Interventional Psychiatry: A practical introduction to modern ECT, rTMS, and ketamine antidepressant therapy for established psychiatric providers

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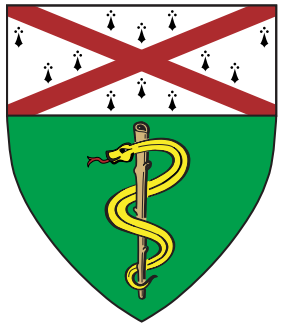


Yale
NewHaven
Health
Yale New Haven
Psychiatric Hospital

Disclosures

- Dr. Kitay receives funding from Janssen Pharmaceuticals for the conduct of clinical trials involving esketamine administered through Yale University.

- Dr. Kitay has also received honoraria from Janssen Pharmaceuticals.

- This presentation will include discussion of off-label use of ketamine.

Program Objectives

• Define “Interventional Psychiatry” (IP) and understand its role in current clinical psychiatric practice. Participants will be able to describe the attributes of an "Interventional Psychiatry Service (IPS)" that may be adapted to various settings of clinical psychiatric care.

• Understand the fundamental mechanistic and technical aspects of electroconvulsive therapy (ECT), repetitive transcranial magnetic stimulation (rTMS) therapy, and ketamine antidepressant therapy. Participants will become familiar with the current evidence base regarding therapeutic efficacy and the risk/benefit profile for each treatment modality towards understanding appropriate indications for referral.

• Describe practical clinical aspects of IP treatments including: pre-procedure counseling/work-up, elements of safe and effective procedure administration, post-procedure evaluation, and the role of the “outpatient psychiatrist” during various phases of treatment.

• Discuss the role of outpatient psychiatrists in mitigating stigma around- and enhancing access to- IP treatments.

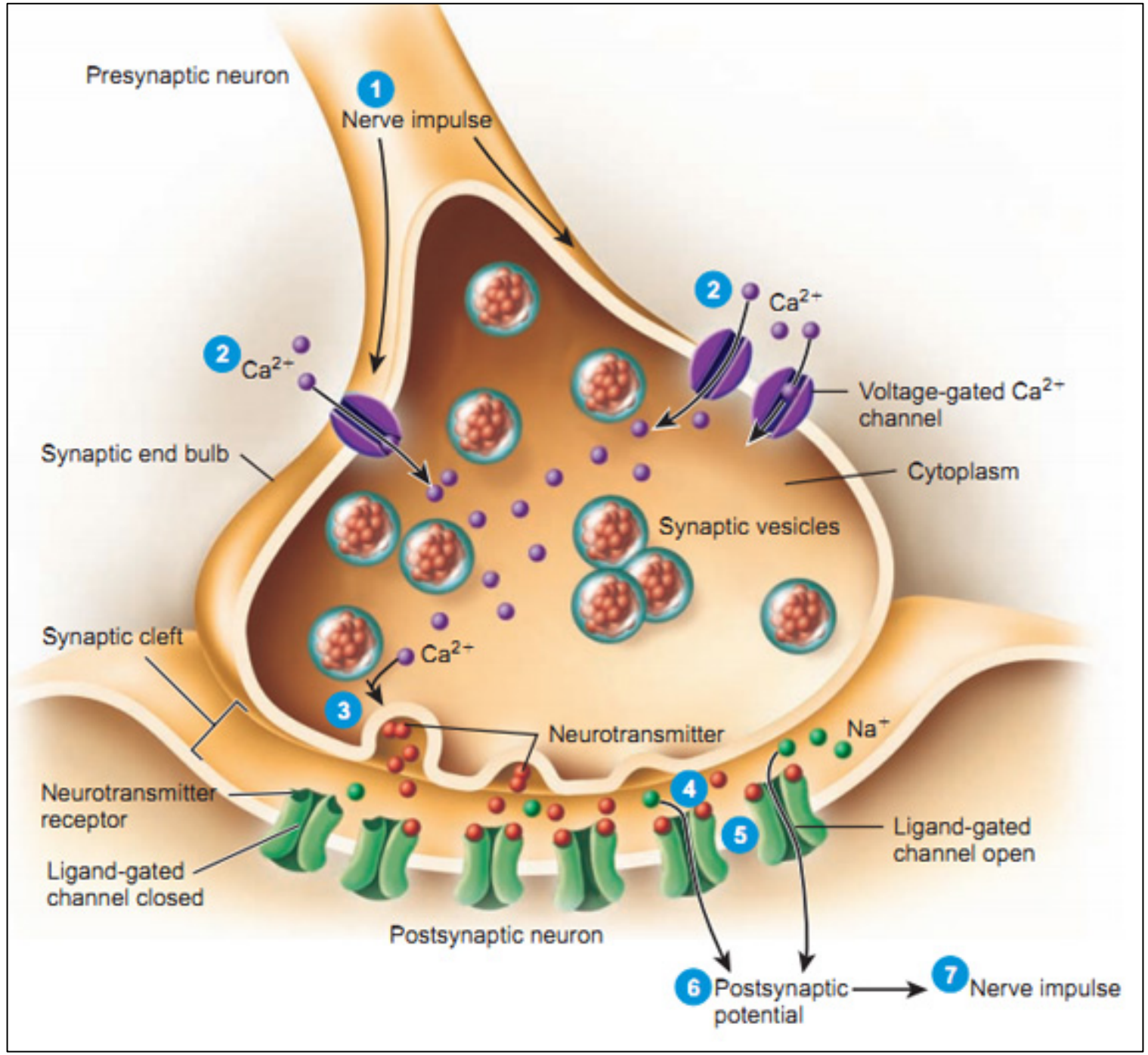
Program Objectives

Through completion of this course, participants will understand how to incorporate these modalities into their treatment planning and develop skills towards:

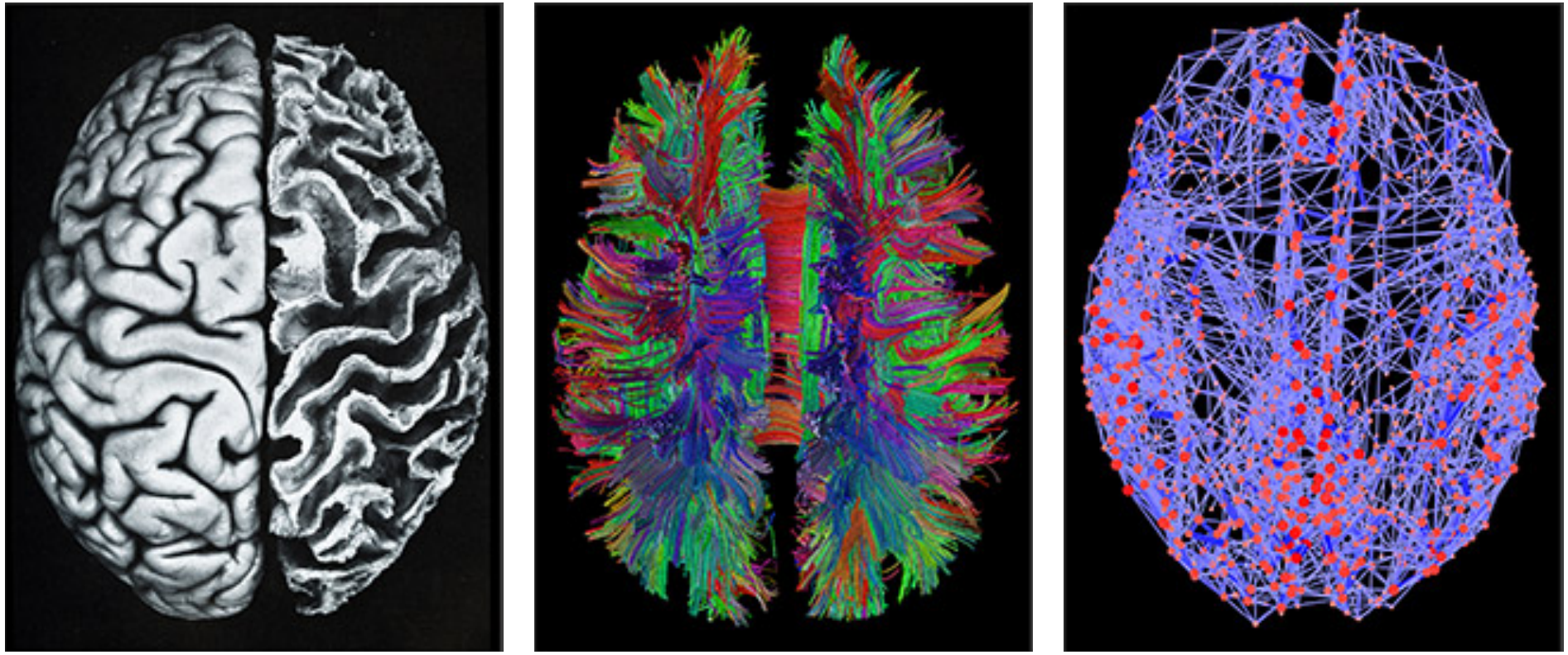
1. Formulating appropriate referrals

2. Providing both accurate and effective pre-treatment counseling in anticipation of referral

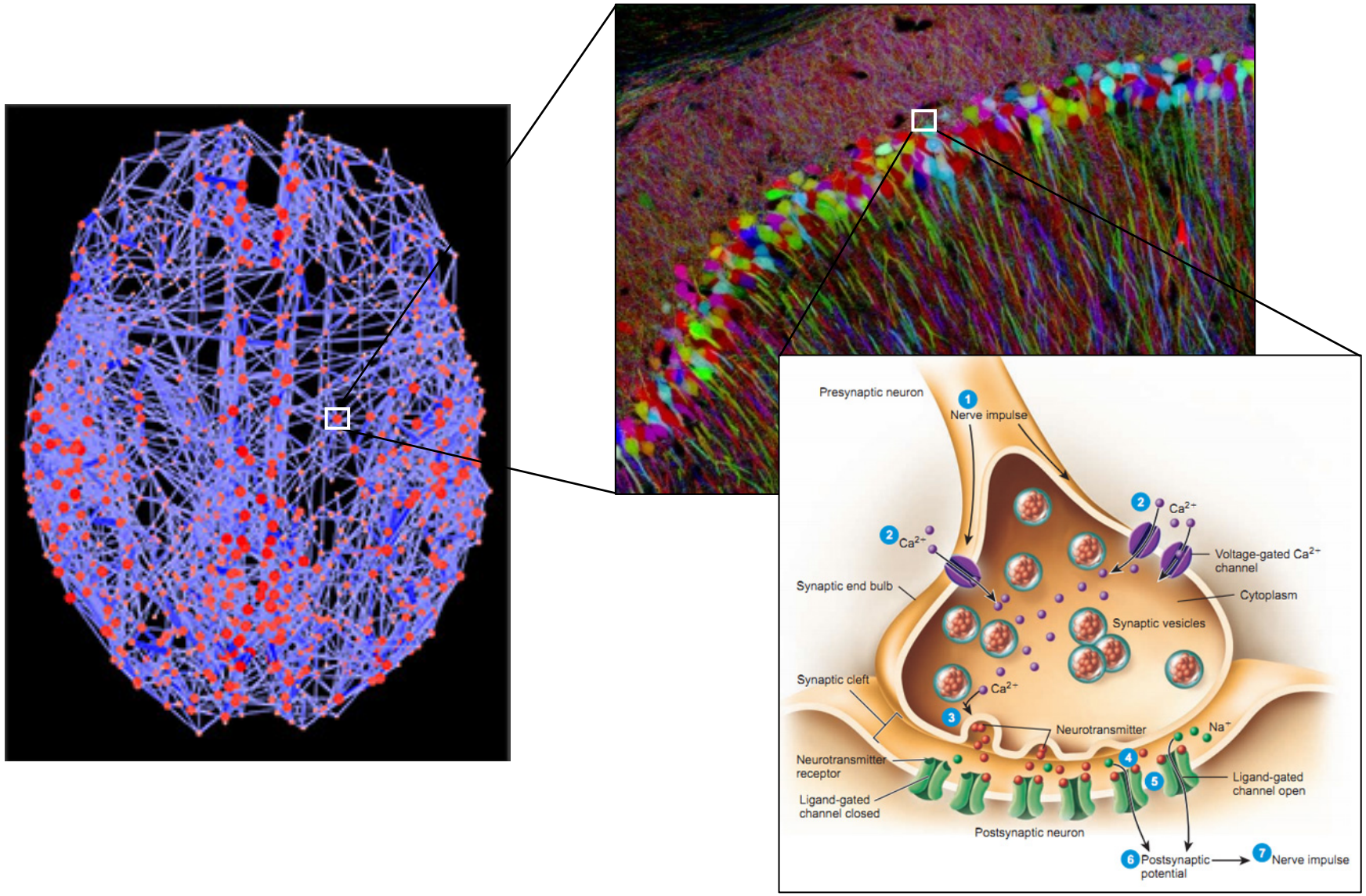
3. Acknowledging and discussing stigma towards enhancing openness to referral


