide attempts in 6 subjects, and one sudden unexplained death. None of that withholds the device where it is indicated; all of it must be described to the patient before implantation.

Vagus nerve stimulation is an implanted but non-cranial system: a generator in the chest, a lead on the left cervical vagus, an afferent signal reaching limbic structures through the brainstem. It stimulates intermittently, so the duty cycle follows from the programmed ON and OFF times. It holds a United States approval for adjunctive long-term treatment after four or more failed adequate antidepressant treatments and, in India, a device classification and nothing further. Its acute randomised comparison against sham did not meet its primary endpoint, the approval resting on a slope across the first year against an observational cohort. Its characteristic adverse effect, voice alteration, is common and does not settle.

22 · Magnetic seizure therapy and emerging neuromodulation

Magnetic seizure therapy uses the induction coil of transcranial magnetic stimulation at parameters chosen to produce a seizure deliberately, under anaesthesia, as an alternative to electroconvulsive therapy. It is the only modality in this group with randomised human comparisons against electroconvulsive therapy, and it is examinable for a reason that is not the hardware: it is this chapter's cleanest example of a claim that must be split in two, one half settled and one open, and a candidate who fuses them writes a sentence the sources do not carry.

This account owns magnetic seizure therapy and the other emerging approaches: low-intensity focused ultrasound, transcranial alternating and random-noise stimulation, and closed-loop or adaptive stimulation. Why an induced seizure helps is taught in this chapter's account of the mechanism of action and neurobiological theories of electroconvulsive therapy, and the cognitive cost of a course, with anterograde and retrograde amnesia held apart, in its account of the cognitive side effects of electroconvulsive therapy; both are named for comparison and neither is re-derived. The ablative form of focused ultrasound belongs with this chapter's account of psychosurgery, and the grid grading the whole band belongs to the cross-cutting synthesis. None of what follows is established psychiatric practice.

100 percent	5.3 percent	animal
OF MAXIMAL STIMULATOR OUTPUT, THE USUAL INTENSITY	REMISSION DIFFERENCE FAVOURING ECT IN THE CONFIRMATORY TRIAL, NON-INFERIORITY MET	THE LEVEL AT WHICH A DIFFERING SEIZURE PHYSIOLOGY IS DEMONSTRATED

22.1 The technique, and how it is dosed

Magnetic seizure therapy is defined by the international safety guideline as the use of transcranial magnetic stimulation to induce seizures deliberately, under anaesthesia, for the treatment of depression or other serious neuropsychiatric conditions. What it demands is electroconvulsive therapy infrastructure, not a transcranial magnetic stimulation clinic: general anaesthesia in a procedure room with a crash cart and an anaesthesia station, a multidisciplinary team, and the standard pre-electroconvulsive-therapy workup. The dose sits at the machine's ceiling rather than a fraction of it. Magnetic seizure therapy is usually given at **100 percent** of maximal stimulator output, at a frequency of 25 to 100 Hz, in a single train lasting up to 10 seconds.

The coil belongs in the prescription. Coils differ in their efficiency at seizure induction, and the larger, less focal designs, the twin coil and the double cone, are more efficient than the focal figure-of-eight; much of the published work has used round or double-cone coils over the vertex, a placement reported to have a lower seizure threshold than midline prefrontal placement. The property that gives the technique its rationale therefore works against the event it must produce, and the operator settles that by coil choice and placement rather than by dose. Threshold is titrated by successively increasing stimuli within a single anaesthesia session, as a seizure threshold is titrated for electroconvulsive therapy; the interval between stimuli is not carried here. Monitoring is two-channel frontomastoid electroencephalography with the motor expression of the seizure, isolated by a leg tourniquet placed before the relaxant. The section ends where the guideline ends: the optimal dose above the seizure threshold for a given coil is not known, and neither is the dosing that would maximise benefit while minimising side effects.

22.2 The rationale, and the two claims that are held apart

The stated rationale is control over where the stimulation acts: increased control over the site of stimulation permits sparing of brain regions related to the adverse effects of electroconvulsive therapy. Two claims sit behind that sentence, they are not equally strong, and separating them is the examinable content here.

What is unambiguous. Studies using realistic head modelling show that, even at maximal stimulator output intensity, the electric field induced in the brain by magnetic seizure therapy is much lower and much more focal than the field induced by electroconvulsive therapy. That follows from the coil geometry, and the site at which the seizure is initiated is correspondingly more restricted. Both statements are about the stimulus, and the modelling is established. Note what is NOT said: no row in this chapter's claim list, and no source quote in it, describes the field or the site as more SUPERFICIAL, so depth is an ungrounded attribute here and is not carried.

What is an evidence question. Whether the seizure that then follows propagates less widely than an electroconvulsive therapy seizure, and how much of the measured cognitive difference such reduced propagation would explain. Here the record is thinner and its level is part of the claim. Animal studies substantiate that seizures induced with magnetic seizure therapy are less robust and differ in their physiological expression from electroconvulsive therapy seizures; in a sensitive non-human primate model, magnetic seizure therapy was significantly safer than electroconvulsive therapy and did not differ from an anaesthesia-alone sham, neither modality showing neuropathological evidence of tissue damage. That is animal evidence, reported as animal evidence. A human demonstration of reduced propagation was not located for this chapter, and the share of the human cognitive difference attributable to propagation is unquantified. The guideline's own bridge from focality to tolerability is a belief, that the field difference is thought to underlie the superior side-effect profile, and prose that hardens it into a finding has changed the evidence while quoting it.

RULE **A focal field is not a confined ictus, and the two claims travel at different evidence strengths.** Say it in one form and keep the two objects straight: the induced electric field is much lower and much more focal than in electroconvulsive therapy, and the site of seizure initiation is correspondingly more restricted. That is settled modelling. Say separately, at the strength the sources carry, that such seizures are less robust and physiologically different in animals, that the human propagation question is open, and that propagation's contribution to the cognitive difference is unquantified. The error in one direction conflates the focal stimulus with the spread of what follows it, which the sources do not show; the error in the other is to deny any difference in propagation, which the animal data contradict.

22.3 The human evidence: two randomised comparisons

Two randomised comparisons against right unilateral ultrabrief electroconvulsive therapy exist, and a candidate should name both: citing the larger alone overstates how settled the position is. Both treated to remission, dropout, or a maximum of 21 treatments, and both paired remission on the 24-item Hamilton scale with worsening of autobiographical memory as co-primary outcomes, worsening defined in the confirmatory trial as a 25 percent reduction in Autobiographical Memory Test score. The confirmatory trial, CREST-MST, used a non-inferiority margin of 15 percent absolute difference in remission and delivered magnetic seizure therapy with a twin coil in a midline frontal position.

AXIS	CONFIRMATORY TRIAL IN MAJOR DEPRESSIVE DISORDER	PILOT TRIAL IN BIPOLAR DEPRESSION
DESIGN	Multisite randomised double-blind parallel-group non-inferiority trial, three academic centres in Canada and the United States	Double-blind randomised parallel-group pilot
NUMBERS	292 enrolled, 239 randomised	55 randomised, 45 given an adequate trial
REMISSION, MAGNETIC SEIZURE THERAPY AGAINST ELECTROCONVULSIVE THERAPY	22.5 percent against 27.8 percent, a difference of 5.3 percent favouring electroconvulsive therapy, non-inferiority established at p = 0.048	5 of 25 (20 percent) against 6 of 20 (30 percent)
MEMORY WORSENING, SAME ORDER	2.7 percent against 17.3 percent, p = 0.0003	2 of 28 (7.1 percent) against 6 of 27 (22.2 percent)
AUTHORS' OWN LIMIT	Enrolment concluded before the intended sample size was reached	Findings to be considered preliminary, given the small pilot sample

The tolerability figures point the same way as the memory outcome: withdrawal for non-serious adverse events ran at 12 participants on electroconvulsive therapy against 3 on magnetic seizure therapy. Its authors read that as supporting magnetic seizure therapy as a first-line convulsive therapy in major depressive disorder, particularly for a patient who refuses right unilateral ultrabrief electroconvulsive therapy. That is an author interpretation of a trial whose enrolment stopped short, in a technique whose optimal dose is acknowledged to be unknown.

TRAP **Reading the memory advantage in these trials as proof that the seizure stayed local.** The trials measured autobiographical memory, not seizure spread. They establish an outcome difference on a named instrument; what produced it, whether the weaker induced field, a difference in how the seizure expresses itself, or something else in the procedure, an outcome trial does not settle. Report the outcome as an outcome and the mechanism as a hypothesis.

22.4 Focused ultrasound, in its neuromodulatory form

Transcranial focused ultrasound delivers acoustic energy through the intact skull to a focus placed at depth, and one name here covers two different interventions. Low-intensity focused ultrasound for neuromodulation is reversible and generally operates at intensities below 100 W per square centimetre. High-intensity focused ultrasound typically employs acoustic intensities greater than 200 W per square centimetre and is an ablation tool; the psychiatric procedure built on it is taught in this chapter's account of psychosurgery. Collapsing the two converts a reversible research technique into a lesion.

One physical trade-off is worth carrying: lower frequencies cross the skull better while higher frequencies give better focal precision, so penetration and precision are bought against each other. The frequency and acoustic pressure bands, and the pulse duration, pulse repetition frequency, duty cycle and train duration, are not carried here. The evidence level is best given in the reviewers' own terms: mechanisms incompletely understood, long-term effects, optimal parameters and safety assessment requiring systematic elucidation, and auditory confounding an unresolved controversy.

22.5 Alternating and random-noise stimulation

The low-intensity transcranial electrical stimulation family is one envelope with several waveforms, defined by the same guideline that defines transcranial direct current stimulation. The current can be of constant intensity and polarity, which is transcranial direct current stimulation and has its own account in this chapter. It can instead be of variable intensity or polarity:

- alternating between polarities at a steady sinusoidal frequency, which is **transcranial alternating current stimulation**;
- alternating at a randomly changing frequency, which is **transcranial random-noise stimulation**;
- varying within a single polarity, as in oscillatory transcranial direct current stimulation.

The examinable point is the shared envelope: these are variants of one low-intensity technique bounded by a maximum current, a maximum session duration, an electrode-area band and a frequency band, rather than separate technologies with limits of their own; the bounds are not carried here. One reading convention travels with them: direct current intensity is reported baseline-to-peak, and for alternating and random-noise stimulation the convention used has to be specified, because the same waveform yields different figures under different conventions. What this record establishes is definitions and a safety envelope, not clinical efficacy for any psychiatric indication, and a tidy definition is not evidence of one.

22.6 Closed-loop and adaptive stimulation

Closed-loop stimulation, also called adaptive stimulation, adjusts its output from a signal recorded in the patient instead of delivering parameters fixed at programming. One regulatory authorisation for the principle exists, and its scope has to be quoted exactly. In 2025 the United States Food and Drug Administration approved, through a premarket approval supplement, an optional programming feature called adaptive deep brain stimulation, for bilateral stimulation of the internal globus pallidus or the subthalamic nucleus, as adjunctive therapy in levodopa-responsive Parkinson disease of at least 4 years duration that is not adequately controlled with medication.

Read the scope rather than the headline. That is a foreign regulatory status, for a neurological indication, at motor targets, on a platform whose psychiatric use is taught in this chapter's own account of deep brain stimulation. It authorises nothing in psychiatry and establishes nothing about the Indian regulatory position for any device. What it does establish is that closed-loop control has crossed from research into an approved product feature in at least one jurisdiction, which is why it appears in a psychiatry chapter at all.

MNEMONIC

MUAC

The four emerging approaches, ordered by how much human evidence each carries. **M**agnetic seizure therapy, with two randomised comparisons against right unilateral ultrabrief electroconvulsive therapy. **U**ltrasound, low intensity and focused, with mechanisms still being worked out. **A**lternating and random-noise currents, defined inside the low-intensity electrical stimulation envelope and no further. **C**losed-loop and adaptive stimulation, approved abroad for a motor indication. None of the four is established psychiatric practice.

Magnetic seizure therapy induces a therapeutic seizure with an induced electric field much lower and much more focal than that of electroconvulsive therapy and with a correspondingly more restricted site of initiation, which is settled modelling; whether the resulting seizure spreads less is supported in animals, open in humans, and unquantified as an explanation of the cognitive difference two randomised trials have measured; and low-intensity focused ultrasound, alternating and random-noise stimulation and closed-loop stimulation are each carried at their own evidence level, none of them established psychiatric practice.

23 · Psychosurgery (history, current indications, ethics)

Ablative psychosurgery treats psychiatric illness by destroying a small, deliberately chosen volume of brain tissue, and the tissue does not come back. That one fact governs the rest: the narrow indications, the weight of the consent, and the statutory machinery around it. The topic is examinable from both ends, the documented ethical failure in its history and the technique of a modern stereotactic theatre.

This account owns ablative neurosurgery for psychiatric illness in full: the descent from frontal leucotomy through its abuses to modern stereotactic and image-guided lesioning, the procedures with their targets and how each lesion is placed, the narrow indications, the evidence and the designs it rests on, the irreversibility, and the ethics. It does not draw the band comparison on invasiveness, seizure induction, target, reversibility, evidence and Indian availability; that grid belongs to the cross-cutting synthesis. Deep brain stimulation belongs to this chapter's own account of it, and the history of electroconvulsive therapy to the item owning it. The statutory restriction on psychosurgery belongs to the Mental health legislation and forensic psychiatry notes, named here in words and never renumbered.

four	60,000	50 to 60 percent
ABLATIVE PROCEDURES IN CURRENT USE, EACH WITH ITS OWN TARGET	PROCEDURES AT THE HEIGHT OF THE LEUCOTOMY ERA	RESPONSE IN REFRACTORY ILLNESS, ON LARGELY UNCONTROLLED DESIGNS

23.1 Two groups, not one family

Ablative psychosurgery is not the invasive end of a stimulation scale. Psychosurgical intervention divides into two groups: **lesioning** destroys tissue, stimulation delivers current to tissue that remains intact. Both groups share one principle, to intervene as little as necessary to restore a dysfunctional circuit, and the choice turns on indication, centre experience and patient preference, weighing the irreversibility and relative simplicity of lesioning against the reversibility, adjustability and cost of stimulation.

One qualification travels with the contrast so it is not overdrawn: deep brain stimulation was initially conceptualised as a reversible lesion, and the effects of lesioning are increasingly attributed to circuit reorganisation rather than to tissue destruction alone, so the families may converge in mechanism where they do not converge in reversibility.

RULE **Ablation and stimulation are two families, and the boundary between them is irreversibility, not degree of invasiveness.** Every stimulation modality of this band shares one property no ablative procedure has: switching the device off ends the intervention. Ablation destroys tissue, so it outlives every later decision, including the patient's own change of mind. It therefore appears in the band's grid for contrast, never as the most invasive rung of a stimulation ladder.

23.2 Frontal leucotomy: what was actually done

The named precursors are Gottlieb Burckhardt's cortical resections in psychotic patients and Fulton and Penfield's work on frontal-lobe physiology. In 1936, Egas Moniz, working with the neurosurgeon Almeida Lima, published his first series of 20 patients who underwent prefrontal leucotomy for severe depression, anxiety and aggression. The technique is the detail candidates omit: the lesions were made by free-hand injection of absolute alcohol into the frontal lobes, with **substantial symptom improvement reported in 14 of 20 patients**. Moniz later replaced the alcohol with a hand-held leucotome, making more restricted lesions.

Anatomically, the operation lesioned prefrontal white matter through small frontal burr holes, partially disconnecting the frontal cortex from its thalamic and limbic connections. The

outcomes were not uniform, and that is the point: anxiety and agitation decreased in some patients, while in others apathy, abulia, personality change or cognitive deterioration emerged. A free-hand disconnection produced benefit and injury from the same act.

23.3 The abuses, and the reform they forced

Abuse here means what was done, not disapproval of an era. Walter Freeman and James Watts popularised the procedure in the United States, redefining its access points and lesion strategies, and by 1942, the year their monograph was published, they had performed 200 lobotomies. Freeman then promoted more expedient versions, including the transorbital lobotomy, and advocated wide adoption with lax indications, with interventions performed outside the operating room and sometimes without a neurosurgeon. Volume reached up to 60,000 procedures, against the overcrowding of psychiatric institutions.

Three features make this an abuse rather than a therapeutic error: indications so loose the operation was offered where no case existed, delivery outside the operating theatre, and delivery sometimes without a surgeon. This high-volume, low-supervision phase is what fuelled the ethical and regulatory backlash that continues to stigmatise these procedures. Reform came from within the discipline: cortical undercutting and topectomies were introduced as more selective approaches, and Spiegel and Wycis then introduced the stereotactic frame in humans, lesioning the mediodorsal thalamic nucleus using X-ray ventriculography. Everything modern descends from that instrument: a lesion positioned by calculation and bounded by design.

TRAP **Telling the Freeman story as though it were the whole of psychosurgery, and stopping there.** The leucotomy era is why the field is regulated, and a candidate who cannot narrate it has not answered. But an answer ending in the mid twentieth century treats a living, narrowly indicated, image-guided operation as a curiosity, and says nothing about targets, technique, evidence or present-day consent.

23.4 The current procedures, their targets and their technique

Each has a named target, and the technique of the first two is what the anxiety and obsessive-compulsive disorder material promises the reader here.

PROCEDURE	TARGET	HOW THE LESION IS PLACED	ORIGIN AND MODERN FORM
ANTERIOR CAPSULOTOMY	The anterior limb of the internal capsule	Thermocoagulation, later radioactive yttrium-90 seeds; Leksell independently used parallel bipolar radiofrequency electrodes	Jean Talairach, 1949, for anxiety neuroses and obsessive compulsive disorders
ANTERIOR CINGULOTOMY	The anterior cingulate, lesioned bilaterally	Stereotactic frame, magnetic resonance guidance and intravenous sedation, through a single burr hole per side	Triple-lesion form standardised since 1996
SUBCAUDATE TRACTOTOMY	The posterior orbitofrontal cortex, bilaterally, using the pituitary sella as a landmark	Rod-shaped yttrium seeds creating radiation-necrotic lesions, later replaced by radiofrequency coagulation	Geoffrey Knight, 1965, adapting Scoville's orbitofrontal undercutting
LIMBIC LEUCOTOMY	The cingulate and orbitofrontal targets together	Cingulotomy combined with subcaudate tractotomy	Kelly and colleagues, 1973; now usually reached as a staged procedure

Anterior cingulotomy is best given as an operative sequence, because the sequence is what changed. The historical procedure made a single lesion of about 2 cm by 1 cm per side, under magnetic resonance guidance and intravenous sedation, using a stereotactic frame. Patients who had not responded at 9 to 12 months underwent anterior extension of the lesion, and since 1996 all three lesions per side are made simultaneously, as the triple-lesion or six-pack cingulotomy, through a single burr hole with a 10 mm exposed-tip electrode redirected along the contour of the cingulate gyrus. The staging follows from that: in the mid-1990s at Massachusetts General Hospital, patients who failed the initial six-pack cingulotomy were offered a subcaudate tractotomy, and cingulotomy followed by subcaudate tractotomy is a staged limbic leucotomy.

Anterior capsulotomy aims at white matter rather than cortex, and its history is one of energy sources rather than targets: thermocoagulation first, then radioactive yttrium-90 seeds, with Leksell independently using parallel bipolar radiofrequency electrodes. **Subcaudate tractotomy** and **limbic leucotomy** complete the set, the last being a deliberate combination on the reasoning that if each independently helps, combining them might do better.

MNEMONIC

CATS

Capsulotomy, anterior: anterior limb of the internal capsule. **A**nterior cingulotomy: anterior cingulate. **T**ractotomy, subcaudate: posterior orbitofrontal cortex. **S**taged limbic leucotomy: cingulotomy plus subcaudate tractotomy in one patient.

23.5 How the lesion is actually made

A target is not a technique. Three energy modalities are in use, each with its own working figures.

MODALITY	HOW THE ENERGY REACHES THE TARGET	WHAT IT DOES TO TISSUE
RADIOFREQUENCY THERMAL ABLATION	A stereotactically inserted electrode whose tip delivers a high-frequency alternating current at about 500 kHz	Ionic agitation and microscopic friction raise tissue temperature to around 70 to 85 degrees Celsius for several seconds, causing coagulative necrosis in a sharply delimited volume that depends on electrode geometry, temperature, duration and local microanatomy
STEREOTACTIC RADIOSURGERY, AS IN THE GAMMA KNIFE	Hundreds of collimated gamma-ray beams focused on an isocentre only a few millimetres across, with no intraparenchymal electrode	**High doses of about 140 to 180 Gy at the target, typically the anterior limb of the internal capsule in radiosurgical capsulotomy**
MAGNETIC RESONANCE GUIDED FOCUSED ULTRASOUND	Ultrasound delivered through the skull and focused on a deep target, using acoustic rather than ionising energy	At the focal point, absorption translates into heating to 55 to 60 degrees Celsius, producing thermal necrosis

PITFALL

Carrying a temperature or a dose across from one energy modality to another. The figures belong to the physics of each method: a coagulation temperature is not an ultrasound temperature, and neither is a radiosurgical dose. Quote a figure only with the modality it was measured in.

23.6 The indications, and what the evidence rests on

Ablation is offered for a very short list of conditions, and only after ordinary treatment has genuinely failed. The two current indications are:

- Genuinely refractory obsessive-compulsive disorder. The algorithm leading to surgical referral belongs to the anxiety, obsessive-compulsive and trauma-related disorder material.
- Refractory depression, on the same requirement of exhausted, adequately delivered treatment.

The headline finding is easy to misquote. Ablative approaches are not inferior in efficacy to stimulation techniques, with response rates around 50 to 60 percent, but the evidence base is largely composed of uncontrolled studies and direct comparative trials remain lacking. The caveat is half the finding: ablation has not been shown less effective than stimulation, it has been shown on weaker designs.

Procedure by procedure, the picture is consistent. Radiofrequency and radiosurgical capsulotomy show similar efficacy in several open-label series in refractory obsessive-compulsive disorder, Gamma Knife capsulotomy is now the more commonly used, and one double-blind controlled trial shows significant differences between active and sham; most studies remain single-centre

and non-randomised, and adverse effects including apathy, fatigue and cognitive change are described as not negligible. For cingulotomy, large cohorts show significant improvement, but effect sizes are more modest under modern outcome criteria: in one cohort of 64 patients with refractory obsessive-compulsive disorder, partial response was seen in 47 percent at 11 months, rising to 69 percent at 64 months, with 30 of the 64 undergoing a further procedure. Subcaudate tractotomy may confer a higher response rate than a repeat cingulotomy in cingulotomy non-responders, on a series of 31 patients. Limbic leucotomy shows the staging effect most sharply: it was effective in only 20 percent as the initial procedure but in 50 percent when staged after one or more failed cingulotomies, while the original series of 66 patients reported improvement in 89 percent with obsessive-compulsive disorder and 78 percent with major depression.

Response is a scale threshold, not a clinical judgement: response in obsessive-compulsive disorder is a 35 percent or greater improvement on the Yale-Brown Obsessive Compulsive Scale and remission a score under 8, with the source's own warning that the scale is not strictly linear, so dichotomous responder classification should be interpreted cautiously. The harms are real if uncommon: apathy, seizures, urinary incontinence and memory problems are reported in a minority.

TRAP **Quoting the response rate without the design that produced it.** Set beside a controlled stimulation trial, such a figure invites a comparison of numbers not measured in comparable ways. Carry both halves, and note that the source's own "not inferior" is a failure to show a difference on uncontrolled designs rather than a demonstration of equivalence: efficacy is not SHOWN inferior, and the studies are largely uncontrolled, single-centre and non-randomised, with direct comparative trials lacking.

23.7 The ethics, and the restriction that governs the operation

Ethics here constrains a living procedure; it is not a coda to the Freeman story. Contemporary psychosurgery is only ethically acceptable if it strictly respects the four principles of bioethics, autonomy, beneficence, non-maleficence and justice, in a population that is highly vulnerable and whose capacity to consent may be impaired and should be assessed with specific tools. The evidence problem then becomes an ethical one: limited worldwide experience and the scarcity of class I to II evidence for most procedures complicate the provision of fair, clear and balanced information to patients and families.

The consent failure to guard against has a name. **Therapeutic misconception** is the risk that a patient interprets participation in a clinical trial as individualised care, which becomes likelier when a neurosurgical option is perceived as a last therapeutic intervention. The corrective sits in the consent: it must clearly convey the experimental nature of some of these interventions, the uncertainty of outcomes, and a realistic balance of potential benefits and risks.

Historical controversies and abuses in prisoners and minors led the National Commission for the Protection of Human Subjects, in 1977, to recommend significant restrictions on psychosurgery. That is United States regulatory history and nothing more. In current practice, ethical tension persists at four levels:

- Social: pressure to control aggression or addictions.
- Medical: research enthusiasm.
- Industrial: device-related economic interests.
- Personal: concerns about identity and integrity.

Justice is easily left abstract; the literature makes it concrete through cost: deep brain stimulation is among the most expensive medical therapies and requires rigorous cost-effectiveness and equity analysis when proposed for treatment-resistant psychiatric disorders, named here for that principle only.

GOOD TO REMEMBER

The ethical pressure does not point in one direction. The same literature describes psychosurgery as occupying a narrow corridor between underuse and misuse, with current referral rates strikingly low relative to the burden of treatment-resistant illness. A trainee who has absorbed only the abuses reads every referral as a warning; one who has absorbed only the modern imaging underweights what a permanent lesion costs when refractoriness was assumed.

IN INDIA

The Indian constraint is statutory, not merely professional, and it is double: psychosurgery may not be performed on a person unless that person's informed consent has been obtained and the concerned Board has also approved it. That provision belongs to the Mental health legislation and forensic psychiatry notes' account of psychosurgery under the mental health legislation, is named here in words and never renumbered; go there for the wording and the route to the Board. Whether an implanted deep brain stimulation system falls inside that statutory definition belongs to this chapter's deep brain stimulation account and is a declared gap. The United States restriction above is not an Indian instrument and must never be written as though it were.

Ablative psychosurgery destroys tissue and is therefore not a member of the stimulation family, however invasive that family becomes; the procedures in use are anterior capsulotomy at the anterior limb of the internal capsule, anterior cingulotomy at the anterior cingulate in its triple-lesion form, subcaudate tractotomy at the posterior orbitofrontal cortex, and limbic leucotomy as the staged combination of the last two, made by radiofrequency, radiosurgery or focused ultrasound with parameters specific to each; the indications are genuinely refractory obsessive-compulsive disorder and depression; efficacy is not SHOWN inferior to stimulation and rests on largely uncontrolled designs with no direct comparative trials; and consent must survive impaired capacity and the therapeutic misconception, with India adding a statutory requirement of informed consent and Board approval that this chapter names and does not restate.