Presynaptic neuron
1
Nerve impulse
2
Ca^{2+}
2
Ca^{2+}
Voltage-gated Ca^{2+} channel
Synaptic end bulb
Cytoplasm
Synaptic vesicles
Synaptic cleft
Ca^{2+}
3
Neurotransmitter
Na^{+}
Neurotransmitter receptor
4
5
Ligand-gated channel open
Ligand-gated channel closed
Postsynaptic neuron
6
Postsynaptic potential
7
Nerve impulse

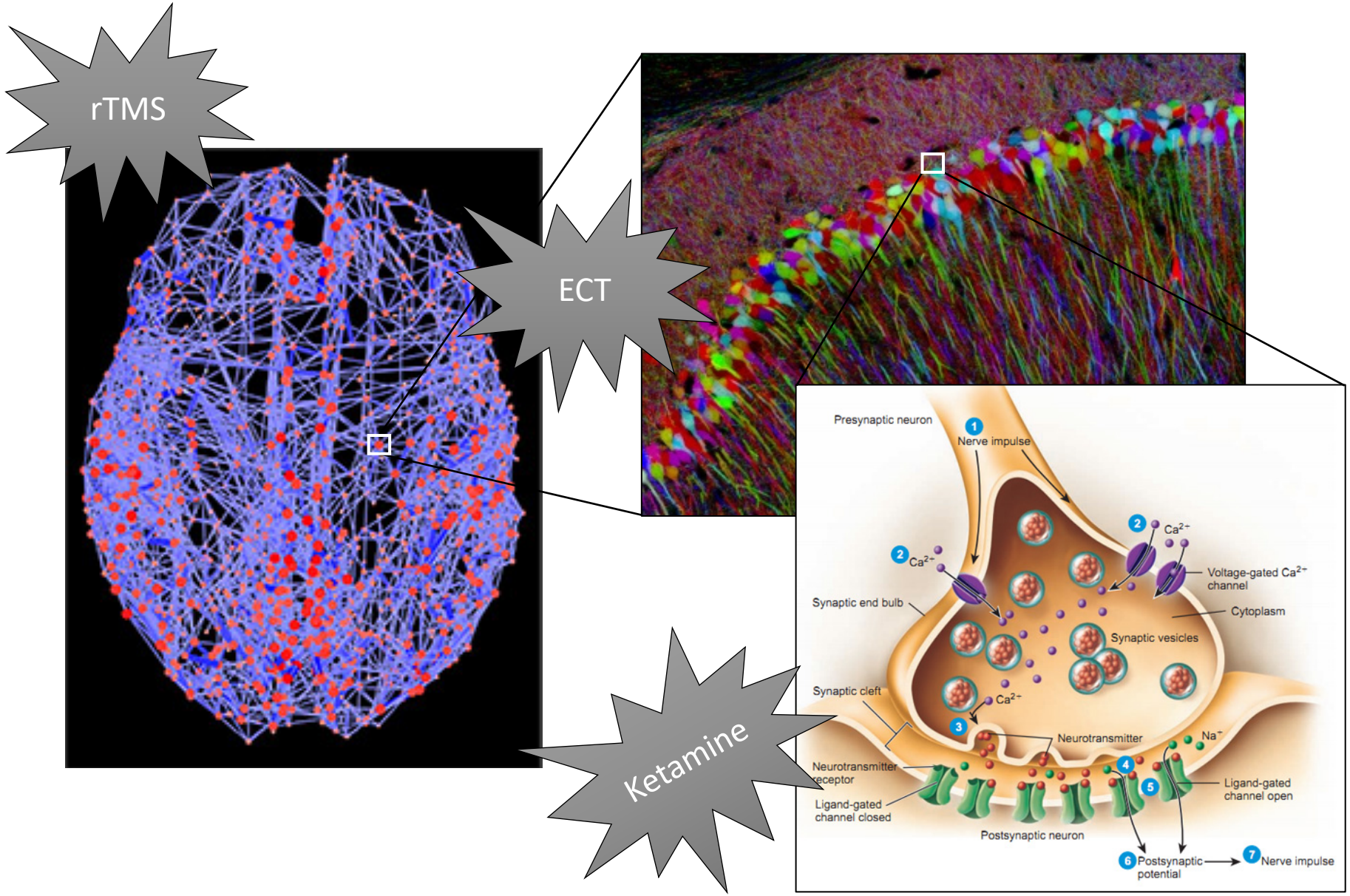
The brain is a complex, *electrochemical network*



The brain is a complex, *electrochemical network*



The brain is a complex, *electrochemical network*



“Interventional Psychiatry” Defined

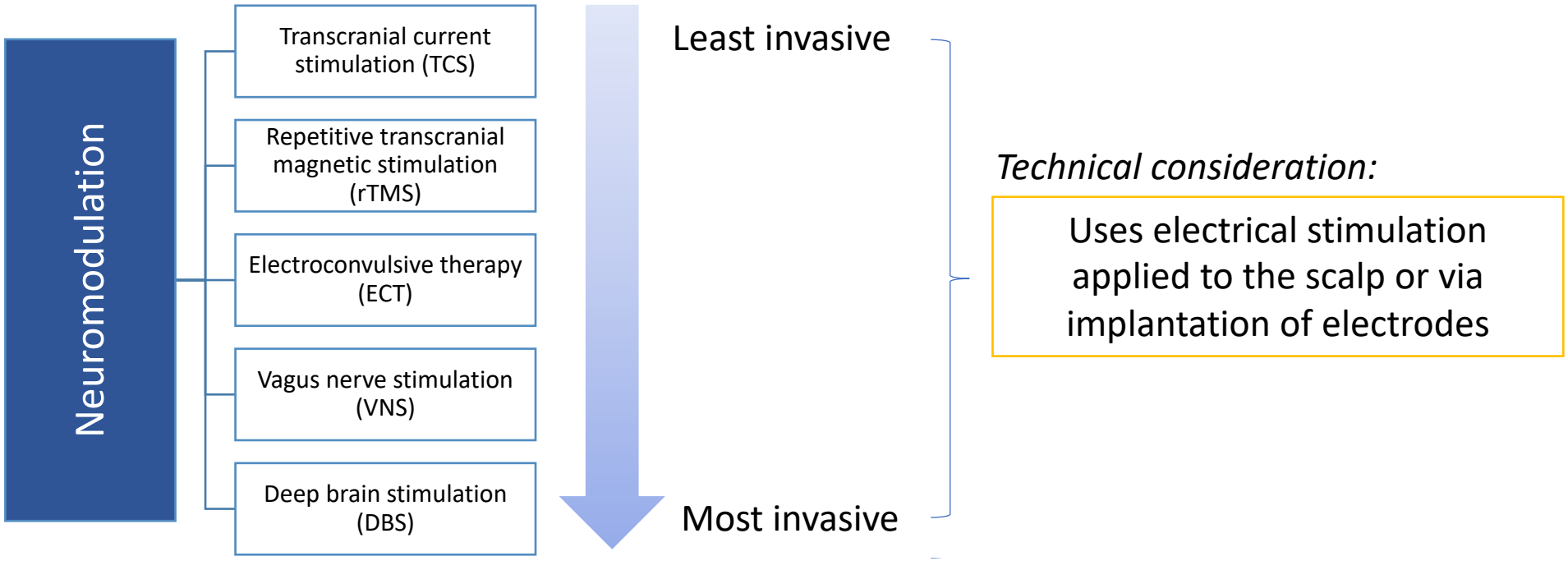
“An emerging subspecialty of general psychiatry that utilizes neurotechnologies to identify dysfunctional brain *circuitry* underlying psychiatric disorders and apply *brain stimulation techniques* to modulate that circuitry.”

Williams NR et al., *J Clin Psy*, 2014

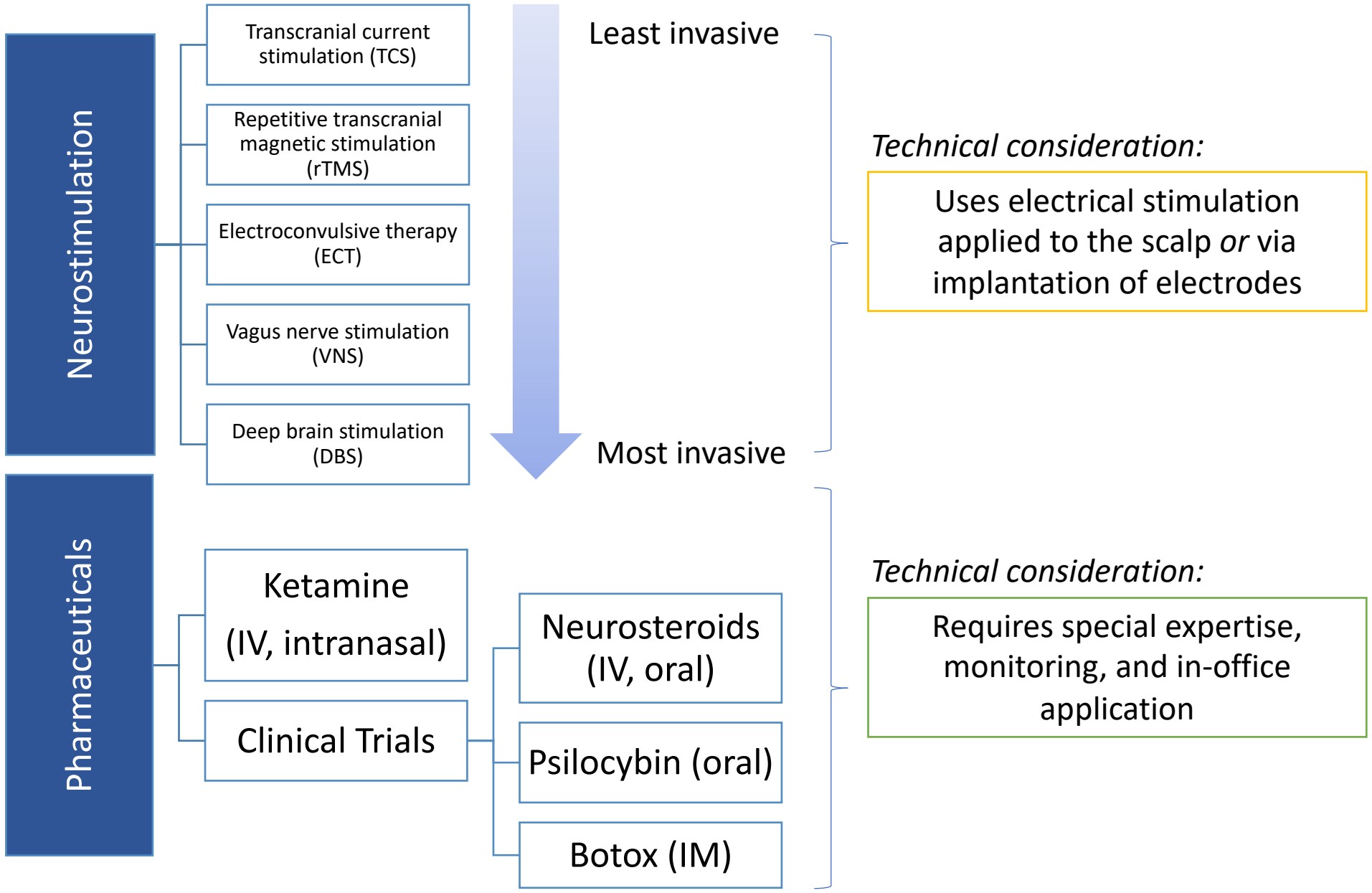
“In addition to neuromodulation, IP requires expertise in pharmacotherapy and new means of delivering of pharmacological treatments.”