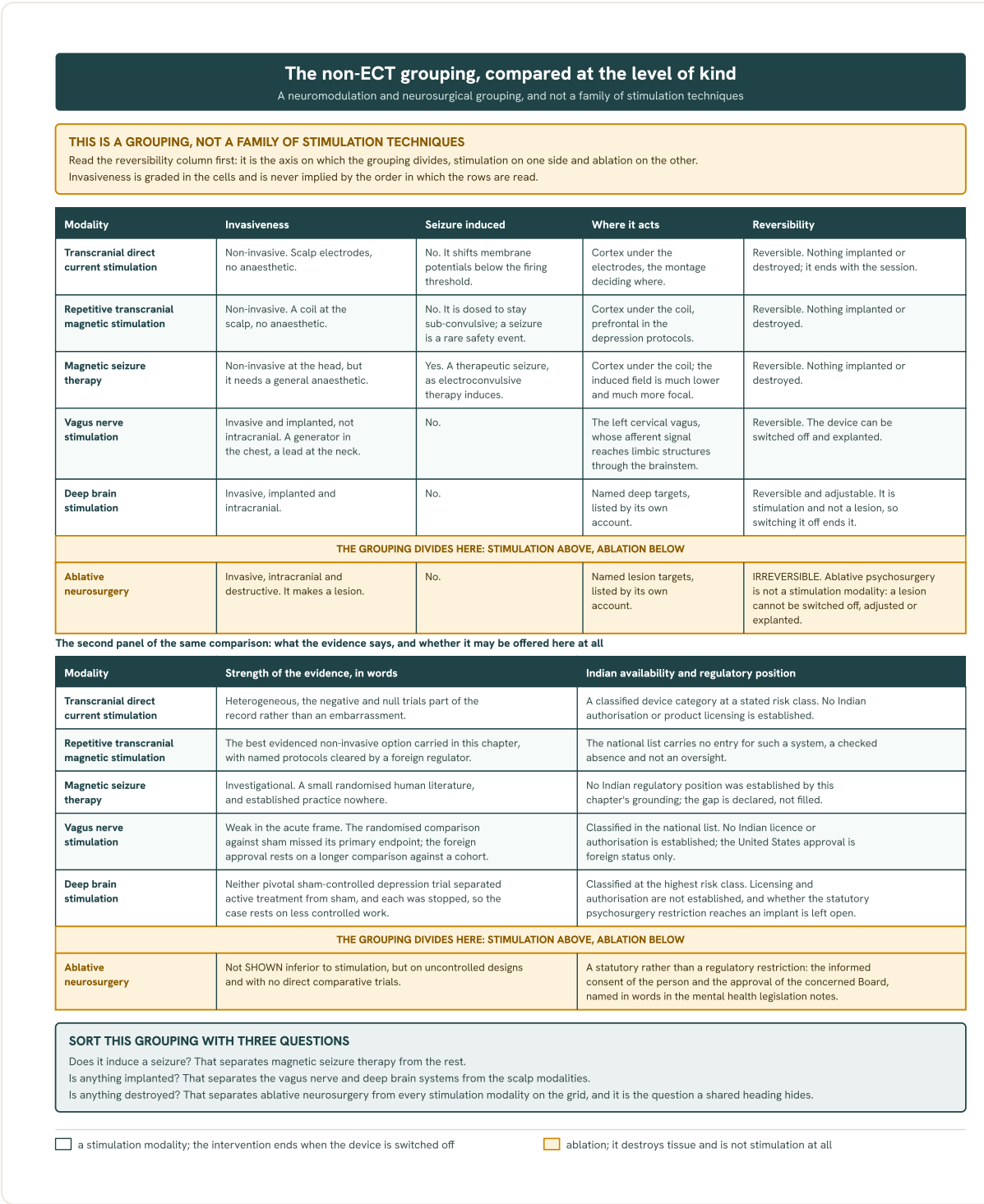
Cross-cutting synthesis

24 · Cross-cutting synthesis

Every other account in this chapter teaches one topic; this one is about how they hold together. An induced seizure is delivered at a measured dose, through a chosen placement, with a chosen shape of stimulus, on a chosen schedule, under an anaesthetic somebody else is giving, to a patient whose agreement has to be lawful. Move any one and something else moves with it: the topics **interlock**. The joint they turn on is the **stimulation dose**: what is delivered, how much, where, and what it costs. One boundary belongs here because it runs across the seam rather than inside any one item: the MOTOR THRESHOLD that bounds repetitive transcranial magnetic stimulation is a different construct from the seizure threshold that bounds this treatment, measured differently and meaning something different, and neither is carried across to the other. Each is taught in full by the item that owns it.

Owned here and nowhere else: the synthesis itself, how the parts move against one another, and the non-ECT comparison at the level of kind. Named in a clause and never re-derived, each with its own account: the mechanism theories, the seizure threshold, the placements and waveform, the placement-by-dose comparison, the cognitive cost, the anaesthetic conduct, the acute course, continuation and maintenance, the interacting drugs, and consent. No dose, multiple, percentage, current or session count is printed here.

Dose	Differs	Kind
THE SEAM THAT ALMOST EVERY OTHER DECISION HERE IS EXPRESSED AGAINST	THE DIRECTION AND THE SIZE OF THE TRADE, BY PARAMETER AND BY PLACEMENT	THE LEVEL THE NON-ECT MODALITIES ARE COMPARED AT, NEVER A SIBLING'S SETTINGS



The non-ECT grouping, compared at the level of kind

A neuromodulation and neurosurgical grouping, and not a family of stimulation techniques

THIS IS A GROUPING, NOT A FAMILY OF STIMULATION TECHNIQUES

Read the reversibility column first: it is the axis on which the grouping divides, stimulation on one side and ablation on the other. Invasiveness is graded in the cells and is never implied by the order in which the rows are read.

Modality	Invasiveness	Seizure induced	Where it acts	Reversibility
Transcranial direct current stimulation	Non-invasive. Scalp electrodes, no anaesthetic.	No. It shifts membrane potentials below the firing threshold.	Cortex under the electrodes, the montage deciding where.	Reversible. Nothing implanted or destroyed; it ends with the session.
Repetitive transcranial magnetic stimulation	Non-invasive. A coil at the scalp, no anaesthetic.	No. It is dosed to stay sub-convulsive; a seizure is a rare safety event.	Cortex under the coil, prefrontal in the depression protocols.	Reversible. Nothing implanted or destroyed.
Magnetic seizure therapy	Non-invasive at the head, but it needs a general anaesthetic.	Yes. A therapeutic seizure, as electroconvulsive therapy induces.	Cortex under the coil; the induced field is much lower and much more focal.	Reversible. Nothing implanted or destroyed.
Vagus nerve stimulation	Invasive and implanted, not intracranial. A generator in the chest, a lead at the neck.	No.	The left cervical vagus, whose afferent signal reaches limbic structures through the brainstem.	Reversible. The device can be switched off and explanted.
Deep brain stimulation	Invasive, implanted and intracranial.	No.	Named deep targets, listed by its own account.	Reversible and adjustable. It is stimulation and not a lesion, so switching it off ends it.
THE GROUPING DIVIDES HERE: STIMULATION ABOVE, ABLATION BELOW				
Ablative neurosurgery	Invasive, intracranial and destructive. It makes a lesion.	No.	Named lesion targets, listed by its own account.	IRREVERSIBLE. Ablative psychosurgery is not a stimulation modality: a lesion cannot be switched off, adjusted or explanted.

The second panel of the same comparison: what the evidence says, and whether it may be offered here at all

Modality	Strength of the evidence, in words	Indian availability and regulatory position
Transcranial direct current stimulation	Heterogeneous, the negative and null trials part of the record rather than an embarrassment.	A classified device category at a stated risk class. No Indian authorisation or product licensing is established.
Repetitive transcranial magnetic stimulation	The best evidenced non-invasive option carried in this chapter, with named protocols cleared by a foreign regulator.	The national list carries no entry for such a system, a checked absence and not an oversight.
Magnetic seizure therapy	Investigational. A small randomised human literature, and established practice nowhere.	No Indian regulatory position was established by this chapter's grounding; the gap is declared, not filled.
Vagus nerve stimulation	Weak in the acute frame. The randomised comparison against sham missed its primary endpoint; the foreign approval rests on a longer comparison against a cohort.	Classified in the national list. No Indian licence or authorisation is established; the United States approval is foreign status only.
Deep brain stimulation	Neither pivotal sham-controlled depression trial separated active treatment from sham, and each was stopped, so the case rests on less controlled work.	Classified at the highest risk class. Licensing and authorisation are not established, and whether the statutory psychosurgery restriction reaches an implant is left open.
THE GROUPING DIVIDES HERE: STIMULATION ABOVE, ABLATION BELOW		
Ablative neurosurgery	Not SHOWN inferior to stimulation, but on uncontrolled designs and with no direct comparative trials.	A statutory rather than a regulatory restriction: the informed consent of the person and the approval of the concerned Board, named in words in the mental health legislation notes.

SORT THIS GROUPING WITH THREE QUESTIONS

Does it induce a seizure? That separates magnetic seizure therapy from the rest.
Is anything implanted? That separates the vagus nerve and deep brain systems from the scalp modalities.
Is anything destroyed? That separates ablative neurosurgery from every stimulation modality on the grid, and it is the question a shared heading hides.

a stimulation modality; the intervention ends when the device is switched off

ablation; it destroys tissue and is not stimulation at all

Fig 27.9. The non-ECT interventions are read as one grouping and not as one family: transcranial direct current stimulation, repetitive transcranial magnetic stimulation, magnetic seizure therapy, vagus nerve stimulation and deep brain stimulation are stimulation modalities whose intervention ends when the device is switched off, while ablative neurosurgery destroys tissue and is irreversible, which is why the reversibility column rather than the order of the rows is what the grid is read on, and the one Indian instrument speaking directly to a modality is statutory, the psychosurgery restriction requiring the informed consent of the person and the approval of the concerned Board.

24.1 One measured quantity, and almost everything expressed against it

No account of how this treatment works is established, and the field says so in its own words, but every candidate account is an account of what an induced seizure does, so the seizure is the therapeutic event and the dose produces it. That dose is not a device setting. It is a multiple of a quantity measured in this patient, the lowest dose at which this brain will seize, which varies enormously and moves across a course. This is why the chapter reads as parameters rather than

protocols: fix the placement and the multiple still has to be chosen; fix the multiple and the placement changes what it buys.

Follow the chain forward and the chapter appears in order. The threshold sets the dose. The dose and placement together settle efficacy and cognitive cost, which is why the comparison between placements has no answer until the multiple is named. The cognitive cost is not one deficit but several on different clocks. The schedule spends the dose over time. Continuation and maintenance settle what happens to the gain once the course stops. Drugs continued across it move the threshold underneath everything. And the law settles, before any of this, whether the treatment may be given.

24.2 The trades differ, and the difference is the finding

Many of the choices along that seam trade among speed of response, efficacy and cognitive cost. Where they do, the direction and size of that trade differ by parameter and by placement, and the chapter asserts no law covering all of them, because several are not that kind of trade at all.

RULE **Read the trade off the parameter in front of you, and never inherit a direction from the parameter beside it.** Every general version of that rule attempted here was contradicted by the parameter next along. State what your parameter trades, at the placement you are standing at. Where a parameter does not behave like its neighbour, that is the finding.

CHOICES THAT DO TRADE AMONG THE THREE	WHAT MOVING IT ACTUALLY DOES	TAUGHT IN FULL BY
STIMULUS DOSE ABOVE THRESHOLD, AT RIGHT UNILATERAL PLACEMENT	The dose-response is steep here, so raising the multiple buys efficacy; the placement's cognitive advantage erodes as that multiple rises, without being abolished.	Stimulus dosing, and the placement comparison.
STIMULUS DOSE ABOVE THRESHOLD, AT BITEMPORAL PLACEMENT	Efficacy is already near maximal at a low multiple, so a higher dose here largely buys cognitive cost alone. The same knob, turned the same way, makes a different purchase.	The placement comparison.
THE PLACEMENT ITSELF	Not a fixed ranking, and never usable as one. Right unilateral treatment is markedly inferior near threshold and converges toward bitemporal efficacy at a high multiple without established superiority over it; the comparison narrows and does not reverse.	The placement comparison.
PULSE WIDTH	A narrower pulse is associated with a cognitive advantage at right unilateral placement in depressive illness, with antidepressant efficacy possibly compromised. The relation is bounded to pulse width inside a placement.	Technique and electrode placement.
TREATMENT FREQUENCY	A faster schedule buys speed of response at a higher cognitive price; end-of-course efficacy is equivalent between the schedules. Frequency is not an efficacy lever, and writing it as one imposes cognitive injury for a gain that does not exist.	The acute course and its schedules.

The second table stops the first from hardening into a law. These choices sit on the same seam and are not purchases among speed, efficacy and cognitive cost at all.

CHOICES THAT ARE NOT THAT KIND OF TRADE	WHAT MOVING IT ACTUALLY DOES	TAUGHT IN FULL BY
THE INDUCTION AGENT AND THE CONDUCT OF THE ANAESTHETIC	A different currency altogether. The agent shapes the seizure the psychiatrist then has to read, and is chosen against separate rankings for its effect on the threshold, the cardiovascular response and emergence, at no cognitive price.	The pre-ECT workup and anaesthesia.
THE AIRWAY, THE MONITORING AND THE TOURNIQUET	Not trades among speed, efficacy and cognitive cost in any form. The airway and monitoring buy safety and information; the tourniquet buys a limb on which the motor seizure can still be seen.	The pre-ECT workup and anaesthesia.
DRUGS CONTINUED ACROSS THE COURSE	A hazard rather than a purchase. They move the threshold underneath everything above, in either direction, each with a management step, and the direction belongs to the drug.	Drug interactions and concurrent medications.

24.3 Buying the right thing at the least price

A map with no single direction still yields a task. Decide first what this patient needs, efficacy or speed, because those are different purchases not made with the same parameter. Then buy it at the least cognitive price the illness and the law allow: the placement and multiple chosen together, the pulse width with them, the schedule for what it genuinely delivers. Where speed itself is the requirement, the schedule that answers it is the accelerated one, assembled only in this chapter's account of the course, frequency and number of sessions.

The cognitive price is not a fixed toll: the same efficacy can often be bought at different prices depending on how the parameters are combined.

> **PITFALL**
>
> The real-world error is not getting a parameter wrong. It is getting one right and then carrying its direction to the next: a clinician who has learned that a higher multiple earns a response at right unilateral placement raises it at bitemporal placement too, where efficacy was already near maximal, buying cognitive cost with nothing on the other side.

24.4 The handoffs a real course crosses

A single patient can walk a resident through most of this chapter. The difficulty is rarely inside a topic but at the joints, where the question changes shape and the governing account with it.

- **An indication becomes a consent question.** The presentation earns the treatment clinically, and the next question is not the dose but the authority: who may agree, and whether the route

is complete. Urgency strengthens the clinical case and completes no route.