Robert Ostroff, MD -- Co-Director, Yale-IPS

Interventional Psychiatry: Treatments



Interventional Psychiatry: Treatments



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Yale-Interventional Psychiatry Service (IPS)

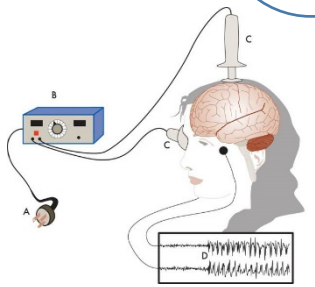
Complex Evaluation

- Outpatient/inpatient psychiatric evaluation
- Medical clearance (primary care)
- Psychometric testing (e.g. mood, cognition scales)
- Triage and informed consent

Neuromodulation

rTMS

ECT



Pharmaceuticals

Ketamine/ Esketamine

Clinical Trials

Yale NewHaven Health
Yale New Haven Psychiatric Hospital

Yale-Interventional Psychiatry Service (IPS)

- Care setting: 6-bed PACU at the Yale Psychiatric Hospital
- ***Mon/Wed/Fri***: ECT service // ***Tue/Thu***: ketamine/esketamine service // ***Mon-Fri***: rTMS service
- ***qMonth:*** Journal Club, Clinical Case Conference, Business Meeting
- Key personnel: attending physician (x4), chief resident (x1), ECT nurses (x4), mental health technicians

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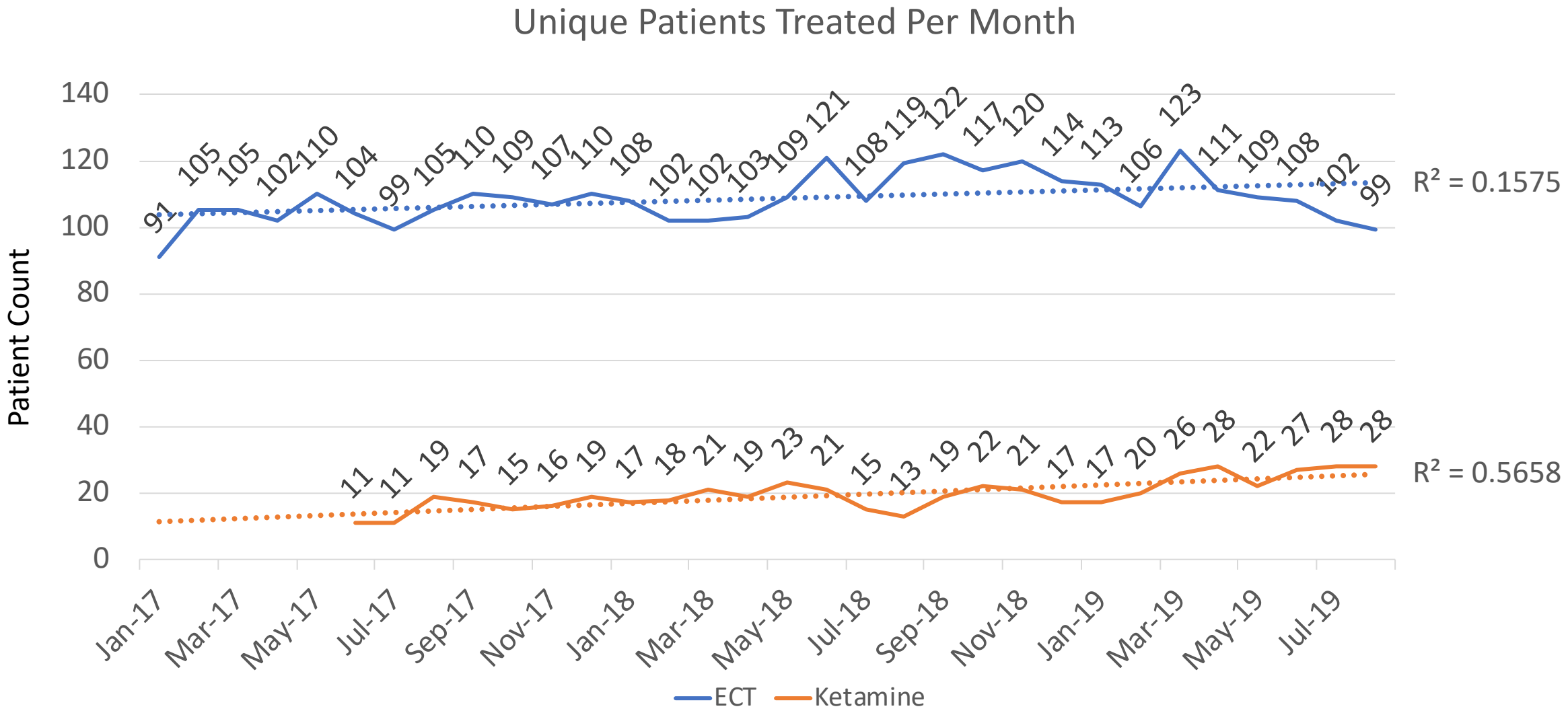
Yale-Interventional Psychiatry Service (IPS)



Yale NewHaven Health
Yale New Haven Psychiatric Hospital

Yale-Interventional Psychiatry Service (IPS)

Fundamental principles of a successful service:

- Establish a *defined* service with clear standards of care
- Provide adequate training for staff to deliver complex medical treatments for challenging patients (without a standardized training pipeline)
- Develop a financial model that supports the service
- Manage the perception of the service to enhance patient access and comfort

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Yale New Haven Psychiatric Hospital

Yale-Interventional Psychiatry Service (IPS)

Logistical wisdom from from a successful service:

- Clear practice standards for medical and ancillary staff
- Clear criteria for each intervention
- Clear standards for *quality* of each procedure
- Uniform assessment suite for each patient
- Clear methods of monitoring change/outcome with defined intervals of assessment (e.g. clinical assessments *and* measurement based care)

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Measurement Based Care:

- Montgomery-Asberg Depression Rating Scale, clinician administered (MADRS)
- Quick Inventory of Depressive Symptomatology, self-report (QIDS-SR16)
- Montreal Cognitive Assessment (MoCA)
- Clinician Administered Dissociative States Scale (CADSS)
- Brief Psychiatric Rating Scale (BPRS), clinician administered
- Bush Francis Catatonia Scale (BFCRS), clinician administered

Interventional Psychiatry: Treatments

Interventional Method	Development	FDA-Approved Uses
Electroconvulsive therapy (ECT)	In use for over 70 years, but with significant recent advances in delivery	"Grandfathered in." APA guidelines indicate MDD, bipolar disorder, schizophrenia, schizoaffective disorder, and catatonia
Transcranial magnetic stimulation (rTMS)	Modern version developed in 1985. Multiple new delivery mechanisms being evaluated	• Acute, treatment resistant unipolar MDD (multiple devices) • OCD* (Brainsway device)
Ketamine/Esketamine therapy	Developed as an anesthetic in the 1960's with good analgesic properties. Antidepressant effects discovered in the early 2000's. Esketamine FDA approved in 03/2019	• Esketamine (Spravato™): *Augmentation to oral antidepressants* for TRD (*not monotherapy*) • Racemic ketamine treatments remain *"off-label." Uses include: MDD/TRD, bipolar depression, PTSD, chronic pain/CRPS*

Adapted from Williams NR, *J Clin Psy*, 2014

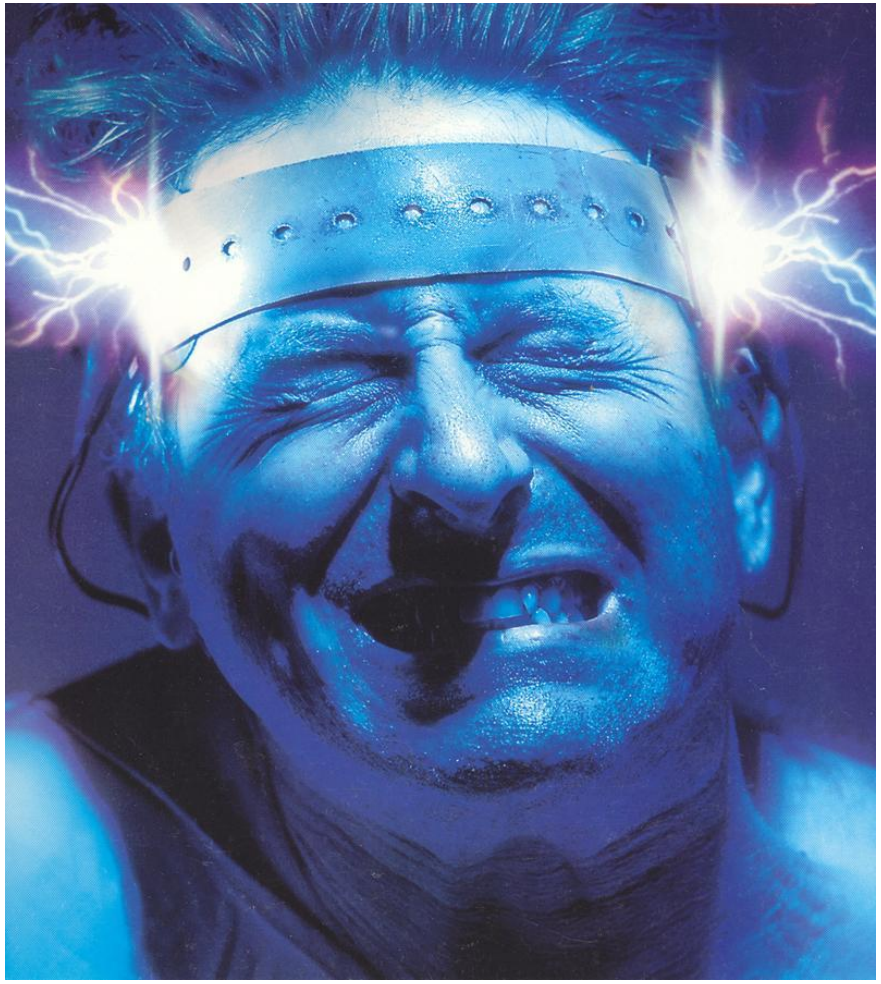
Electroconvulsive Therapy (ECT)

Outline:

- Understanding the stigma, a historical perspective
- Understanding the ECT candidate
  - Diagnoses
  - Demographics
  - Prognostic indicators of response
- Understanding the procedure
  - Goals of the procedure itself
  - A typical treatment course
  - Treatment day
  - Anticipatory side-effects
- Role of the outpatient psychiatrist
  - Practical considerations and formulating candidacy
  - Preparing the patient for consultation/referral
  - Remaining the “primary treater” through an ECT course

Electroconvulsive Therapy (ECT)

The *most effective* treatment in psychiatry today and the most burdened. . .



Electroconvulsive Therapy (ECT) – Brief Historical Perspective

- "Convulsions" and "electricity" have been known to reduce symptoms in people with neurological disorders for centuries:

➢ Hippocrates saw that insane patients showed reduced symptoms after suffering from convulsions brought on by malaria.

➢ There is an account in 47AD, of a physician using an electric eel to cure headaches of the roman emperor Claudius.

➢ 16th Century:
  - A Jesuit missionary wrote of Ethiopians using electricity to "expel devils."
  - Paracelsus, a Swiss physician, used camphor to produce seizures to cure "insanity."

Electroconvulsive Therapy (ECT) – Brief Historical Perspective

- In the 18th Century:
  - Individuals treated with hellebore went into convulsions and coma curing “mania” and “raving madness.”
  - In 1792 John Birch used electric shocks to the head to cure patients.

- 1927: Manfred Sakel develops insulin coma therapy (hypoglycemic shock).

- 1932: Ladislaus von Meduna used camphor to treat schizophrenia. This technique was later modified to us Metrazol as it led to a faster onset of convulsions.

- 1937: Ugo Cerletti and Lucio Bini decided to use electric shock to induce seizure in order to mitigate the side effects of metrazol.

- 1938: The first “electroconvulsive therapy” treatment was tested on a “schizophrenic” (likely catatonic) in Rome. He had a full recovery.

- 1940: The first ECT treatment was given in the United States.

Major Advances in ECT since 1938

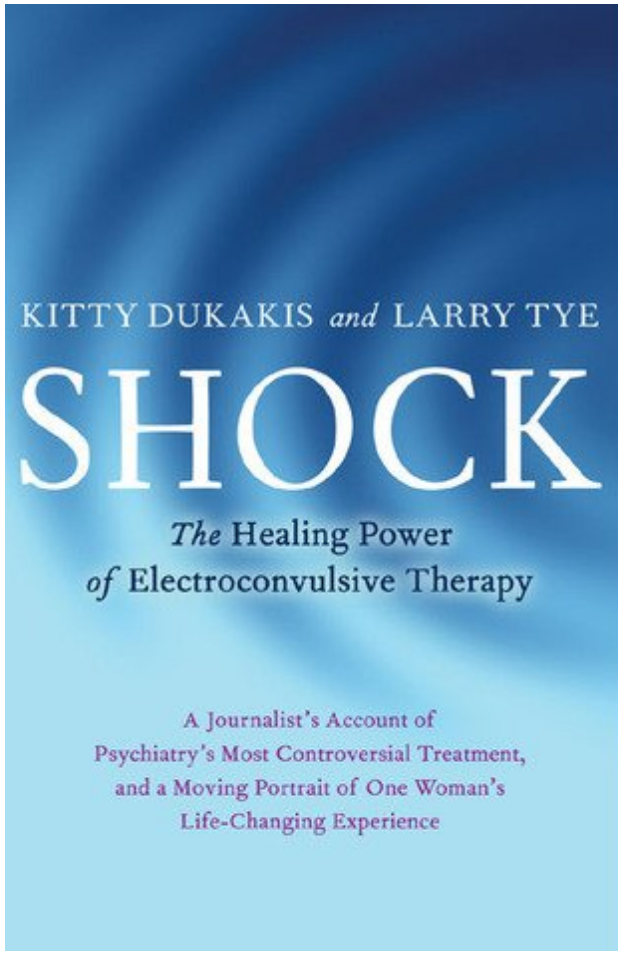
- The introduction of modern anesthesia including neuromuscular blocking agents in the 1960's (e.g. ECT was performed "***unmodified"***)

- The development of devices to minimize electrical energy exposure, allowing electrical dose titration and determination of individual patient's "seizure threshold."

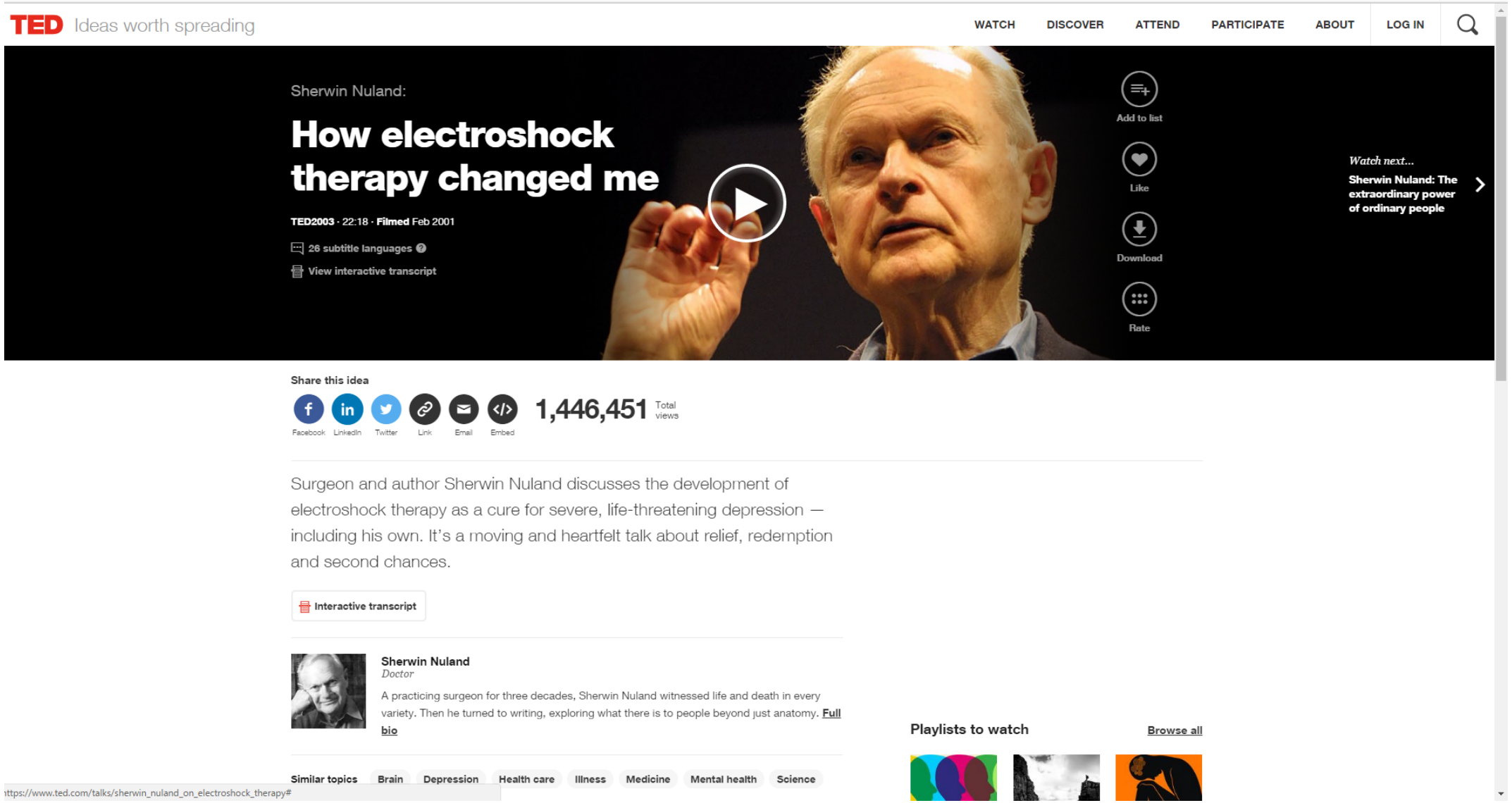
- Advances in lead placement (e.g. site of electrical delivery) to minimize treatment side-effects

- The appreciation of the importance of seizure morphology, not seizure duration, as a determinant of efficacy.

Correcting perception: the new faces of ECT



Correcting perception: the new faces of ECT



Who is the appropriate ECT candidate? *Diagnoses*

- Major depressive disorder, severe *(+/- psychotic features* or *significant suicide risk*)
- Treatment resistant depression (TRD)
  - Failure of at least 2 medication trials for an adequate *time* at an adequate *dose*
  - *Remission rates: 60 – 80% amongst TRD patients!*
- Bipolar disorder, depressed or manic phase
- Treatment refractory psychosis in schizophrenia, schizoaffective disorder
- Catatonia *irrespective* of precipitating cause (e.g. 2/2 a medical or psychiatric condition)
- Parkinson's disease with or without co-morbid mood disorder
- Treatment refractory epilepsy (*status epilepticus*)

Who is the appropriate ECT candidate? *Demographics*

- There is *no age* cutoff! Adolescents through geriatric patients are appropriate for treatment.
- There are *no absolute contraindications* to ECT!
  (…except known increased intracranial pressure)
- Safe during pregnancy.
- Relative risk considerations:
  - Cardiac conditions (arrhythmia, unstable CVD)
  - Neurological disorders (cerebrovascular)
  - Implanted intracranial devices (size, location, material of objects)

Who is the appropriate ECT candidate? *Risk Factors*

CNS:

- Cerebral Infarction
- Cerebral Hemorrhage
- Tumor
- Dementia
- Hydrocephalus
- AV malformation
- Multiple sclerosis
- Seizure Disorder
- Spinal Cord injury

Cardiovascular:

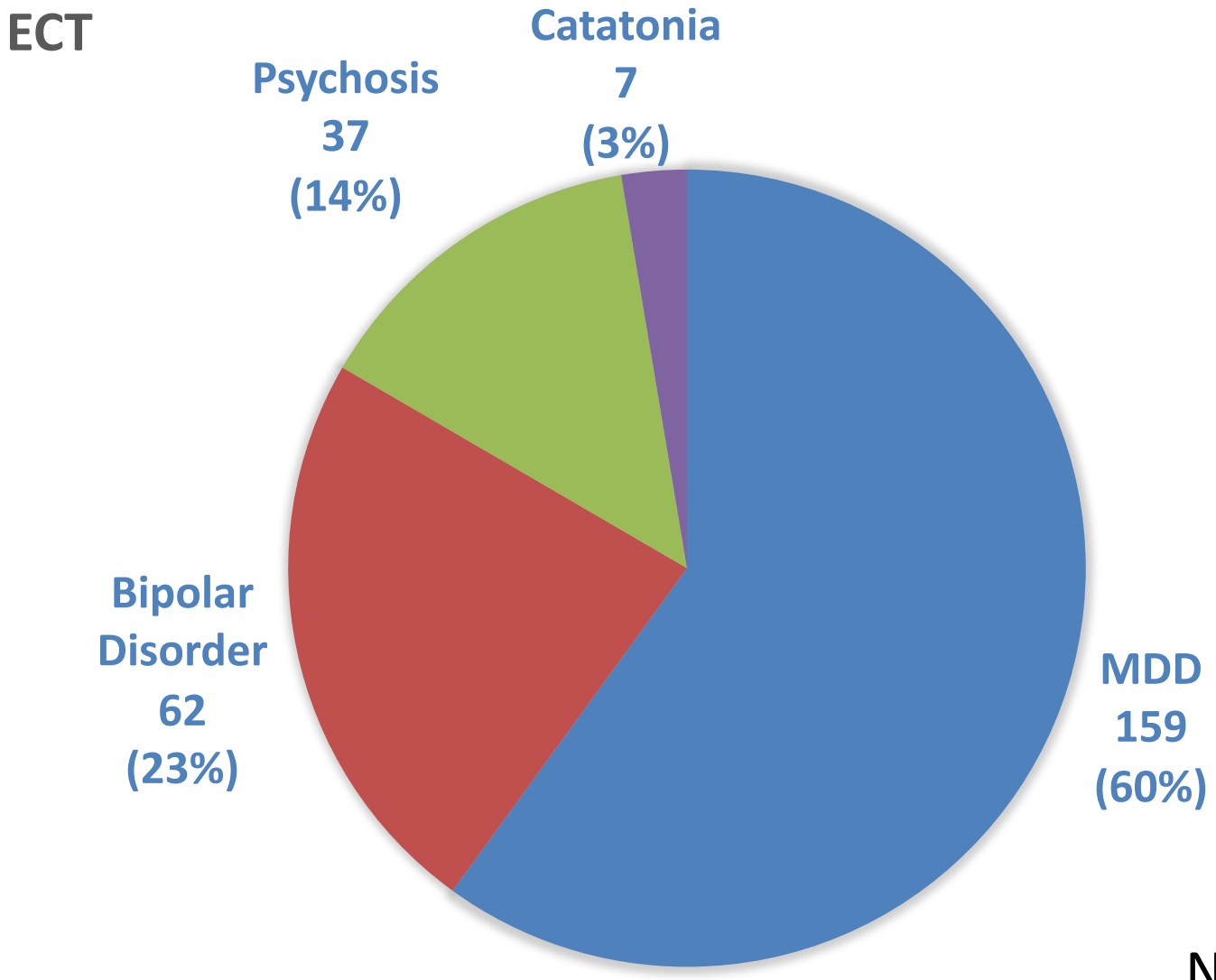
- Coronary ischemia
- Hypertension
- CHF
- Arrhythmia
- Recent MI
- Aneurysm
- Cardiac pacemaker
- Aortic stenosis

Others:

- History of neuroleptic malignant syndrome
- Theophylline
- Increased intracranial pressure
- Aortic Stenosis
- Digitalis toxicity
- Renal dialysis

- Risk:Benefit ratio is important to consider.
- Mortality associated with ineffectively treated depression >>> ECT (mortality rates comparable to outpatient colonoscopy)

Unique patients by diagnosis receiving ECT, Yale-IPS 2018-19



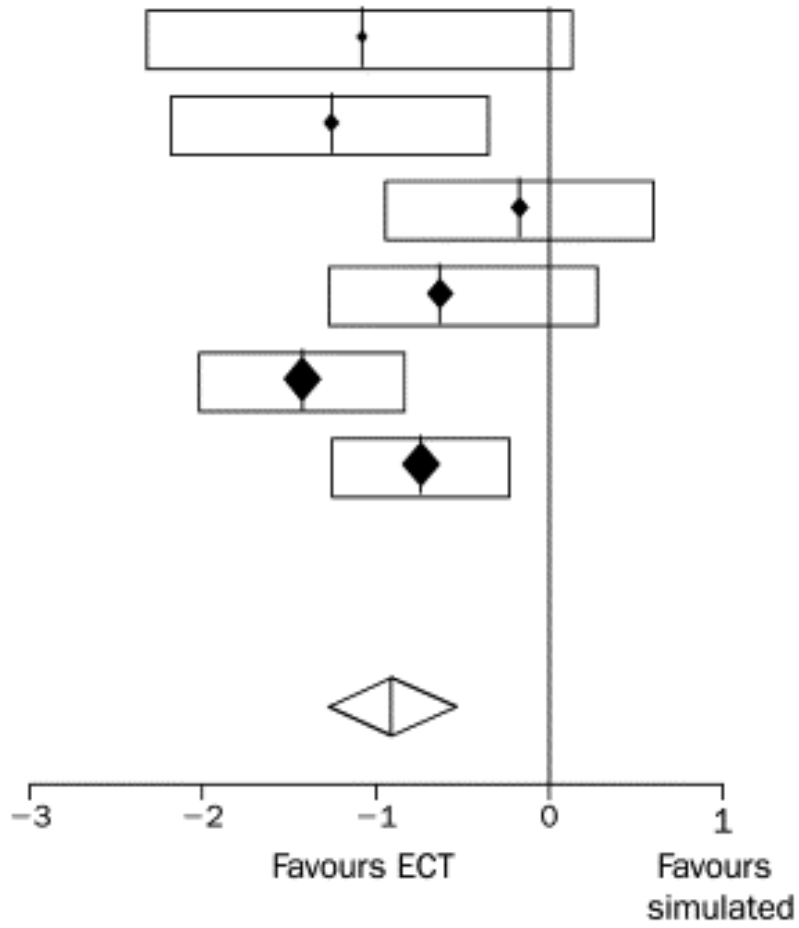
$N_{total} = 265$

ECT for Treatment Refractory Depression

- Gold-standard for TRD
- Response rates are 60-80%
- Remission rates are 50-70%
- Should NOT be option of last resort
- If poor functioning (work, relationships, FTT), recommend discussion of ECT after 1st or 2nd failed antidepressant trial
- Techniques have improved substantially
  - Cognitive side effects still exist but less than previous modalities