- **A threshold titration becomes a placement decision.** The threshold is only part of an answer, because the multiple worth using depends on where the electrodes sit. They are chosen together, or the dose is chosen wrongly with great precision.
- **A cognitive complaint becomes a dosing change.** A patient reporting memory loss is a finding, not a nuisance, and the response is a change in the parameters that move it: placement, multiple, pulse width, schedule, drugs.
- **An acute response becomes a maintenance question.** A course that worked does not end the decision: relapse is the rule when nothing active follows.
- **A failed medication ladder becomes a modality question.** When the drugs have not worked, the question stops being which drug and becomes which intervention.

24.5 The law bounds the seam rather than sitting on it

The last element in the chain behaves unlike the others, and treating it as one more parameter is the commonest structural error. Consent, capacity and the statutory provisions do not tune the treatment; they settle whether it may be given at all, and on whose authority. Clinical urgency does not convert a missing authority into a present one: a lawful route has to be established separately, the emergency-treatment provision of the Act cannot supply it, and that is a point about treatment authority and not about admission, because a capacitous adult can consent to this treatment with no admission at all. The routes, an advance directive refusing treatment, the nominated representative and the minor's gate belong to this chapter's account of consent.

Statutory bars sit at the edges of the chapter, not inside its parameters. Unmodified, or direct, electroconvulsive therapy is unlawful in India rather than merely inadvisable, as this chapter's modified-against-unmodified contrast states with the history behind it. And ablative psychosurgery may not be performed without the person's informed consent and the approval of the concerned Board. Each provision belongs to the Mental health legislation and forensic psychiatry notes, named here in words and renumbered nowhere.

24.6 The non-ECT grouping, compared at the level of kind

The second part of the chapter meets the other interventions that act on the brain directly. They are read in the bank's own sequence, which ranks nothing: the grading of **invasiveness** is taught here, never implied by which item comes first. Each modality's own settings belong to its own account, and the accompanying plate carries this comparison as one grid.

Before the grid can be read honestly, the grouping needs its right name. This is a **neuromodulation and neurosurgical grouping**, not a family of stimulation techniques. The stimulation members share a property mattering more than their differences: switching the device off ends the intervention. Ablative neurosurgery does not share it. It destroys tissue, it is not stimulation at all, and it sits on the grid for contrast rather than as the invasive end of one gradient. That is why **reversibility** is the column to read first. It is the axis on which the grouping divides, stimulation on one side and ablation on the other, and the ablative psychosurgery account owns that irreversibility in full.

MODALITY	INVASIVENESS	SEIZURE INDUCED	WHERE IT ACTS	REVERSIBILITY
TRANSCRANIAL DIRECT CURRENT STIMULATION	Non-invasive. Scalp electrodes, no anaesthetic.	No. It shifts membrane potentials below the firing threshold.	Cortex under the electrodes, the montage deciding where.	Reversible. Nothing implanted or destroyed; it ends with the session.
REPETITIVE TRANSCRANIAL MAGNETIC STIMULATION	Non-invasive. A coil at the scalp, no anaesthetic.	No. It is dosed to stay sub-convulsive; a seizure is a rare safety event.	Cortex under the coil, prefrontal in the depression protocols.	Reversible. Nothing implanted or destroyed.
MAGNETIC SEIZURE THERAPY	Non-invasive at the head, but needs a general anaesthetic.	Yes. A therapeutic seizure, as electroconvulsive therapy induces.	Cortex under the coil; the induced field is much lower and much more focal.	Reversible. Nothing implanted or destroyed.
VAGUS NERVE STIMULATION	Invasive and implanted, not intracranial. A generator in the chest, a lead at the neck.	No.	The left cervical vagus, whose afferent signal reaches limbic structures through the brainstem.	Reversible. The device can be switched off and explanted.
DEEP BRAIN STIMULATION	Invasive, implanted and intracranial.	No.	Named deep targets, listed by its own account.	Reversible and adjustable. It is stimulation and not a lesion, so switching it off ends it.
ABLATIVE NEUROSURGERY	Invasive, intracranial and destructive. It makes a lesion.	No.	Named lesion targets, listed by its own account.	Irreversible. Ablative psychosurgery is not a stimulation modality: a lesion cannot be switched off, adjusted or explanted, and this cell is why the grouping is not one family.

The remaining axes are the second panel of the same comparison: what a modality is settles what it could do, while its evidence and regulatory position settle whether it may be offered at all.

MODALITY	STRENGTH OF THE EVIDENCE, IN WORDS	INDIAN AVAILABILITY AND REGULATORY POSITION
TRANSCRANIAL DIRECT CURRENT STIMULATION	Heterogeneous, the negative and null trials part of the record rather than an embarrassment.	A classified device category at a stated risk class. No Indian authorisation or product licensing is established.
REPETITIVE TRANSCRANIAL MAGNETIC STIMULATION	The best evidenced non-invasive option carried in this chapter, with named protocols cleared by a foreign regulator.	The national list carries no entry for such a system, a checked absence and not an oversight.
MAGNETIC SEIZURE THERAPY	Investigational. A small randomised human literature, and established practice nowhere.	No Indian regulatory position was established by this chapter's grounding; the gap is declared, not filled.
VAGUS NERVE STIMULATION	Weak in the acute frame. The randomised comparison against sham missed its primary endpoint; the foreign approval rests on a longer comparison against an observational cohort.	Classified in the national list. No Indian licence or authorisation is established; the United States approval is foreign status only.
DEEP BRAIN STIMULATION	Neither pivotal sham-controlled depression trial separated active treatment from sham, and each was stopped, so the case rests on less controlled work.	Classified at the highest risk class. Licensing and authorisation are not established, and whether the statutory psychosurgery restriction reaches an implanted system is left open.
ABLATIVE NEUROSURGERY	Not SHOWN inferior to stimulation, but on uncontrolled designs and with no direct comparative trials.	A statutory rather than a regulatory restriction: informed consent of the person and approval of the concerned Board, named in the Mental health legislation and forensic psychiatry notes.

GOOD TO REMEMBER

Sort this grouping with three questions. Does it induce a seizure? That separates magnetic seizure therapy from the rest. Is anything implanted? That separates the vagus nerve and deep brain systems from the scalp modalities. Is anything destroyed? That separates ablative neurosurgery from every stimulation modality on the grid, and it is the question a shared heading hides.

IN INDIA

Read the Indian column as four claims and never as one. A device classification says which regulatory pathway a device sits in; a product licence says a named device may be sold here; an indication-specific authorisation says it may be sold for a stated psychiatric use; a foreign clearance speaks to another jurisdiction, not this one. On this grid the classification claim is usually the only one available: Indian licensing and authorisation are not established for any modality, written as not established rather than left for a reader to infer. The one Indian instrument speaking directly to a modality is statutory, the psychosurgery restriction named above.

MNEMONIC

ONE KNOB AT A TIME

Before stating any trade, name the knob exactly: the dose, and at which placement; the pulse width, and at which placement; the schedule; the anaesthetic. Then read that knob's trade and stop. Do not carry the direction to the next, which has its own. And some controls here, the airway, the monitoring and the tourniquet, are not on this trade at all.

The stimulation dose is the seam: mechanism proposes it, the threshold measures it, placement conditions what it buys, the cognitive cost prices it, and the law bounds who may receive it. Many of the choices along that seam trade among speed of response, efficacy and cognitive cost, and where they do the direction and size of the trade differ by parameter and by placement; several, the airway, the monitoring and the tourniquet among them, are not that kind of trade in any form. The non-ECT grouping is not one family: its stimulation members end when the device is switched off, while ablation destroys tissue and cannot be undone.

TRAP **Answering a synthesis question with a single general rule.** The question invites it, because an examiner asking what the trade-off is sounds like one wanting a single sentence, and one sentence sounds like mastery. It cannot be right here: the relation at right unilateral placement is not the one at bitemporal placement, and the schedule's is different again. Name the parameter, give its relation with its condition, then say the others run differently.

Apply and consolidate

25 · Applying the chapter

A vignette in this territory almost never asks whether electroconvulsive therapy works; it asks whether this patient may lawfully be given it, with what, and at what price. Those are separate findings reached by separate reasoning, and the candidate who learned the chapter as a list of facts answers the question nobody asked. Name the kind of question before reaching for what you know.

These vignettes assert nothing of their own. Every threshold, multiple, interval, effect size and statutory provision they deploy is taught, and cited to its source, by the account that owns it, and is named here rather than restated. Cover each answer, decide what you would do, then read on.

Earned	Authorised	Priced
WHETHER THE PRESENTATION INDICATES THE TREATMENT	WHETHER A LAWFUL ROUTE EXISTS TO GIVE IT	WHAT THE CHOSEN PARAMETER ACTUALLY BUYS

25.1 How to work a vignette in this chapter

Sort the case before you answer it.

- An **indication question** asks whether this presentation earns the treatment.
- An **authority question** asks on what lawful basis it may be given, answered from the Act and never from severity.
- A **parameter question** asks what a change to placement, dose, pulse width, schedule or anaesthetic conduct buys and costs.
- A **modality question** asks whether a different device is the better answer, a family whose terms are not the terms of electroconvulsive therapy.

Each parameter has its own relation to efficacy and its own to cognition, so a relation established for one is never inherited by another, and some decisions here are not trades of that kind at all.

RULE **An indication and a lawful route are separate findings, and neither supplies the other.** Deciding that a presentation earns this treatment settles the clinical question completely and the legal question not at all. Answer both, in that order, wherever treatment is proposed, and say which of the two your reasoning has discharged.

25.2 Vignette: the emergency the emergency provision cannot authorise

Presentation. A woman in her thirties is in the third week of a severe depressive episode. She has taken almost nothing by mouth for days, is at times mute and immobile, refuses oral medication, and is dry, tachycardic and deteriorating. The consultant says she needs electroconvulsive therapy tonight. The resident proposes to treat under the Act's emergency-treatment provision and asks the family to sign an admission form.

Decision point. On what authority may this be given tonight, and does the urgency change the answer.

Reasoning to show. Split the case at once. The clinical half is uncontentious: food and fluid refusal, immobility and a deteriorating physical state secondary to a psychiatric condition are what the indications account carries as first-line where a rapid response is needed to avert harm. That finding is complete, and it authorises nothing. The legal half is where the resident errs: this treatment can be clinically indicated in an emergency, and the Act's emergency-treatment provision cannot authorise it, because that provision expressly excludes it. A lawful route must be established separately, and urgency does not cure an incomplete one.

The limb candidates lose: treatment authority is not admission authority. A capacitous adult can consent with no admission at all, so the emergency route closing never converts the question into whether she must first be admitted, and a family signature on an admission form is nobody's consent to this treatment. Assess her **capacity**, now and for this decision, the Act presuming every person to have it. If she retains it, the route is her own informed consent, intervention-specific and covering risks, benefits and alternatives; if not, the supported-admission route runs in its fixed order, the directive first, then her own consent with her representative's support, then temporary substituted consent.

Answer. Say the indication is clear and that it is not the authority. Do not treat under the emergency provision, which excludes this treatment by name. Assess capacity tonight, because that is what urgency buys: speed in establishing a route, never a shorter one. With capacity, take her own informed consent; without it, look for a directive first, then work the supported route. Record which route was used, not only that a form was signed, and rehydrate her now.

PITFALL

Writing the emergency in as though it were a source of authority. It is a clinical urgency and nothing more, and the Act's emergency-treatment provision excludes this treatment by name. The paired error costs the same marks: assuming that because that route is closed, an admission must be arranged first. It need not be, if the adult has capacity and consents.

25.3 Vignette: the directive that says no

Presentation. A man in his forties with recurrent severe depression had electroconvulsive therapy in an earlier episode. While well, he made a written **advance directive** refusing it in future. He is now in severe relapse, has lost capacity, is refusing food and is at high risk. His wife, his nominated representative, wants it given. The senior registrar says the directive cannot have been meant to cover a picture like this, and that the team should proceed.

Decision point. Does the directive bind the team, and if it does, what is the route out of it.

Reasoning to show. Name the instrument first, because a resident who has met it only as a way of saying yes reads refusal as obstruction. It runs in either direction, is invoked only when the maker ceases to have capacity, lapses when capacity returns, and is overridden by a later capacitous decision. A refusal is the instrument working, not a defect in it. The duty to treat in accordance with a valid directive falls on the medical officer in charge of the establishment and on the psychiatrist in charge of treatment, subject only to Board review. What that duty lacks is the examinable part: no bedside-override limb, no urgency limb, no senior-clinician limb.

The route out is an application. Where a professional, a relative or a care-giver desires not to follow a directive, the Act directs an application to the concerned Board, which hears all parties and may uphold, modify, alter or cancel it against the considerations the provision lists: voluntariness, whether he intended it to apply to circumstances such as these, whether he was sufficiently informed, whether he had capacity, and whether its content is contrary to other law.

That intention limb is what the registrar is arguing, and it is argued to the Board, never settled at the bedside.

Answer. The directive binds the team. Do not give the treatment. File the application to the concerned Board today, framed on the intention limb the registrar has already argued, and state the clinical urgency in it, urgency being a reason for the Board to move rather than to be bypassed. Meanwhile treat the relapse by every lawful means that remains. If capacity returns the directive lapses. Record the decision not to override it and the date of the application.

TRAP **Treating a refusing directive as a clinical problem to be solved.** A resident who has met only the authorising face of the instrument reads a refusal as an obstacle to good care, then solves it the way clinical obstacles are solved, at the bedside and under time pressure. That is the unlawful act. The only lawful exit is the application, and urgency creates no exception to it.

25.4 Vignette: the hemiplegic arm and the wrong clock

Presentation. A man in his sixties has a dense right hemiplegia following a stroke some months ago, and has since developed a severe depressive episode with poor oral intake. Electroconvulsive therapy is planned. The registrar reasons that the stroke is no longer recent, that the risk has settled, and tells the anaesthetist that the usual depolarising relaxant may be used.