ECT for Treatment Refractory Depression

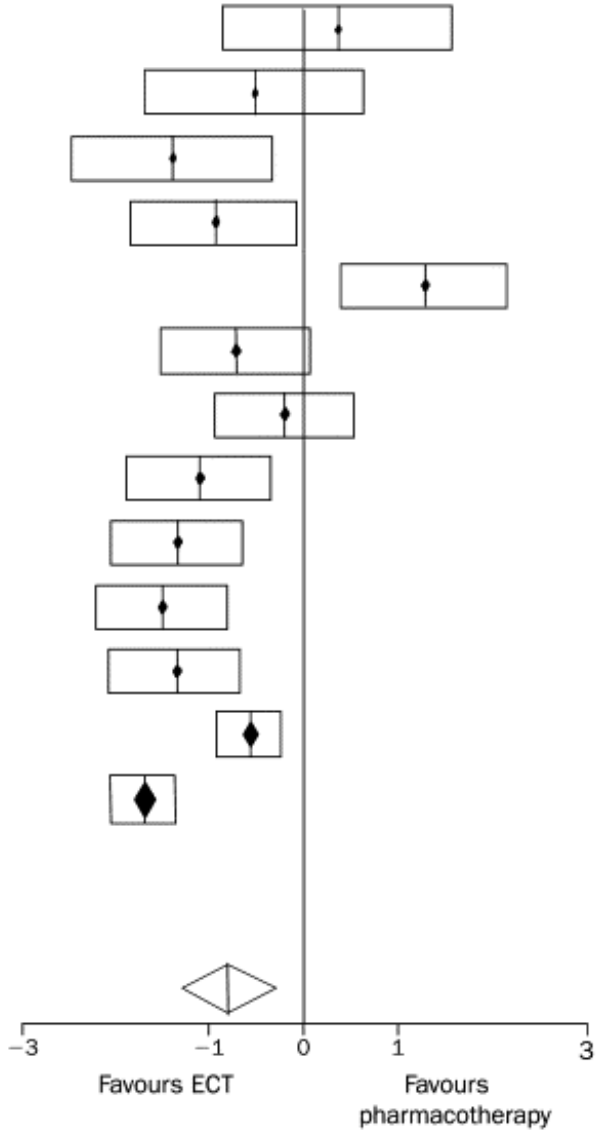
Trial	Number of participants	Standardised effect size (95% CI)
Wilson 1963[10]	12	–1·078 (–2·289 to 0·133)
West 1981[11]	25	–1·255 (–2·170 to –0·341)
Lambourn 1978[15]	40	–0·170 (–0·940 to 0·600)
Freeman 1978[12]	40	–0·629 (–1·264 to 0·006)
Gregory 1985[13]	69	–1·418 (–2·012 to –0·824)
Johnstone 1980[14]	70	–0·739 (–1·253 to –0·224)
Pooled fixed effects		–0·911 (–1·180 to –0·645)
Pooled random effects		–0·908 (–1·270 to –0·537)



Null, Volume 361, Issue 9360, 2003, 799–808

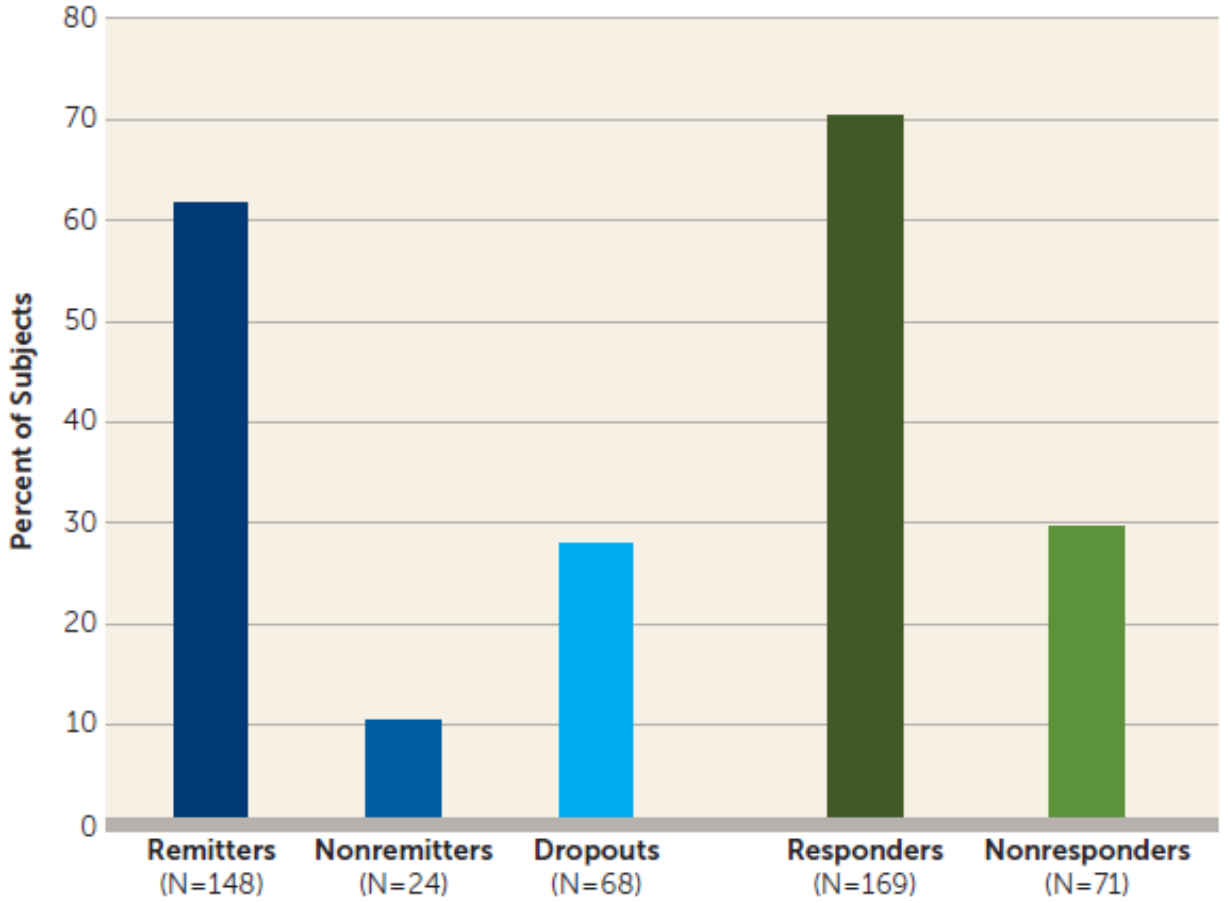
ECT for Treatment Refractory Depression

Trial*	Number of participants	Standardised effect size (95% CI)
Steiner 1978[16]	12	0·369 (–0·840 to 1·578)
Wilson 1963[10]	12	–0·513 (–1·663 to 0·637)
Davidson 1978[17]	19	–1·389 (–2·449 to –0·328)
McDonald 1966[18]	22	–0·930 (–1·813 to –0·047)
Gangadhar 1982[19]	32	1·287 (0·406 to 2·169)
MacSweeney 1975[20]	27	–0·714 (–1·492 to 0·065)
Dinan 1989[21]	30	–0·196 (–0·926 to 0·534)
Janakiramaiah 2000[22]	30	–1·095 (–1·863 to –0·328)
Folkerts 1997[23]	40	–1·336 (–2·032 to –0·640)
Herrington 1974[24]	43	–1·497 (–2·174 to –0·821)
Stanley 1962[25]	47	–1·342 (–2·047 to –0·638)
Medical Research Council 1965[26]	204	–0·559 (–0·883 to –0·234)
Greenblatt 1964[27]	242	–1·683 (–2·020 to –1·346)
Pooled fixed effects		–1·010 (–1·170 to –0·856)
Pooled random effects		–0·802 (–1·290 to –0·289)



ECT for Treatment Refractory Depression

FIGURE 1. Remission, Response, and Dropout in a Study of ECT and Venlafaxine in Geriatric Depression[a]

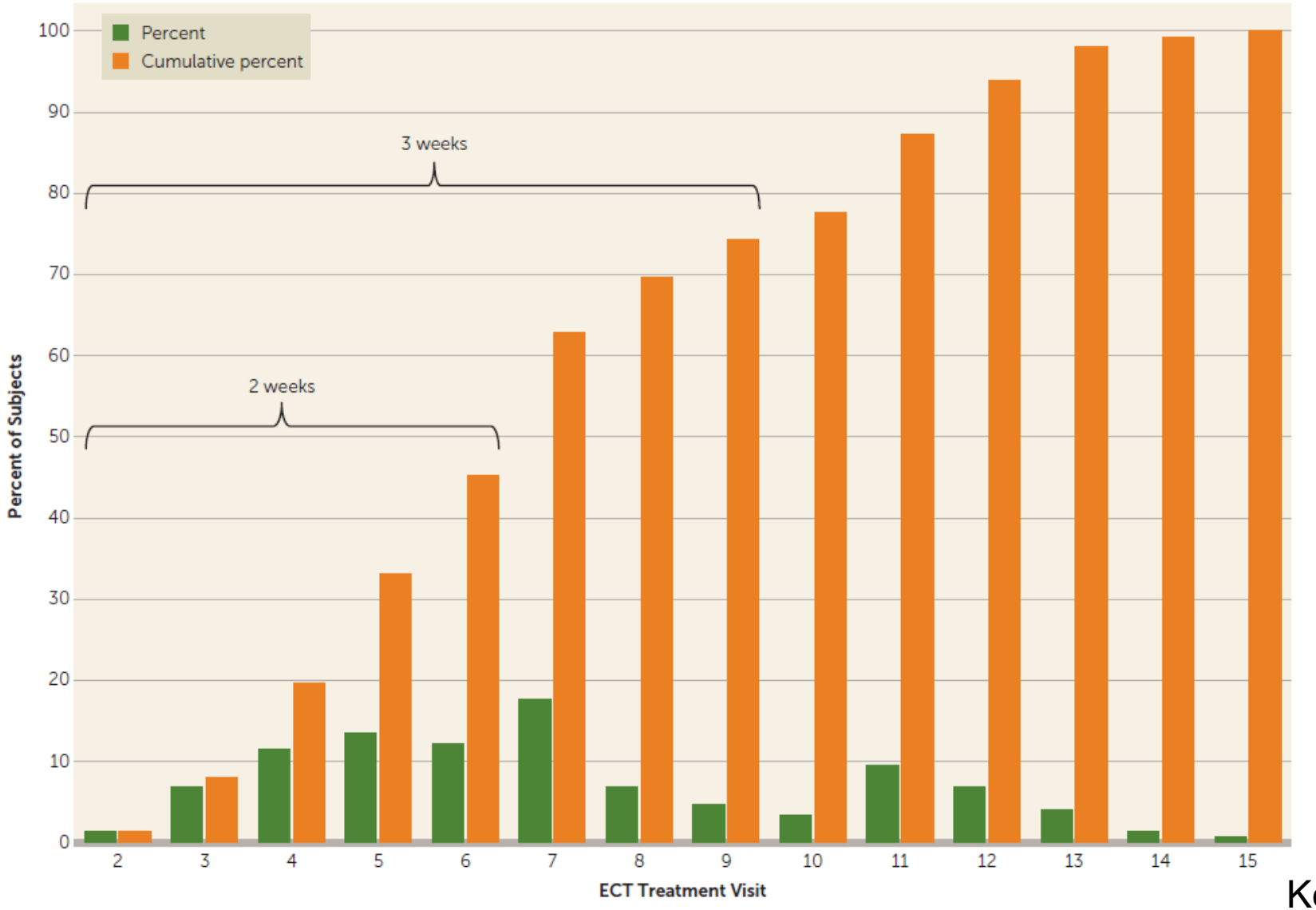


[a] Remission was defined as having a score ≤10 on the 24-item Hamilton Depression Rating Scale (HAM-D) on two consecutive ratings; response was defined as having at least a 50% decrease in HAM-D score from baseline to last assessment.