Decision point. Is the recency of the stroke the right marker for the blockade decision, and what emergency is being risked.

Reasoning to show. Two risks run here on opposite clocks, which is why one word carried from one to the other is dangerous. The word recent, as it attaches to a cerebrovascular or cardiac event in the risk-profile account, concerns the cardiovascular and cerebral risk of the induced seizure: highest close to the event and falling with time, which is why that account recommends postponement. The registrar has read that direction correctly and applied it to a question it does not govern. The blockade question has its own mechanism: after denervation or disuse, extrajunctional acetylcholine receptors up-regulate, so a depolarising agent depolarises a greatly enlarged receptor population and releases potassium from it.

The receptors must change first, so the danger RISES as the up-regulation proceeds, peaking at the interval the workup and anaesthesia account states, and the label's own contraindication applies after the acute phase. The onset and duration of that risk period are expressly unknown, so no date of safety is printed here. Reading recency across therefore inverts the window: it refuses the relaxant before the receptors have up-regulated, where the label still requires great caution and prints no interval of safety, and hands it to the established hemiplegic in whom danger has built for weeks. It clears exactly the wrong patient, and the emergency it clears him into is severe hyperkalaemia which may result in cardiac arrest.

Answer. The stroke being old is a reason for concern about the relaxant, not reassurance about it, and saying so explicitly is the mark. Nor is this a decision for the anaesthetic room: the relaxant question belongs to the pre-treatment assessment, before the patient is fasted and listed. Ask what denervating or disuse lesion exists and how long ago the insult was, remembering that the

risk rises toward the stated peak rather than falling away from the insult, and ask about the emergencies that do not share this mechanism, lowered plasma cholinesterase ending in prolonged apnoea and the ryanodine route in malignant hyperthermia. The choice and dose of relaxant remain the anaesthetist's decision.

25.5 Vignette: which placement, and at what multiple

Presentation. A schoolteacher in her fifties with a severe first depressive episode has consented to electroconvulsive therapy and is frightened, specifically and articulately, of losing her memory. She was started on right unilateral placement at a dose just above her measured seizure threshold, on the stated ground that this is the gentle option. After several treatments she is no better, and the team is discussing a switch to a bilateral placement.

Decision point. Was the near-threshold unilateral course the gentle choice, and what does the switch change.

Reasoning to show. Refuse the question as posed: whether right unilateral placement is as good as a bilateral one has no answer until the dose is named, because the placements have different dose-response relations. Near the threshold, right unilateral treatment is markedly inferior while bilateral efficacy is already near its maximum, so the near-threshold unilateral course is not a gentle version of the treatment but an under-treated one. As the multiple rises, right unilateral treatment converges toward bitemporal efficacy without establishing superiority over it, and the cognitive advantage it buys erodes as that same multiple rises; state both halves or neither, since erosion is not abolition and a real advantage survives at the top of the range, domain-specific rather than broad.

Then the relation the chapter exists to protect. Stimulus dose above the threshold buys efficacy in right unilateral placement, where the dose-response is steep; in bitemporal placement, where efficacy is already near-maximal at a low multiple, a higher dose largely buys cognitive cost alone. That is one relation for one parameter and its mirror for another, and it is not a direction to be carried anywhere else. The threshold is measured in this patient rather than looked up and rises across a course, so the multiple she receives today is not the one she received in the first week unless somebody has checked.

Answer. Name the multiple before answering anything. The failure so far is a dosing failure rather than a placement failure, so the first correction is to dose the unilateral placement adequately against a re-measured threshold rather than abandon the placement carrying the advantage she asked for. Tell her what that advantage is and is not: it survives in named domains rather than across the board, and the same rising dose that buys the efficacy partly spends it. If an adequate course still fails, a switch is legitimate on efficacy grounds, with the cognitive cost stated.

25.6 Vignette: the memory complaint, and the family who want it faster

Presentation. A man in his forties is midway through an acute course for a severe depressive episode and is improving, slowly. His wife says he cannot recall the week of his admission at all and has patchy gaps for the months before it, including a family wedding. He scores normally on

the bedside screen the unit uses. The family, impatient with the pace, ask that treatments be given more often so that the treatment works better.

Decision point. What to do about the memory complaint, and whether to raise the frequency to improve the outcome.

Reasoning to show. Take the frequency request first, because it rests on a false premise. Treatment frequency buys speed of response; it is not an efficacy lever. The acute-course account established that the thrice-weekly and twice-weekly schedules reach equivalent antidepressant outcome at the end of the course, that the faster schedule reaches response sooner, and that it costs more in memory impairment even where the total number of treatments does not differ. The cost belongs to the spacing, not the number of treatments. The family are asking, without knowing it, to pay a cognitive price for a gain that does not exist; the malignant presentations measured in days earn speed, and this man does not.

Then the memory complaint, separating report from measurement. The cognitive cost is not one deficit but separate phenomena on separate clocks. Postictal confusion belongs to the minutes after each treatment. **Anterograde amnesia** is the difficulty laying down new memories while the course runs, which is why the week of admission is patchy, and it is the limb that recovers after the course. **Retrograde amnesia** is the loss of what was already laid down, with a gradient toward events near the treatment, which is what the missing wedding is; the Indian guideline calls it among the most distressing adverse effects and difficult to measure using objective tests. The normal bedside screen therefore excludes nothing.

Where his wife's account and the ward's testing diverge, the divergence is the finding. The Indian guideline directs that subjective memory impairment be given due importance alongside objective assessment, the reason report exceeds measurement being a caution about the instruments, not the patient. Its menu is what can be done: space the sessions, switch to unilateral placement, switch to a briefer pulse width, reduce the stimulus dose, reduce or stop drugs affecting cognition, reduce a high anaesthetic dose, and terminate the course where risks outweigh benefits, each step balanced against the effectiveness it may cost.

Answer. Decline the frequency increase on the right ground: not that it is unsafe, but that it does not buy what is being asked for, the end-of-course outcome being equivalent and only the speed and the cognitive price differing. On the memory, ask about life memories rather than orientation and word lists, because the instrument that finds retrograde loss is a conversation about his own past; record the wife's account as a finding; and space the sessions if anything, the opposite of what was requested. The encoding gap recovers; the loss around the wedding is contested.

RULE **Name the parameter before you name its price, because each has its own relation and none lends its direction to another.** The stimulus dose, the placement, the pulse width, the schedule and the anaesthetic conduct each stand in a different relation to efficacy and to cognition. A candidate who has learned one direction as the law of the chapter will apply it to the parameter it does not fit.

25.7 Vignette: the patient for whom the answer may not be electroconvulsive therapy

Presentation. A software engineer in his thirties has a moderate depressive episode that has not responded to successive adequate antidepressant trials. He is working, eating, sleeping badly, and denies suicidal intent. He is frightened of the memory loss he has read about, and asks instead about the magnetic treatment he has seen advertised; his family have read about a home device and about an implanted stimulator a relative received.

Decision point. Is a non-electroconvulsive device the right answer, and on what axes is that choice made.

Reasoning to show. Establish first that the question is genuinely open, because this case is mishandled by letting a device stand in for an urgently indicated treatment. He carries none of the features that move electroconvulsive therapy to a first-line position: no food or fluid refusal, no catatonia, no psychotic features, no acute suicidal risk. His treatment resistance is a second-line indication, so there is time to choose, and his fear is a reason to explain the cognitive cost rather than to steer him elsewhere. Then refuse the shape of his question: these devices are not a ladder with a single gradient. They differ on whether a seizure is induced at all, on invasiveness, on target, on reversibility, on the evidence for the indication, and on the Indian regulatory position, and those axes do not move together.

Then take each device on its own account. Repetitive transcranial magnetic stimulation induces no seizure, is referenced to a motor threshold measured in this patient, and needs weeks of daily attendance, a real question for a man with a job; its Indian position is a checked absence establishing nothing about licensing. Transcranial direct current stimulation induces no seizure, is authorised for no psychiatric indication in the Indian classification, and did not separate from sham in the depression trial carried here, which answers the home-device question. Deep brain stimulation is a permanently implanted electrode reached only after everything else has failed, and its sham-controlled depression trials missed their primary endpoints. Vagus nerve stimulation drives a peripheral nerve in the neck, its acute sham-controlled endpoint was not significant, and its approval rested on an effect measured over a long window. Carry nothing across: those relations belong inside electroconvulsive therapy.

Answer. The honest reply is a conversation rather than a device. Tell him his presentation does not make electroconvulsive therapy urgent, so there is room to choose; correct what he fears in the separated terms the cognitive account supplies; and if he then refuses it, record that as the valid refusal of a capacitous adult rather than an obstacle. Choose among the devices on the axes that differ, naming for each the evidence for this indication and what the Indian record establishes.

IN INDIA

Three Indian facts decide these cases. The Act supplies the routes and closes the emergency route to this treatment by name. The Indian guideline gives patient-reported memory impairment due importance alongside objective testing. And the Indian device-classification record governs the non-electroconvulsive family, a foreign clearance never being readable across.

25.8 The cases on one grid, and the thread through them

Set side by side, the cases are one discipline in different clothes, and the right-hand column is a single family of mistakes.

VIGNETTE	THE QUESTION IT REALLY ASKS	WHERE THE ANSWER COMES FROM	THE CLASSIC WRONG ANSWER
THE EMERGENCY AT NIGHT	Indication, then authority, as separate findings	The indications account, then the Act's routes	Treating under the emergency provision, or assuming admission comes first
THE DIRECTIVE THAT SAYS NO	Whether a valid refusal binds the team	The duty to follow a valid directive, and Board review	Deciding the maker's intention at the bedside instead of applying to the Board
THE HEMIPLEGIC ARM	Which clock the blockade risk runs on	The workup and anaesthesia account, which owns the window	Reading recency across from the seizure risk, clearing the wrong patient
THE FRIGHTENED TEACHER	What the dose does at this placement	The placement comparison and the stimulus-dosing account	Calling a near-threshold unilateral course the gentle option
THE IMPATIENT FAMILY	What a faster schedule actually buys	The acute-course account and the cognitive account	Raising the frequency to improve the end-of-course outcome
THE ENGINEER AND THE DEVICES	Whether another modality fits, and on what axes	Each device's own account, and the cross-cutting grid	Ranking the family on one gradient, or carrying the electroconvulsive relations across

The thread, stated once. Every case turns on refusing to let one finding answer for another: a clinical indication does not answer an authority question, a risk on one clock does not answer a risk on another, a relation established for one parameter does not answer for a second, and nothing established inside electroconvulsive therapy answers for devices whose terms differ.

MNEMONIC

ROUTE, RELAXANT, RELATION, REMAINDER

Rebuild the closer from these when a vignette empties your head. **Route**: an indication says the treatment is earned, never that it may be given, and urgency does not cure an incomplete route. **Relaxant**: the safety question runs on its own clock, recency is the wrong marker, and it is asked before the patient is fasted. **Relation**: name the parameter before you name its price, because none inherits a direction from another. **Remainder**: everything outside electroconvulsive therapy is a different family on different terms, compared on the chapter's grid rather than ranked.

Every vignette here separates findings that residents merge: the indication from the lawful route, one risk's clock from another's, and each parameter's own relation from a general direction that does not exist.

26 · Consolidation

The last useful thing to do with this chapter is not to read it again but to produce it from memory, with no answer in sight. Everything below is a question, and nothing is answered. That is the design: **active recall**, forcing an answer out of memory before checking it, fixes material for an examination, while an answer printed beside its question converts the exercise back into rereading. Answer each prompt in full, then turn back to the topic that owns it.

This section owns the device alone: the prompt list, a map of the errors that decide marks, and one frame for an unseen question. No multiple, threshold, rate, interval, dose or session count is printed, because an answer beside its question destroys the exercise. Where a prompt reaches a doctrine this chapter does not teach, it names the owner: the statutory provisions to the Mental health legislation and forensic psychiatry notes, the catatonia entity to the Other psychotic disorders, delusional syndromes and catatonia notes, and the algorithms and resistance ladder to the Mood disorders and Schizophrenia notes.

Retrieve	Route	Parameter
THE DEVICE THIS SECTION RUNS ON, NEVER REREAD	WHAT AN INDICATION NEVER SUPPLIES	NAMED BEFORE ANY TRADE IS STATED

26.1 How to use this list

Work the four groups in order; they follow the chapter's sequence: decide whether to treat, how to deliver it, live with what it does, then step outside the induced seizure. Answer aloud or on paper before opening anything.

RULE **Retrieve first, check second.** An answer-free list loses its value the moment you read an answer before producing one. **Retrieval practice** strengthens a memory trace in a way recognising a stated answer does not, and the two feel identical from the inside, which is why the candidate who revises by rereading empties out on a blank page.

26.2 Two habits that answer most of the list

Two moves produce most of the answers below. The first splits the clinical question from the legal one: whether this presentation earns the treatment is settled clinically, whether it may be given by establishing a **lawful route**, and a strong answer to the first is no answer to the second. The second names the parameter before any trade.

RULE **Name the parameter, then state its own relation.** Many choices in this chapter trade among speed of response, efficacy and cognitive cost, and where they do, the direction and the size of that trade differ by parameter and by placement. Some are not trades among those three at all: the airway, the monitoring and the tourniquet buy safety and information at no cognitive price. The chapter therefore asserts no law covering all of them. Stimulus dose above threshold behaves one way in right unilateral treatment and another in bitemporal; treatment frequency is a different trade again. State the relation for the parameter in front of you and inherit no direction from any other.

26.3 Group one: foundations and indications

Where the treatment came from, how it works, which presentations earn it, and what raises its risk.

1. Narrate the arc from a false premise to a modern procedure: whose hypothesis began it, what produced the first therapeutic convulsions, what ended the chemical era, and who achieved the first electrical induction.
2. State the field's own position on how the treatment works, then give the successive accounts in order, each with the observation it rests on and the residue it leaves.
3. List the presentations that earn the treatment and grade each by its evidence. Say where the Indian and United Kingdom positions on schizophrenia diverge, and what an indication never settles.
4. Give both positions on contraindication with the tradition attached to each, what stands in place of a contraindication list today, and why pregnancy belongs on the risk list.

26.4 Group two: technique and dose

Here the answers are parameters, and a parameter is correct only with its document.

1. Say what drives investigation before a course. Then take the depolarising relaxant: name the mechanisms that bar it, and which way the hyperkalaemic danger moves with time after a denervating insult.
2. Name the members of the bilateral and unilateral classes with each placement's landmark. Then name the stimulus parameters in the order of the charge equation, and why a charge figure alone specifies no dose.
3. Answer the placement comparison properly: what the evidence shows near threshold, what happens to the efficacy difference and separately to the cognitive advantage as the multiple rises, and the qualifier that stops convergence being read as overtaking.
4. Define the seizure threshold as a measured quantity rather than a machine setting, and why the therapeutic multiple is not one number across placements. Then judge a seizure: the criteria in order of weight.
5. State what modification modifies and what it leaves untouched, and name the injuries the unmodified technique produced. Then give the Indian position in the correct register: a statutory bar and not clinical advice, the modified form being the only lawful one, the

provision belonging to the Mental health legislation and forensic psychiatry notes and named there in words.

26.5 Group three: effects, consent and course

The longest group, and the one deciding most marks: where the treatment meets the patient, the law and the calendar at once.

1. Separate the three cognitive phenomena and give the clock each runs on. Say which recovers, which stays contested, and why a recovery finding from a corpus with no instrument for the second is not reassurance.
2. Give the cardiovascular sequence in its fixed order, and what a candidate naming only the second phase has dropped. Then explain why the two mortality estimates differ without either being wrong.
3. Name the lawful routes to giving this treatment in India, with the limit that travels with each, and say what urgency changes and what it never changes. Then state why the Act's emergency-treatment provision cannot authorise this treatment even where the presentation plainly earns it, why **treatment authority** is not **admission authority**, and what gate applies to a minor.
4. For pregnancy, state the comparative safety claim exactly as its source bounds it and what it does not say. For the elderly, what leads. For the adolescent, what comes first.
5. Say what the acute schedule is, what a faster schedule buys, what it costs and where both schedules end up. Then separate the point at which technique is changed from the point at which failure may be declared.
6. State what happens when nothing active follows a successful course, and what is therefore started in every case. Give the taper in the order it is asked for.
7. Give the three statements any drug interaction across a course requires, then sort the common agents into those that raise the threshold, those that lower it and those that do neither.

> **GOOD TO REMEMBER**
>
> Do not run this list once, the night before. Retrieval strengthens memory most when spaced, so sweep the four groups now, mark the prompts you missed, and return to those after some days.

26.6 Group four: the other neurostimulation therapies

Every modality here is defined by contrast with the induced seizure, so each prompt asks what the device does before what the evidence says.

1. Explain how current reaches cortex without touching the skin, and why the treatment is dosed to stay below a seizure. Then say why parameters quoted without their protocol and indication describe nothing ever trialled.
2. State what a weak constant current does to cortical neurons and what it never does. Give the polarity rule as a first approximation, then why a device classification is not an authorisation.

3. Name the psychiatric targets, and what separates this intervention from an ablative one. Then state what the two pivotal sham-controlled depression trials found.
4. Describe the implanted system and say why it is not a cranial implant, then why an effect appearing as a slope over months cannot be detected by a trial reading its endpoint early.
5. For magnetic seizure therapy, separate what the modelling settles about the induced field and the site of initiation from what remains open in humans about spread.
6. State the single property separating ablation from every stimulation modality, and why the two therefore do not form one family however the syllabus groups them. Name the current procedures with their targets, and give the double Indian statutory constraint in words.

26.7 The five answers this chapter punishes

More marks are lost on this chapter to five wrong answers than to genuine ignorance, because each is a compact, confident sentence that sounds like clinical sense. If anything you produced matches the middle column, reopen that topic first.

AXIS	THE ANSWER THAT LOSES THE MARK	THE ANSWER THAT EARNS IT	WHERE TO REOPEN
UNMODIFIED, THAT IS DIRECT, TREATMENT	Filing a statutory bar under clinical advice, as though the question were which technique is safer	In India it is unlawful, not merely inadvisable, and the modified form is the only lawful one; what remains teachable about the older technique is historical	modified against unmodified treatment, and the Mental health legislation and forensic psychiatry notes
THE EMERGENCY	Reaching for the Act's emergency-treatment provision because the presentation is desperate	The presentation can genuinely earn the treatment while that provision cannot authorise it, because it excludes this treatment by name; a lawful route is established even so, and urgency changes the timetable, never the route	indications, and consent
AUTHORITY	Assuming that because the emergency route is closed, the patient must be admitted first	Treatment authority is not admission authority: a capacitous adult can consent to this treatment with no admission at all	consent and the legal aspects
EFFICACY AND COGNITIVE COST	Carrying one parameter's relation across to another as though the chapter held a single law of stimulation	There is no such law; many choices trade among speed, efficacy and cognitive cost, the direction and size differing by parameter and by placement, and several are not trades among those three at all	the cross-cutting synthesis, and the item owning each parameter
TREATMENT FREQUENCY	Writing the faster schedule as the stronger one	What the faster schedule buys is speed of response; end-of-course efficacy is equivalent between the schedules, and the faster one reaches response sooner at a higher cognitive price	course, frequency and number of sessions

PITFALL

A sixth error runs underneath all five: quoting a figure away from the document stating it. A device's specification is that device's and not the field's; a guideline's threshold is that guideline's; a formula holds only inside the parameter set it was derived in; and a rate quoted without its comparator reports a hazard the study never established.

IN INDIA

Many answers here are Indian, and several differ from the Western default. The technique is settled by statute rather than preference; the minor's gate is statutory and comes first; the emergency route is closed by name; the national guideline, not a foreign one, governs the parameter answers where the two diverge; and for every device in the final group the Indian record is a risk classification and nothing more.

26.8 The master frame to run on any question

The whole chapter collapses into one procedure, its own spine gathered into five moves, so an unfamiliar question becomes a sequence of decisions rather than a scramble for the nearest rule.

MNEMONIC

SPINE

Settle the indication: does this presentation earn the treatment, and on what evidence? That is answered clinically, and first. **P**rove the route: an indication is never an authority, so establish a lawful route, remembering that urgency changes the timetable and not the route, and that treatment authority is not admission authority. **I**dentify the parameter: before stating any trade, say which is in front of you, because placement, stimulus dose, treatment frequency, pulse width and anaesthetic conduct each carry their own relation. **N**ame the source beside the figure: the device, guideline, trial or formula that produced it. **E**very provision in words: the Act's provisions touching this treatment are named, never renumbered, and belong to the Mental health legislation and forensic psychiatry notes.

TRAP **Answering a question that has not been fully asked.** Which placement is better, does memory loss persist, which schedule is stronger: each is under-specified, and the examiner knows it. The first has no answer until the multiple of threshold is named, the second none until you say which memory and by which instrument, the third none until you say what is bought.

Settle whether the presentation earns it, prove a lawful route before anything is given, name the parameter before you state its trade and inherit no direction from another, keep every figure beside the document that produced it, and name the Act's provisions in words: the rest is detail hanging off those five.

Previously asked

There is no indexed past-question bank for this chapter, so nothing in the table below is a catalogued past question and no board or year is claimed for any of them. Attributing a paper the index does not hold would be a fabrication, so in place of a board of origin each row is marked Representative, and the questions are written in the register an examiner uses. Electroconvulsive therapy is examined heavily and in every form the paper allows, as a long answer on indications, technique and dose, as a short note on a single population or a single

complication, and in the viva on the seizure the candidate has just been asked to describe, while the other neurostimulation therapies are examined mostly as short notes and as the second half of a compare-and-contrast. The list therefore maps the examined spine of this chapter rather than one board's recent record. Read it as a high-yield revision map, not as a set of questions on record.

QUESTION	MARKS	ASKED
Enumerate the indications for electroconvulsive therapy and discuss the strength of the evidence behind each, including its place in the psychiatric emergency.	10	Representative
Describe the pre-electroconvulsive-therapy workup and the anaesthetic conduct of a modified treatment, including the muscle relaxant and the situations in which the usual agent must not be used.	10	Representative
Electrode placement in electroconvulsive therapy. Compare bilateral with right unilateral placement on efficacy and on cognitive cost, stating the dose at which each comparison holds.	10	Representative
Define the seizure threshold, describe how it is measured, and explain how the treatment dose is set against it and judged adequate.	10	Representative
Modified against unmodified, that is direct, electroconvulsive therapy, and the Indian legal position on the direct technique. Short note.	*5*	Representative
Cognitive adverse effects of electroconvulsive therapy. Distinguish anterograde from retrograde amnesia, give the course of each, and discuss whether the retrograde deficit persists.	10	Representative
Complications of electroconvulsive therapy other than the cognitive ones, and their management in the suite and in the recovery room.	10	Representative
Consent for electroconvulsive therapy under the Mental Healthcare Act: the lawful routes, the advance directive, and the position of a minor.	10	Representative

QUESTION	MARKS	ASKED
Electroconvulsive therapy in pregnancy. Short note.	*5*	Representative
Electroconvulsive therapy in the elderly patient. Short note.	*5*	Representative
Discuss the frequency and number of electroconvulsive therapy sessions in an acute course, how response is judged, and what ends the course.	10	Representative
Continuation and maintenance electroconvulsive therapy. Short note.	*5*	Representative
Drug interactions and concurrent medication during a course of electroconvulsive therapy. Short note.	*5*	Representative
Mechanism of action of electroconvulsive therapy. State the principal neurobiological hypotheses, the observations each rests on, and what each fails to explain.	10	Representative
Repetitive transcranial magnetic stimulation: principle, the motor threshold, stimulation parameters, indication and adverse effects.	10	Representative
Transcranial direct current stimulation. Short note.	*5*	Representative
Vagus nerve stimulation in treatment-resistant depression. Short note.	*5*	Representative
Deep brain stimulation in psychiatry: the targets by indication, the evidence from the sham-controlled trials, and the Indian regulatory and legal position.	10	Representative
Psychosurgery: history, the current procedures and their indications, and the ethical and statutory constraints on the operation today.	10	Representative
Magnetic seizure therapy. Short note.	*5*	Representative

The shape of these questions is worth reading before their content. A ten-mark question that opens with enumerate, describe or compare wants a structured map rather than a definition, and the marks are won on the discriminations this chapter turns on: the difference between an indication and a lawful route, the difference between a definition and a permission, the difference between speed of response and efficacy, and the difference between a device classification and an indication-specific authorisation. A short note carries fewer marks and the same demand for the exact figure with the document that states it, because a device specification is that device's, a guideline threshold is that guideline's, a formula holds only inside the parameter set it was derived in, and a trial's parameters travel as a tuple with their protocol and their indication. Two habits earn marks across the whole list. Say which parameter is in front of you before stating any trade, since placement, stimulus dose, treatment frequency, pulse width and anaesthetic conduct each carry their own relation and none inherits another's. And name the Act's provisions in words, because a candidate who reprints a section number he has not read is doing the one thing this chapter is examined to prevent.

The quick review

One table, read down the left column and cover the right. Every line is taught in full somewhere above and nothing here is new: each is that topic's single must-hold line, so a line you cannot recover is a topic to reread rather than a fact to memorise here. Four things are load-bearing across the chapter. Every figure travels with the document that produced it: a specification, a threshold, a formula and a trial's parameters hold only inside the protocol they came from. There is no chapter-wide law about efficacy and cognitive cost: the direction and the size of any such trade differ by parameter and by placement, and several choices are not trades among those three at all, airway management and monitoring being done because they are correct. An indication is never an authority, so a lawful route is established separately and urgency changes the timetable, never the route. The Mental Healthcare Act's provisions are named in words throughout and never renumbered here. Where the Indian guideline and a foreign college guideline differ, both are printed with their body and their year.

ASK	HOLD
ELECTROCONVULSIVE THERAPY: FOUNDATIONS AND INDICATIONS	
MEDUNA, AND THE CHEMICAL ERA	An inverse relationship between seizures and schizophrenia, from a lack of glial cells in schizophrenia against an overgrowth in epilepsy: the founding premise, and it did not survive. Camphor in oil, then cardiazol; the era ended not on efficacy but on the unpredictable interval between injection and seizure.

ASK	HOLD
CERLETTI AND BINI, AND THE DECLINE	First electrical induction in a psychotic patient, Rome, April 1938, reported August 1939 at Copenhagen; the Central Institute of Psychiatry, Ranchi, followed, cardiazol in 1938 and electroconvulsive therapy in 1943. The mid-century fall was part earned, by regressive treatment to a deliberate acute brain syndrome, and part not, running well past those abuses.
THE MECHANISM: REGISTER AND HYPOTHESIS	Nobody has established how it works and no single mechanism should be assumed, so naming the anticonvulsant hypothesis as the mechanism contradicts the literature; low-dose right unilateral seizures lacked antidepressant efficacy, so seizure induction may be necessary but insufficient. That hypothesis is a postictal prefrontal inhibitory surge which terminates the seizure and enhances GABAergic function, and it does not cover efficacy outside depression.
THE TWO BIOMARKER ACCOUNTS	Neuroendocrine: a transient adrenocorticotrophic hormone, cortisol and prolactin rise, back to baseline in hours, so evidence of delivery rather than mechanism. Neurotrophic: hippocampal volume rises with session number, but greater enlargement went with worse outcome and volumes returned by six months, so not a biomarker of response.
DEPRESSION: INDICATION, EFFECT SIZES, SPEED	First line for a severe episode with psychotic features, catatonia, high suicide risk, or food or fluid refusal; second line where it has not responded to psychotherapy or medication. Effect sizes minus 0.91 against simulated treatment and minus 0.80 against pharmacotherapy, the negative sign being the direction of benefit. Suicidality fell to zero in 38.2 per cent after three treatments and 80.9 per cent by the end, so consider it earlier than its last-resort position.
CATATONIA, MANIA, SCHIZOPHRENIA: TRUE STRENGTH	Catatonia: response 80 to 100 per cent, including after benzodiazepines have failed, but on retrospective observational studies, the strongest indication on the weakest design. Mania: remission or marked improvement in 80 per cent. Schizophrenia: moderate-quality Cochrane evidence added to standard care, risk ratio 2.06; clozapine-resistant response 53.6 against 25.4 per cent, remission 13.3 against 3.7, so response is common and remission is not.
THE EMERGENCY, AND THE AUTHORITY WITHHELD	The treatment can be clinically indicated in an emergency, and the Act's emergency-treatment provision cannot authorise it, because that provision expressly excludes electroconvulsive therapy from the treatment it permits. Both are true: a lawful route is established separately, and no emergency doctrine supplies the authority the Act withholds.