Kellner C et al., 2016

ECT for Geriatric Depression

FIGURE 3. Speed of Remission Among Remitted Patients (N=148) in a Study of ECT and Venlafaxine in Geriatric Depression



Kellner C et al., 2016

Clinical Predictors of ECT Response for Depression and Mania

Depression Predictor	Description of the association	Supporting evidence	Contrasting evidence
Age	Older patients have higher likelihood of good ECT response	O'Connor et al. [17], Spashett et al. [21], Wesson et al. [22], Tew et al. [23]	Cattan et al. [18], Burke et al. [19], Karlinsky et al. [20]
Sex	Absence of preferential response to ECT according to sex	Kindler et al. [25], De Vreede et al. [26], Okazaki et al. [27], Tominaga et al. [28]	M > F: Andrade et al. [24]
Psychotic depression	Psychotic depression predicts a good response to ECT	Carney et al. [29], O'Leary et al. [31], Petrides et al. [32], Spashett et al. [21]	
Suicide behavior	High severity of suicidal intent predicts a good response to ECT	Kellner et al. [33], Gupta et al. [34]	
Melancholic features	Melancholic depressive features, including psychomotor retardation/agitation, predict a good response to ECT	Ziskind et al. [36], Rasmussen et al. [39],	Fink et al. [37], Sobin et al. [40], O'Leary et al. [31], Tominaga et al. [28]
Chronicity of episode	The longer the duration of the index episode the poorer the response to ECT	Kukopulos et al. [42], Magni et al. [43], Prudic et al. [44], Dombrovski et al. [45], Kho et al. [46]	
Diagnostic subgroup	No difference in rates of response to ECT between BD and UD patients	Grunhaus [47], Daly et al. [48], Abrams and Taylor [49], Black et al. [50]	UD > BD: Medda et al. [51], Perugi et al. [52] BD > UD: Perris and D'Elia [54], Agarkar et al. [55]
Speed of response	Early symptom change after ECT predicted a later good response	Husain et al. [56], Kho et al. [57]	
Mania			
Poor premorbid adjustment	Poor premorbid adjustment predicts lack of response to ECT	Schiele and Schnieder [61], Mukherjee [65]	
Whole brain cortical blood flow	Reduction of whole brain cortical blood flow predicts a good response to ECT	Mukherjee [65]	
Psychotic features	Presence of psychotic features predicts a poor response to ECT	Black et al. [62], Black et al. [63]	
Anger, suspiciousness, irritability	Presence of anger, suspiciousness, irritability predicts a poor ECT response	Schnur et al. [64]	
Greater severity of manic symptoms at baseline	Greater severity at baseline predicts a good response to ECT	Small et al. [59]	Mukherjee [65]

ECT: electroconvulsive therapy; BD: bipolar disorder; UD: unipolar disorder.

Pinna M et al., *Neurosci Letters*, 2018

ECT for Psychosis

Themes of outcomes:

1. Reduction in symptoms (positive > negative)
2. Effective augmentation for neuroleptics (most consistent evidence for clozapine)
3. Reduction in aggression, suicide, self-harm
4. Reduction in hospital re-admission rates
5. Reduction in relapse (psychotic episodes)
6. Side-effects well-tolerated, variable drop-out rates

Electroconvulsive therapy (ECT) in schizophrenia: a review of recent literature

Sohag N. Sanghani[a], Georgios Petrides[a], and Charles H. Kellner[b]

KEY POINTS

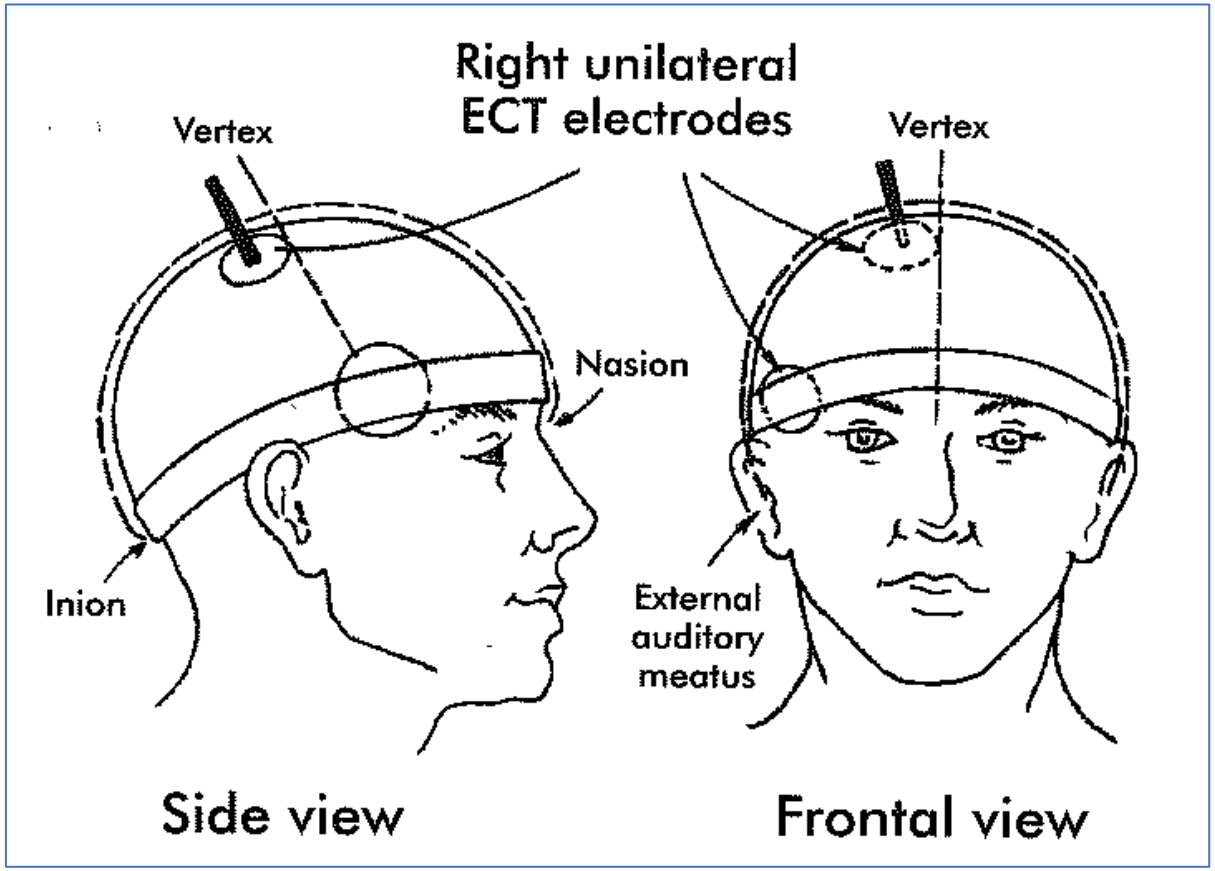
- There is wide variation across countries in the use of ECT for treating schizophrenia.
- Emerging evidence shows that ECT augmentation of antipsychotic medication is highly effective in the treatment of schizophrenia, including medication-resistant schizophrenia.
- Clozapine is a particularly effective antipsychotic medication to combine with ECT
- The cognitive effects of ECT are mostly mild and transient.
- The absolute differences between various ECT electrode placements and stimulus parameters appear to be minor, but further studies are needed.

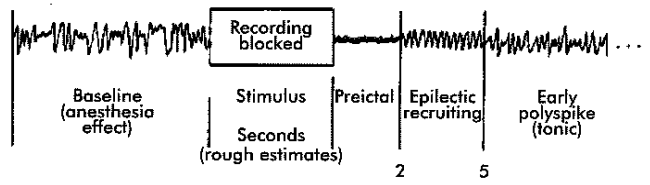
Ward HB et al., *Psychiatry Research*, 2018

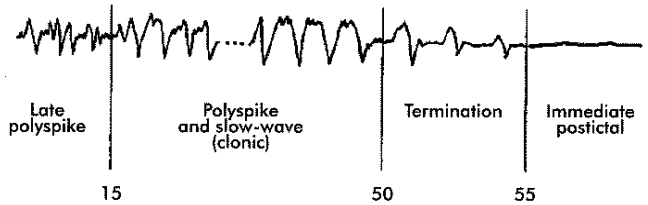
Sanghani SN et al., *Curr Opin Psy*, 2018

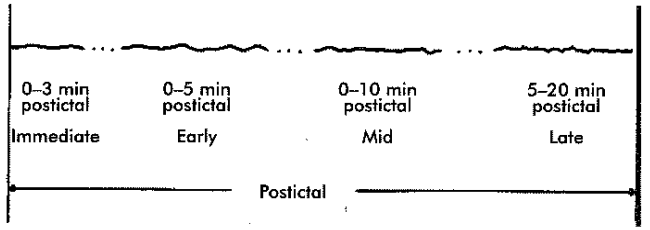
Goal of the treating ECT physician

- Deliver an electrical stimulus great enough to induce an *adequate seizure* while minimizing the risk of significant side-effects.









ECT – Treatment Course

Initial Consult

- Pre-ECT workup
- Consent

"Index" Series

- Tx 1: Seizure threshold (ST) determination
- Tx 2 + N: Consecutive treatments with stimulus above ST (e.g. M/W/F)
- Serial symptom and side-effect monitoring
- Treat to response (50% symptom reduction) vs. remission

Continuation Phase (cECT)

- Up to 6 months post-index series
- Titrate treatments: q2x/week → q1x/week → qbi-weekly → qmonthly
- +/- Symptom triggered treatment (PRIDE study)
- ***Relapse*** prevention strategy (50 – 60% relapse rates amongst remitters following an index series)

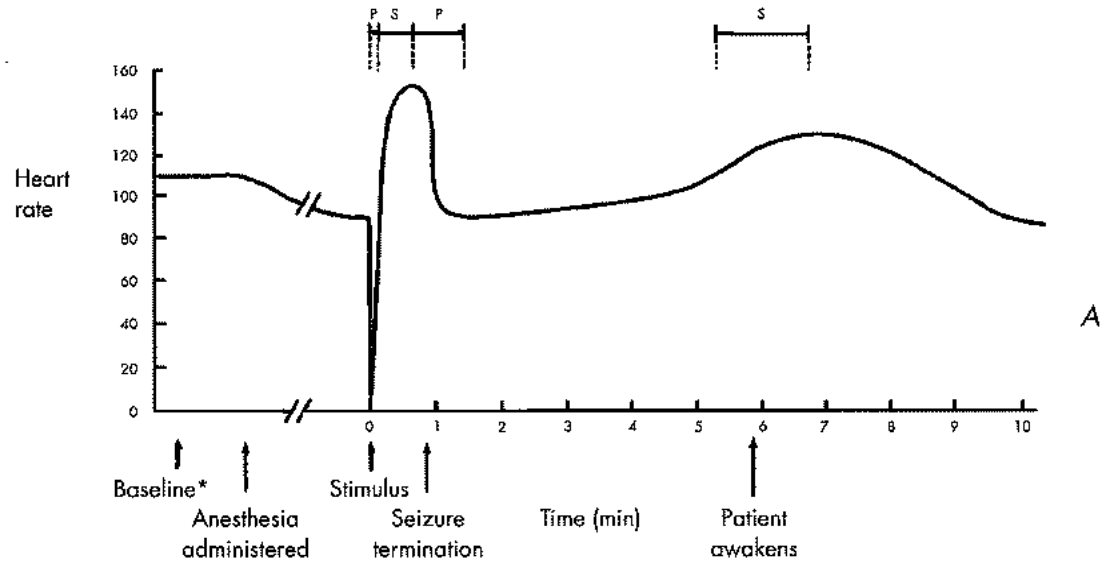
Maintenance Phase (mECT)