ASK	HOLD
CONTRAINDICATION: TWO POSITIONS WITH THEIR DATES	Position one, the modern task-force position: no absolute contraindications, reaching the page through an anaesthesia review of 2022 citing the consensus statement of 2001. Position two, standard in Indian teaching: raised intracranial pressure with a space-occupying lesion, Maltbie 1980, on Carter's chain of rising cerebral blood flow, oedema and pressure. Both printed, neither graded above the other; routine neuroimaging is not mandatory, bedside fundoscopy is.
ELECTROCONVULSIVE THERAPY: TECHNIQUE AND DOSE	
THE EVALUATION, THE FAST AND THE MORNING LIST	As close to the course as possible, because a workup done weeks earlier certifies a patient who no longer exists; examination-led, the blood panel not mandatory and imaging indication driven. Fasting 2 hours from clear liquids, 6 from milk and light meals, over 8 from fried or fatty food. Cerebral oxygen consumption rises almost 200 per cent, so 100 per cent oxygen from 1 minute before induction until breathing resumes. Antihypertensives, thyroxine, antianginals and bronchodilators are not withheld.
SUCCINYLCHOLINE, WHAT BARS IT, AND RESTIMULATION	Blockade in about 1 minute, MAXIMUM blockade about 2, recovery in 4 to 6; the dose is the anaesthetist's. The under-dosing warning belongs to MIVACURIUM, where trimming to shorten its longer block is the temptation, and a dose trimmed there buys an unmodified convulsion under a patient who looks paralysed. Three mechanisms bar it: potassium efflux from abnormal muscle, deficient plasma cholinesterase, malignant hyperthermia. Prophylactic anticholinergics are not routine, and bradycardia is commoner after a second dose of relaxant, so a restimulation is a cardiac event.
BILATERAL IS A CLASS, AND THE LANDMARKS	Bilateral has three members, bitemporal, bifrontal and left anterior right temporal; bitemporal is one member, not a synonym for the class. Bitemporal: one inch above the outer canthus to external auditory meatus line. Right unilateral, d'Elia: right frontotemporal and 1 inch right of the vertex. Bifrontal: 2 inches above the outer canthus.
WAVEFORM, THE DISPUTED BAND, AND COGNITION	A brief or ultrabrief pulse with a constant current device is strongly recommended. The two GUIDELINE brief-pulse bands agree at 0.5 to 2 ms, and that agreement is bounded to them, a meta-analytic band of 0.5 to 1.5 ms also appearing in the chapter; ultrabrief does not agree at all, 0.2 to 0.4 ms in the Indian guideline of 2023 against 0.25 to 0.3 in the college guideline of 2019. Broader widths carry worse cognitive effects, and 0.3 ms has a cognitive advantage with right unilateral placement though efficacy may be compromised.

ASK	HOLD
THE FOUR PARAMETERS, AND WHY CHARGE IS NOT THE DOSE	Charge is current intensity multiplied by pulse width, frequency and train duration, but charge as the chief measure of dosing is faulty: equal charge from different parameters is a different stimulus. Intensity is held constant during incrementation, train duration is what moves, and lower frequencies are the more efficient.
IMPEDANCE, AND MONITORING THE SEIZURE	Static impedance is read before stimulation, dynamic during it; on one named device static should be above 100 and below 3000 ohms, too low meaning the current found a path that is not the patient, too high that it will struggle to find one. Monitoring is three records of one event, cortical, muscular and cardiac, with the Hamilton cuff isolating a limb from the relaxant.
THE SEIZURE THRESHOLD, AND THE RANGE	The lowest electrical dose eliciting seizure activity, electroencephalographic and motor. Found only by producing a response, not transferable across pulse widths, and a floor rather than a treatment, which is why the prescription is a multiple of it. A 12-fold range across 52 titrated patients, 40-fold across the wider literature: neither is the number.
FOUR DOSING METHODS, AND THE FORMULA'S ERROR	Stimulus titration in regular practice; formula-based methods guide its first dose rather than substitute for it; fixed high dosing for serious medical comorbidity. Half-age formula: half the chronological age as the percentage of charge on one device family, the device clause being part of the formula. Estimates averaged 18 per cent above the empirical THRESHOLD, which is the authors' own summary figure and sits outside the quoted passage, and matched the titrated TREATMENT level in only 14.7 per cent. Two benchmarks, not two directions of one error.
THE MULTIPLE DIFFERS BY PLACEMENT, AND THE THRESHOLD RISES	Right unilateral dose-response is steep, needing markedly suprathreshold dosing, while bilateral efficacy arrives marginally above threshold. The right unilateral brief-pulse cell is disputed, 4 to 6 times threshold in the Indian guideline of 2023 against five to six in the college guideline of 2019, and averaging invents a figure neither states. The threshold rises across a course, about 47 per cent on average but in only 56 per cent of patients.
RESTIMULATION BY KIND, AND THE ADEQUATE SEIZURE	A missed seizure is no seizure at all: nothing motor or electroencephalographic 20 seconds after the stimulus, restimulate at an increased dose. An inadequate seizure happened but was not good enough, and the delay is disputed, 45 seconds in the Indian guideline of 2023 against 60 to 90 in the college guideline of 2019. Quality matters more than duration: the earlier 15 and 25 second definition is of little prognostic benefit, so the question is demoted, not re-answered.

ASK	HOLD
BILATERAL AGAINST RIGHT UNILATERAL, BY MULTIPLE	As a ranking the comparison is memorable and clinically false; as a function of the multiple of threshold it is correct. Right unilateral 17 per cent at a low dose against 43 at 2.5 times threshold, while bilateral was 65 against 63; at a high multiple both reached 65 per cent. They converge toward one another, with no superiority of right unilateral established anywhere.
WHAT THE HIGHER MULTIPLE COSTS, AND ITS LIMIT	Some, but not all, of the cognitive advantage is lost when right unilateral is dosed to equal bitemporal efficacy: erosion, not abolition. Two domains survive, reorientation time and retrograde autobiographical memory. The unilateral efficacy evidence exists only for depression, so carrying the argument to schizophrenia or mania exceeds it.
UNMODIFIED, THAT IS DIRECT, TREATMENT: THE INDIAN POSITION	The statute prohibits electroconvulsive therapy given without muscle relaxants and without anaesthesia, so the modified form is the only lawful form and the direct technique is unlawful, not merely inadvisable. No degree of urgency turns it into something a clinician is entitled to weigh. The provision is named in words and never renumbered. Filing that statutory bar under clinical advice, as though the question were which technique is safer, is one of the five answers this chapter punishes.
WHAT MODIFICATION MODIFIES, AND ITS ONE GAP	Three steps: the motor seizure injures muscles, joints, teeth and bones; it is irrelevant to the therapeutic action; so a relaxant given after the anaesthesia abolishes it. The middle step carries the argument, and the cerebral seizure is untouched. The masseter is stimulated directly rather than through the junction the relaxant blocks, which is why bite blocks and jaw immobilisation still matter.
ELECTROCONVULSIVE THERAPY: EFFECTS, CONSENT AND COURSE	
THREE PHENOMENA ON THREE CLOCKS	Minutes for postictal confusion, orientation back within 5 to 45 minutes; days to weeks for anterograde amnesia; months, and contested, for retrograde. One clock never tells the time on another. The anterograde deficit is significant at 0 to 3 days, recovered by 15, and domain-specific rather than global.
RETROGRADE AMNESIA, AND WHAT STAYS CONTESTED	Hard to measure objectively, so a normal bedside screen does not exclude it; a temporal gradient, personal events within about 6 months rather than the distant past. Brief pulse, unilateral placement and titration each reduce autobiographical loss. Persistence stays contested: standardised mean difference 0.55 against controls, unchanged at 6 to 12 months, while the recovery meta-analysis pooled no standardised retrograde tests. At least one third of patients report persistent loss, so subjective assessment counts alongside objective.

ASK	HOLD
THE CARDIOVASCULAR SEQUENCE, AND THE PROLONGED SEIZURE	Parasympathetic first, 10 to 15 seconds of vagal outflow in the tonic phase with bradycardia and occasional asystole, then the sympathetic surge in the clonic phase, peaking at 1 minute. About 2 per cent of patients have a major adverse cardiac event, roughly 1 per 200 treatments. Prolonged means beyond 180 seconds while termination can be attempted beyond 120: a definition and a permission, and collapsing them loses the mark.
POSTICTAL AGITATION, THE TARDIVE SEIZURE AND THE JAW	Agitation: 13.9 per cent at patient level, 18.0 once studies at high risk of bias are removed, which is the authors' recommended figure, with no validated predictor among 7 factors. A tardive seizure comes within hours, after recovery, and may be non-motor. Temporomandibular dislocation happens under modification because those muscles are stimulated directly.
WHAT THE ACT MEANS BY INFORMED CONSENT	Consent for a specific intervention, without force, undue influence, fraud, threat, mistake or misrepresentation, after adequate information including risks, benefits and alternatives, in a language and manner the person understands. It is intervention-specific, so a general admission consent is not consent to this, and the alternatives limb is the one most often dropped.
TREATMENT AUTHORITY IS NOT ADMISSION AUTHORITY	The second half of the emergency error, and the more often lost. Assuming that because the emergency-treatment provision is closed the patient must first be admitted inverts the position: a capacitous adult can consent to this treatment with no admission at all. Urgency changes the timetable, never the route.
THE LAWFUL ROUTES, AND THE ADVANCE DIRECTIVE	His own consent first, on a presumption of capacity that a decision others think wrong does not rebut. Where capacity is absent: the advance directive first, then his own consent supported by his nominated representative, and only where he needs nearly total support may that representative temporarily consent. A valid directive is a duty on the treating psychiatrist, with no bedside-override, urgency or senior-clinician limb, so the route out of a refusal is an application to the Board.
THE MINOR'S GATE, AND RE-CONSENT	Below eighteen there is no capacity to consent, the legal guardian being the nominated representative unless the Board orders otherwise. Two limbs bind, the guardian's informed consent and prior permission of the concerned Board, with no clinical discretion; assent and two independent psychiatrists are professional additions, not statutory. Re-consent runs on triggers, not intervals: capacity returning, a minor attaining eighteen, and before continuation or maintenance. Refusal and withdrawal carry the best available alternative treatments without prejudice.

ASK	HOLD
PREGNANCY AND THE ELDERLY	Pregnancy: most applicable in the second and third trimesters, with left lateral tilt, routine anticholinergic premedication avoided and cardiotocography around each session. Causation is unsettled, and in the Swedish cohort the comparator decides the answer, premature delivery significant against one control group and not the other. The elderly run the other way: older age predicted remission and response, so preferentially good candidates rather than merely tolerable ones.
TREATMENT FREQUENCY BUYS SPEED, NOT EFFICACY	The acute course runs 2 to 3 sessions per week, spaced evenly. Two double-blind trials and their joint synthesis settle it: end-of-course antidepressant effect was equal, while the thrice-weekly schedule induced more severe memory impairment. The cost belongs to the spacing, not to receiving more treatments. Where rapid improvement is warranted the faster schedule may be preferred, both being equally efficacious, the slower one priced at roughly 4.8 to 6.5 days longer. Writing the faster schedule as the stronger one is one of the five answers this chapter punishes.
THE ACCELERATED SCHEDULE, AND WHAT BUYS IT	In malignant catatonia, bilateral treatments daily or twice daily for 3 to 5 days, then conventional frequencies until resolution, 5 to 20 treatments at expert-consensus grade. What buys the extra cognitive cost is mortality: 51 of 54 treated patients survived against 6 of 14 given antipsychotics and supportive care.
HOW MANY TREATMENTS, AND THE MID-COURSE THRESHOLDS	Not predetermined but based on individual need; the task force gives 6 to 12 sessions for depression. Two thresholds must not be merged: technique changes first, unilateral to bilateral or ultrabrief to brief pulse after four to eight sessions without improvement, while failure is declared later, at a minimum of 8 to 12. The United Kingdom handbook is stricter; both are printed. The endpoint is stable remission, not response.
CONTINUATION, MAINTENANCE AND THE INTERVAL PRINCIPLE	Continuation consolidates the index course, maintenance prevents a new episode, the line drawn at 6 months. Over 24 weeks relapse was 84 per cent on placebo, 60 on nortriptyline and 39 on the combination. The taper runs twice weekly to weekly, fortnightly, monthly, never prefixed. What is managed is cumulative exposure and the interval is how: extend it and minimise the cumulative number of treatments.

ASK	HOLD
CONCURRENT MEDICATION, AND THE THREE MOST EXAMINED	Anticonvulsants raise the threshold; chlorpromazine and clozapine are pro-convulsant; lithium raises the risk of postictal delirium; tricyclics raise cardiac risk. An interaction is never one fact: a direction on the seizure, a consequence, a management step. Benzodiazepines raise the threshold, shorten the seizure and decrease potential efficacy, the third mattering most. Theophylline is stopped 24 hours before in the Indian guideline while a second source gives no interval at all.
OTHER NEUROSTIMULATION THERAPIES	
RTMS: SUB-CONVULSIVE BY DESIGN	A high-voltage discharge drives a coil, producing a time-varying magnetic field of 1 to 2.5 Tesla under about 1 millisecond; parameters are chosen to minimise seizure risk, so a seizure here is an adverse event, not the mechanism. At therapeutic intensity it does not produce the amnestic syndrome that follows an induced generalised seizure, because it induces none, while an unintended seizure can still occur: the absence of the first neither abolishes nor excuses the second.
THE MOTOR THRESHOLD, FREQUENCY, COILS AND TARGETING	Rossini criterion: the lowest intensity, as a percentage of maximum stimulator output, inducing a motor evoked potential in 5 of 10 trials; resting and active thresholds differ, and a threshold read by eye comes out higher, inflating everything derived from it. Protocols divide at 1 Hz, low frequencies reducing excitability and faster ones raising it; the figure-of-eight coil is the most focal, and F3 by head measurement is the most practical targeting.
THE COURSE, THE SEIZURE RISK, AND THE CEILING	20 to 30 sessions over 6 weeks are very likely needed for results consistent with the regulatory trials. Seizure risk 7 per 100,000 sessions pooled and 33 at elevated risk. The most recent international update declined to update the safety tables, keeping one ceiling: the parameters used to induce a seizure deliberately must not be exceeded, usually 100 per cent of output at 25 Hz for up to 10 seconds.
TDCS: ENVELOPE, MECHANISM, POLARITY	A family defined by a parameter envelope rather than a mechanism: 4 mA or less, up to 60 minutes, electrodes 1 to 100 square centimetres, 0 to 10,000 Hz. It works by subthreshold polarisation, shifting membrane potentials without causing action potentials, so no step fires a neuron. Anode excites and cathode inhibits is a simplification: every montage is active at both ends, and electrode size is part of the dose.
THE TUPLE, AND A NEGATIVE TRIAL	In DepressionDC, a constant 2 mA for 30 minutes, 24 sessions within 6 weeks, added to a selective serotonin reuptake inhibitor, anode F3 and cathode F4. The investigators state their result plainly: no antidepressant benefit over sham and no difference in neurocognitive change, so the technique bought neither benefit nor cognitive cost.

ASK	HOLD
INDIA: A CLASSIFICATION IS NOT AN AUTHORISATION	The national risk-based list enumerates 131 devices in the Neurological category with no entry for a repetitive transcranial magnetic stimulation system, while electroconvulsive therapy systems, deep brain stimulators and vagus nerve stimulators are listed. That is classification and nothing more, establishing nothing about product licensing or indication-specific authorisation. The Act restricts psychosurgery but nowhere defines it, so whether an implanted system falls inside that restriction does not arrive from the statute's text.
VNS: THE SYSTEM, THE ROUTE, THE TRIAL	The implanted device whose electrode never enters the brain: a pulse generator in a subcutaneous chest pocket, a helical lead on the left cervical vagus. The vagus there is predominantly afferent, running nucleus tractus solitarius to locus coeruleus and raphe and on to limbic structures. In the acute trial 15.3 per cent responded against 10.0 on sham, not significant; approval rested instead on 12 month continuation results compared against a separate observational study.
DBS: THE TARGETS, AND THE WIDTH OF THE FIGURES	Not a reversible lesion but a circuit therapy that replaces a pattern. Obsessive-compulsive disorder: ventral capsule and ventral striatum and nucleus accumbens; depression: ventral capsule and ventral striatum, subgenual cingulate, medial forebrain bundle. Not the same set. Both sham-controlled depression trials failed their primary endpoints, 20 against 17 and 20 against 14.3 per cent, while obsessive-compulsive response ranges from 10 to 61 per cent, so a rate without its target and settings says nothing.
MAGNETIC SEIZURE THERAPY: SETTLED AND OPEN	Magnetic stimulation at parameters chosen to produce a seizure deliberately, under anaesthesia, demanding electroconvulsive therapy infrastructure. Settled: the induced electric field is much lower and much more focal, the site of initiation more restricted. Open: whether the ictus that follows propagates less widely. The guideline's bridge from focality to tolerability is a belief, not a finding.
PSYCHOSURGERY: THE GROUPS, HISTORY AND ABUSE	Lesioning destroys tissue, stimulation delivers current to tissue that remains intact. Moniz with Almeida Lima, 1936, 20 prefrontal leucotomies by free-hand injection of absolute alcohol, substantial improvement in 14 of 20 and apathy, abulia or personality change in others. Three features made it an abuse: indications so loose it was offered where no case existed, delivery outside the operating theatre, and sometimes without a neurosurgeon. Reform ended in Spiegel and Wycis's stereotactic frame.

ASK	HOLD
PSYCHOSURGERY: CURRENT SET, EVIDENCE, ETHICS	Anterior cingulotomy, anterior capsulotomy, subcaudate tractotomy, limbic leucotomy; since 1996 all three cingulotomy lesions per side are made simultaneously. The caveat is half the headline: ablative approaches are not SHOWN inferior to stimulation, response 50 to 60 per cent, but on largely uncontrolled studies with no direct comparative trials: not shown less effective, shown on weaker designs. Ethically acceptable only with strict respect for autonomy, beneficence, non-maleficence and justice; the named consent failure is therapeutic misconception.

References

Every specific in this chapter is bound to one of the sources below, and the count is the number of claim rows each carried. This chapter was grounded from PRIMARY sources read at origin rather than from any textbook or from the repo corpus, and the reason is specific to this chapter: the corpus carries no current guideline for the technique, no device manual and no regulator label, while this chapter's numbers are electrical parameters, anaesthetic doses, device authorisations and statutory provisions, where a textbook paraphrase is not good enough and a stale or summarised figure changes what a clinician does at the machine. Each of the 436 rows carries the verbatim quote it was read from, together with a receipt naming the issuer, the document, the version or year, the jurisdiction and the retrieval date. Four classes of primary source carry the chapter. Current clinical practice guidelines were read at the guideline text, the Indian Psychiatric Society guideline of 2023 controlling Indian practice questions and the Australian, South Australian, United Kingdom and international bodies printed beside it with their own bodies and years wherever they diverge, the divergences being recorded and not averaged. Regulator records were read at the regulator, meaning device approval and classification records and product labelling, and a classification is reported as a classification, never as an indication-specific authorisation, and never read across from one jurisdiction to another. Trial reports, meta-analyses and reviews were read at the published article rather than at a summary of it, so that a population, a comparator and a confidence interval travel with every effect size. And the statutory rows were read at the bare Act, on India Code and at the Legislative Department text, because a provision is what decides whether a lawful route exists; the Act's provisions touching this treatment are named in words throughout the chapter and never renumbered, their numbering belonging to the Mental health legislation and forensic psychiatry notes. Of the 436 claim rows, 319 are recorded at verified confidence and 117 at partial, the latter authored from established clinical knowledge or from a source read at abstract or at second reporting and checked by the adversarial content pass rather than bound to a single primary quote at origin; 4 rows are carried forward from the Mental health legislation & forensic psychiatry notes with their reuse declared on the row. Where a specific could not be grounded it was declared as a gap in the prose and taught around rather than supplied from memory, which is why several passages state plainly that no figure exists. The must-have-papers cross-check was NOT performed for this Paper III chapter,

because the paper lists do not yet exist in the intel index; for Paper III the topic bank plus the syllabus scaffolding, together with the inbound-promise sweep of the built corpus, is the completeness authority. This table records provenance, not proof: it says where each specific came from, not that the chapter's prose uses it correctly, which the content pass and the ship review test.

SOURCE	CLAIMS CARRIED
Peer-reviewed trial reports, meta-analyses and reviews (read at the published article; verbatim quote and URL on file for each row)	204
Indian Psychiatric Society, Clinical Practice Guidelines for the Use of Electroconvulsive Therapy (Indian Journal of Psychiatry, 2023)	121
SA Health, Government of South Australia, Electroconvulsive Therapy Policy Guideline (read at the numbered policy sections)	23
United States Food and Drug Administration: device approval and exemption records and product labelling (Center for Devices and Radiological Health; Structured Product Labels)	21
The Mental Healthcare Act, 2017 (Act 10 of 2017), bare Act text read at India Code and at the Legislative Department, Ministry of Law and Justice	17
International Federation of Clinical Neurophysiology, with the European Society for Brain Stimulation: safety and application guidelines for magnetic and low-intensity electrical stimulation	15
National Institute for Health and Care Excellence (technology appraisal and depression guidance)	7
Royal Australian and New Zealand College of Psychiatrists, professional practice guidelines for the administration of electroconvulsive therapy (2019)	7
Somatics LLC, Thymatron System IV instruction manual and instructions for use (manufacturer device documentation, not a regulatory statement)	7
British Association for Psychopharmacology, evidence-based consensus guidelines for the management of catatonia (2023)	6
Central Drugs Standard Control Organisation, classification of non-notified medical devices under the Medical Devices Rules 2017	5

SOURCE	CLAIMS CARRIED
National Network of Depression Centers rTMS Task Group with the American Psychiatric Association Council on Research task force (2018)	3

Index

ablative neurosurgery 8
Ablative psychosurgery 147
accelerated schedule 103
active motor threshold 121
active recall 169
adequate seizure 60
admission authority 171
advance directive 91
advance organizer 4
Anterior capsulotomy 149
Anterior cingulotomy 149
Anterograde amnesia 75
anticonvulsant hypothesis 17
Apnoea 87
assent 93
authority question 162
autobiographical memory 75
baseline cognitive assessment 80
bifrontal 46
Bilateral 46
bite block 72
bitemporal 47
brain-derived neurotrophic factor 20
capacity 90
cardiazol 11
centimetre rule 123
circuit therapy 131
classification 139
Closed-loop stimulation 146
cognitive cost 6
Continuation electroconvulsive therapy 108
Continuation pharmacotherapy 107
convergence without established superiority 56
Convulsive therapy 9
current density 127
Current intensity 50
D-02 139
Deep brain stimulation 131
dose is matched to placement 59
dose-response relation 55
Dosing from benchmark 63
duty cycle 139
dynamic impedance 51
Electromagnetic induction 120
epilepsy 10
explantation 135
extrajunctional acetylcholine receptors 41
figure-of-eight coil 122
Fixed high dosing 63
Food refusal 25
foreign clearance or approval 139
Formula-based methods 63
half-age method 63
Hamilton cuff method 45
Headache 86
helical lead 138
high-frequency rTMS 122
Humanitarian Device Exemption, number H050003, decided in 2009 133
implant effect 135
implantable pulse generator 131
inadequate seizure 66
index course 108
indication 22
indication question 162
indication-specific authorisation 139
induced seizure 4
informed consent 89
inter-treatment interval 109
interictal delirium 76
invasiveness 158
jaw immobilisation 72
lawful route 8
left cervical vagus 138
left lateral or pelvic tilt 95
legal guardian 92
lesioning 147
licensing 139
limbic leucotomy 149
locus coeruleus 138
low intensity transcranial electrical stimulation 126
low-frequency rTMS 122
Magnetic resonance guided focused ultrasound 150
Magnetic seizure therapy 142
maintenance electroconvulsive therapy 108
malignant hyperthermia 41
Manual restraint 70
mid-course review 105
missed seizure 66
modality question 162
modified ECT 12
montage 127
motor seizure 67
Motor seizure duration 67
motor threshold 120
Muscle soreness 86
network and connectivity account 21
neuroendocrine hypothesis 18
neuromodulation 142
neuromodulation and neurosurgical grouping 158
nominated representative 92

non-depolarising blockers 43
nucleus tractus solitarius 138
papilloedema 33
parameter question 162
Parasympathetic discharge 82
Patient-reported memory loss 78
plasma cholinesterase 41
Position one 31
Position two 31
Postictal agitation 85
Postictal confusion 75
postictal delirium 98
postictal inhibitory surge 17
postictal suppression 67
pre-ECT evaluation 39
Pre-oxygenation 39
pro-convulsant 113
programming wand 138
prolonged neuromuscular blockade 114
prolonged seizure 82
Prolonged seizures 99
prolonged time to reorientation 76
Pulse frequency 50
pulse generator 131
QT dispersion 83
Radiofrequency thermal ablation 150
receptor-sensitivity account 19
RECOVER 140
regressive ECT 13
relative contraindications 33
remotely supervised at-home tDCS 129
resting motor threshold 121
Retrieval practice 169
Retrograde amnesia 74
return electrode 127
reversibility 158
schizophrenia and epilepsy antagonism hypothesis 10
Seizure modification 72
seizure threshold 60
sham 127
Speed of response 102
stable remission 105
standardised cognitive assessment 111
Static impedance 51
Status epilepticus 85
Step-down 104
Step-down ECT 109
Stereotactic radiosurgery 150
stimulation dose 153
stimulation parameters 121
Stimulus titration 62
stimulus waveform 49
stupor 25
sub-convulsive 120
Subcaudate tractotomy 149
Substituted consent 91
subthreshold neuronal polarisation 126
Succinylcholine 41
Sugammadex 44
supported admission 90
sympathetic surge 82
Symptom-titrated schedule 109
tardive seizure 85
temporal gradient 77
Temporomandibular joint dislocation 87
The neurogenesis hypothesis 19
Therapeutic misconception 151
Theta-burst stimulation 123
thrice-weekly schedule 102
tocodynamometer 95
tocolytics 95
Train duration 51
transcranial alternating current stimulation 145
Transcranial direct current stimulation 125
Transcranial focused ultrasound 145
transcranial random-noise stimulation 145
treatment authority 171
Treatment frequency 101
twice-weekly schedule 102
ultrabrief pulse 79
Under-reporting 71
unintended seizure 124
Unmodified 68
unmodified (direct) 68
VNS Therapy System 137
withhold half 113

PRINTING AND DISTRIBUTION

Notes Next Door (NND), Delhi is the official printing and distribution partner for Weave Sprint. The soft-copy PDFs and the complete print-ready set are always available at weave.clinic/sprint, should you prefer to print the notes yourself. Weave Sprint is open source and free, and as part of Weave's commitment to residents it will stay that way, forever. We do, however, strongly recommend a printed set from Notes Next Door: in our experience, they have been the most reliable printing partner we have worked with.