- Beyond 6 months post-index series
- ***Recurrence*** prevention strategy

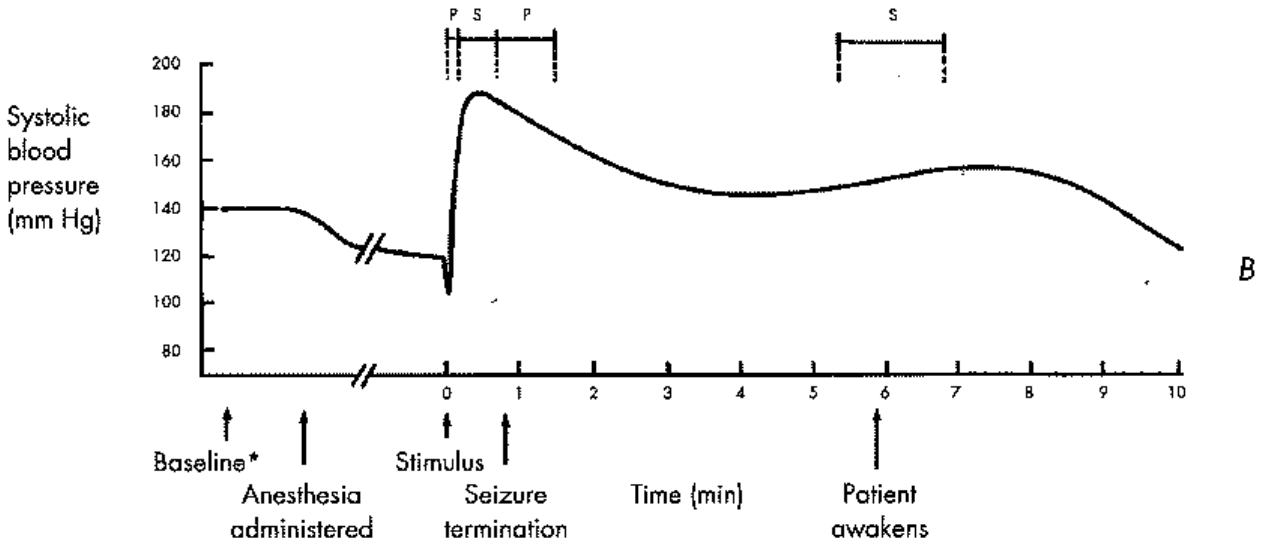
ECT – Treatment Day

1. Patient presents for first treatment, checks in with a psychiatrist
   - Review interim progress
2. *Received by nursing:*
   - Check NPO status, review outpatient medications, height/weight, vitals, connect rhythm EKG, insert IV, administer pre-procedures medications (e.g. anti-hypertensives, anti-emetics).
   - *Provides therapeutic support.*
3. Received by anesthesia/psychiatrist/nurse in treatment area
   - *Connection of EEG leads and ECT electrodes*
   - Pre-induction bag-mask ventilation (*no routine intubation*)
   - Induction of general anesthesia + neuromuscular blockade (succinylcholine)
   - Stimulus delivery → generalized tonic clonic seizure (max. 120sec)
   - Emergence and re-orientation; bag-mask respiratory support PRN
4. Patient received by nursing in recovery area:
   - Co-management of emergence delirium with anesthesiologist
   - Review of discharge readiness and administrative paperwork
   - Charting post-procedure vitals

ECT – Physiological response during the seizure



*In ECT treatment room
P = Parasympathetic response
S = Sympathetic response



*In ECT treatment room
P = Parasympathetic response
S = Sympathetic response

- Electrical stimulus induced, centrally mediated vagal discharge – bradycardia/asystole
- GTC induced, centrally mediated sympathetic discharge – tachycardia/HTN
- Post-ictal, peripherally mediated vagal discharge - bradycardia
- Minor arrhythmias: Inc. parasympathetic tone → atrial arrhythmias //. Inc. sympathetic tone → PVC, SVT

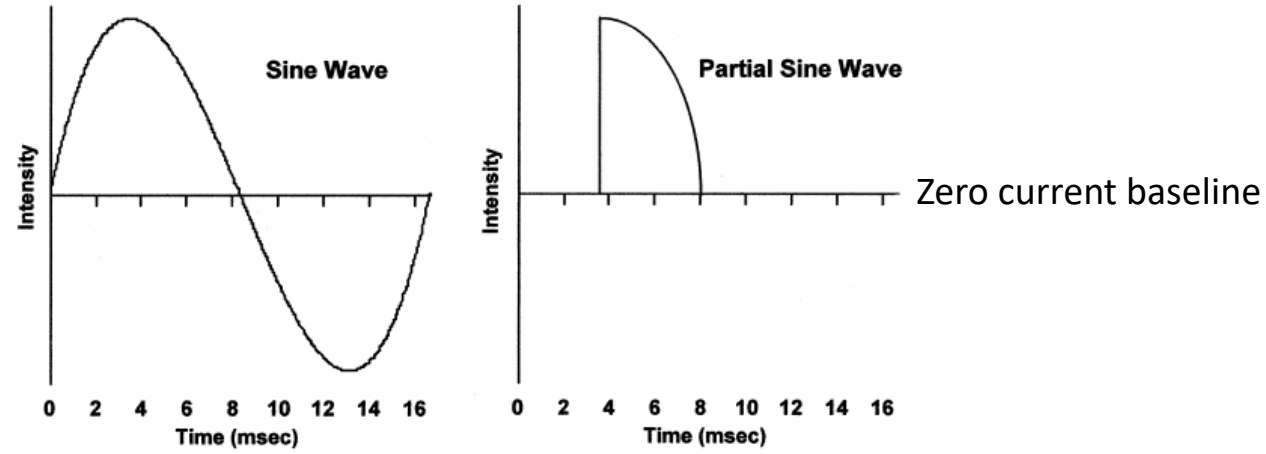
ECT – Common side-effects

- Myalgias after the first few treatments (succinylcholine)
- Nausea, typically anesthesia associated
- Temporalis/masseter tenderness, often transient
- Mild-headache
- Cognitive side-effects
  - Most common: short-term memory deficits, diminished concentration*, decreased reaction speed
  - Less common: autobiographical retrograde amnesia

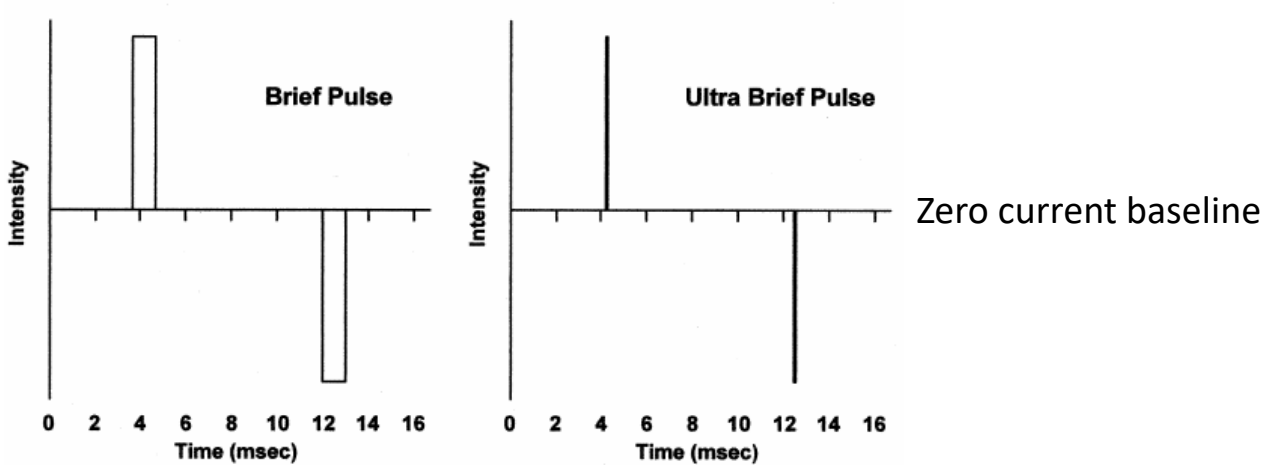
ECT: Mitigating side-effects, optimizing efficacy

- Energy dose

(60 Hz)



Zero current baseline

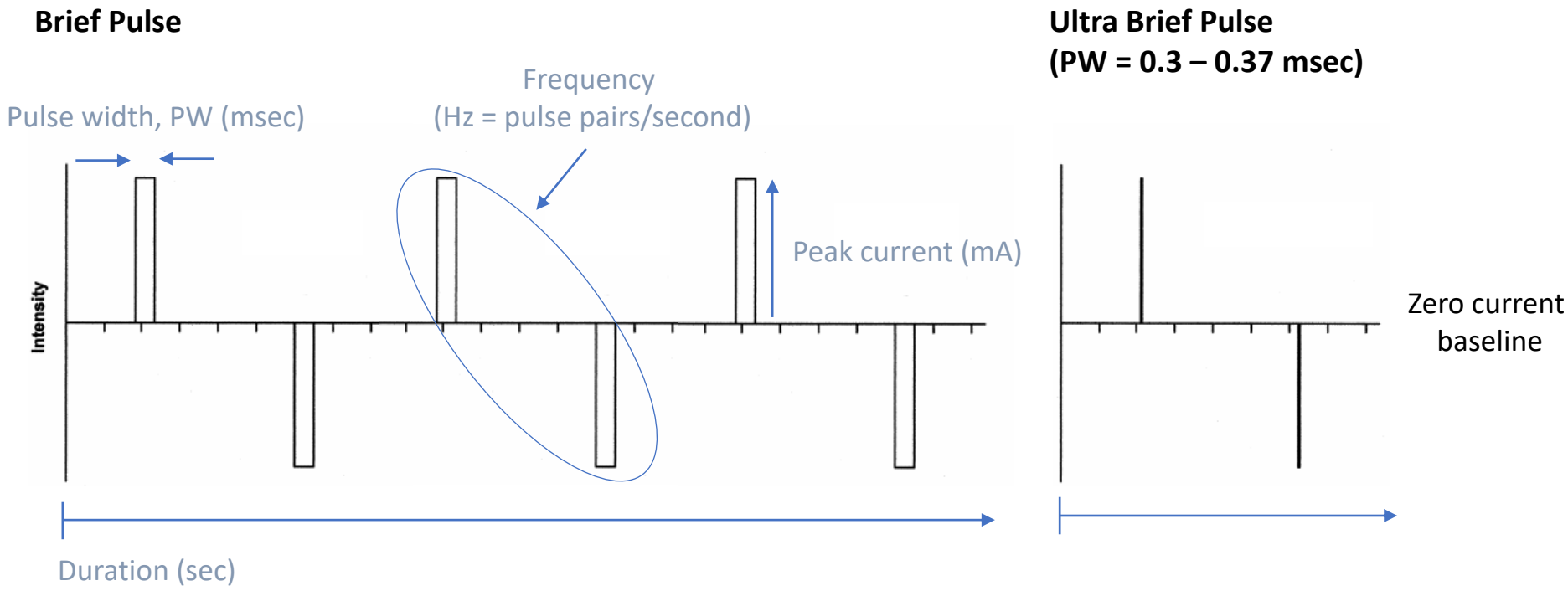


Zero current baseline

Sackeim HA, *Clin Neurosci Res,* 2004

ECT: Mitigating side-effects, optimizing efficacy

- Energy dose

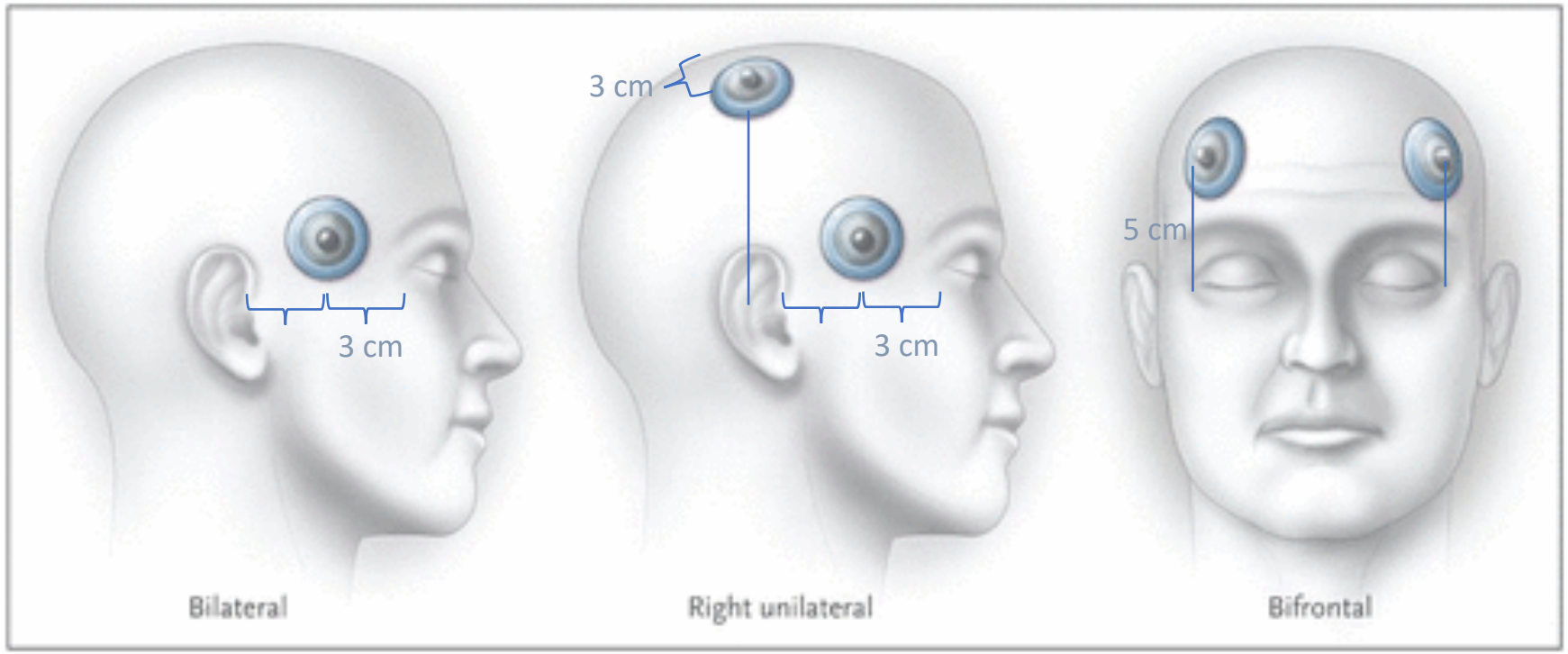


- *Energy (Joules)* = cumulative AREA under the "curve" of all square waves for a given stimulus duration.

Adapted from Sackeim HA, *Clin Neurosci Res,* 2004

ECT: Mitigating side-effects, optimizing efficacy

- Lead placement



Adapted from Lisanby S, NEJM, 2007 and Beyer JL et al., Electroconvulsive Therapy: A Programmed Text, 2nd Ed.

ECT: Mitigating side-effects, optimizing efficacy

- Lead placement: right unilateral (RUL) versus bilateral (BL)

1. Both generally effective in the treatment of *depression* (RUL @ 5-6x ST, BL @ 1.5 – 2x ST); mania and psychosis may *not* benefit from unilateral treatment.

(Sanghani et al., *Curr Opin Psychiatry*,2018; Ward HB et al., *Psych Res,* 2018)

2. Patients may respond *faster* (fewer treatments) to BL placement.

3. Switching lead placement (RUL → BL) is an *effective* approach in non-responsive patients.

(Abrams et al., *Am J Psych*,1983; Sackeim et al., *Arch Gen Psych*, 2009)

4. ***Cognitive side-effects:*** BL ECT > RUL; persistant memory complaints are more common in patients with BL ECT.

Impact of lead placement on cognitive side-effects

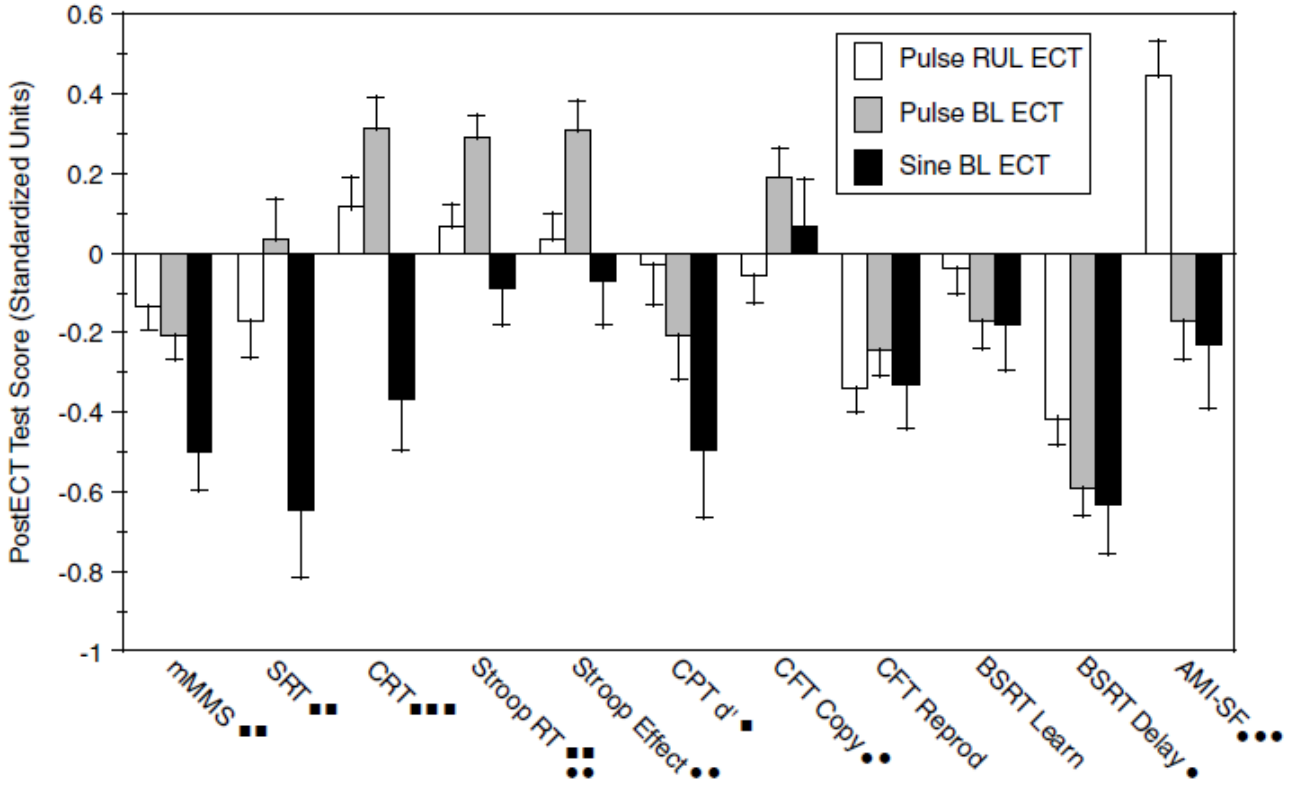


Figure 4 Scores on the 11 cognitive measures immediately following the ECT course for patients treated with brief pulse right unilateral (RUL). ECT, brief pulse bilateral (BL). ECT, and sine wave BL ECT. Filled boxes indicate a significant effect of waveform in the ANCOVA (■ = $p<0.05$; ■■ = $p<0.01$; ■■■ = $p<0.001$). Filled circles indicate a significant effect of electrode placement in the ANCOVA (● = $p<0.05$; ●● = $p<0.01$; ●●● = $p<0.001$).

Caveats:
Significant interactions with LP, # of treatments, premorbid IQ, HRSD at 6 month f/u, and baseline test score!!!

Sackeim HA, *Neuropsychopharmacology*, 2007

Impact of lead placement on cognitive side-effects

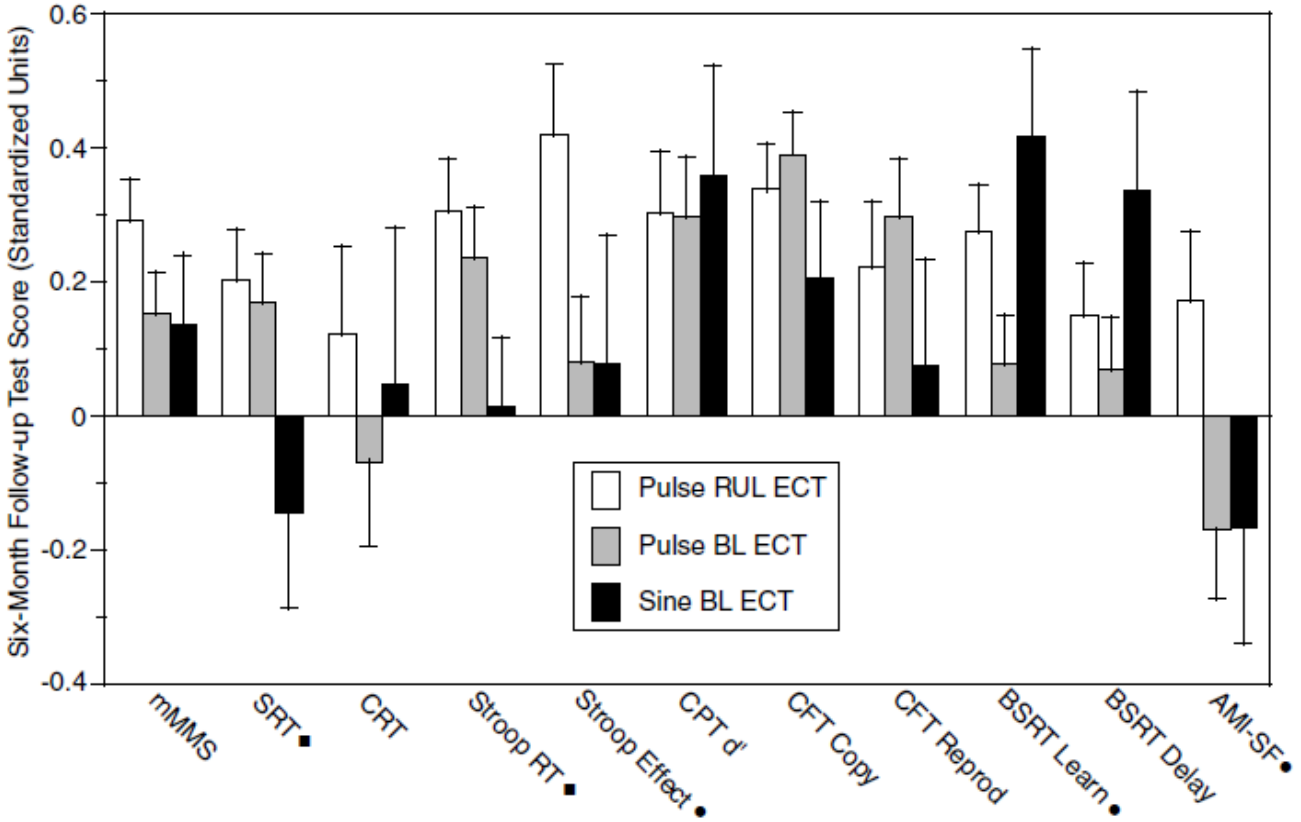


Figure 5 Scores on the 11 cognitive measures 6 months following the ECT course for patients treated with brief pulse right unilateral (RUL). ECT, brief pulse bilateral (BL). ECT, and sine wave BL ECT. Filled boxes indicate a significant effect of waveform in the ANCOVA (■ = $p < 0.05$). Filled circles indicate a significant effect of electrode placement in the ANCOVA (● = $p < 0.05$).

Caveats:
Significant interactions with LP, # of treatments, premorbid IQ, HRSD at 6 month f/u, and baseline test score!!!

Sackeim HA, *Neuropsychopharmacology*, 2007

Practical considerations in formulating candidacy

- Barriers to care:
  - Stigma
  - Provider/geographic access
  - State legislation regarding the use of ECT and requirements for informed consent
    - *How does your state handle ECT for patients without capacity to consent?*
  - Social support
    - Transportation; patient's can *not* drive at all during an "acute series" or on treatment days during "maintenance" phase

- Other considerations:
  - ✓ Covered by insurance (co-pay may apply)
  - ✓ May start as an *outpatient* or an *inpatient*
  - ✓ Many patients continue to work while receiving ECT

Preparing a patient for referral

- The proposal. . . “you’re recommending I have *WHAT?!*”

1. *Put the treatment in perspective:* extent and risk of ongoing suffer compared to the evidence for safety and efficacy.

2. *Acknowledge and confront the stigma head on.*

3. Understand the procedure and know what you are referring the patient for.

4. Reassurance that this is not abandonment nor an indication that “I can not help you.”

5. Invite the proposal to be an ongoing discussion

6. Provide literature/resources, but warn about common misconceptions and falsehoods on the internet.

Role of the outpatient psychiatrist

- Finding a place to refer, know your local resources.
- Pre-referral workup: Physical examination, CMP, CBC, TSH, EKG, urine toxicology, imaging (*not required, but if indicated*)
- Communicating with the ECT consultants.
    1. You are the BEST source for providing context for the referral; collateral is invaluable throughout an ECT course
    2. Help the consultant understand the overall formulation, e.g. What is a reasonable treatment goal for this patient based on pre-morbid functioning?
    3. Provide anticipatory guidance regarding possible barriers, e.g. stigma associated concerns, lack of family support, transportation/financial issues.
    4. Providing a comprehensive past-psychiatric history, especially with past medication trials and description of response
    5. Continue to provide medications and evaluate the patient throughout the ECT course; ECT providers usually do *not* assume the role of primary psychiatrist while performing ECT
    6. Consider ECT as a time to further optimize medications: lithium augmentation, cross-taper to TCA/MAOi

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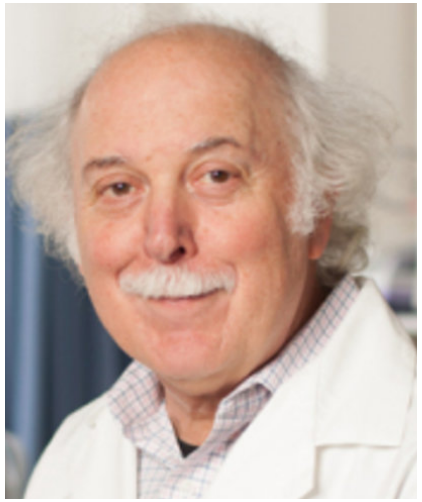

Referral Inquiries:

Clinical Treatment, ECT/ketamine/rTMS:

(203) 281-4106, extension 10

Clinical Trials, Yale Depression Research Program:

(203) 764-9131

Opportunities for CME and CEU's in ECT:

Yale-IPS Mini-Fellowship (2.5 day experience)

Please contact me! brandon.kitay@yale.edu

Yale NewHaven Health

Yale New Haven Psychiatric Hospital