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

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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
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

Neuromodulation

- Transcranial current stimulation (TCS)
- Repetitive transcranial magnetic stimulation (rTMS)
- Electroconvulsive therapy (ECT)
- Vagus nerve stimulation (VNS)
- Deep brain stimulation (DBS)

Least invasive

- Pharmaceuticals
  - Ketamine (IV, intranasal)
- Clinical Trials
  - Neurosteroids (IV, oral)
  - Psilocybin (oral)
  - Botox (IM)

Most invasive

Technical consideration:
Uses electrical stimulation applied to the scalp or via implantation of electrodes
Interventional Psychiatry: Treatments

Neurostimulation

- Transcranial current stimulation (TCS)
- Repetitive transcranial magnetic stimulation (rTMS)
- Electroconvulsive therapy (ECT)
- Vagus nerve stimulation (VNS)
- Deep brain stimulation (DBS)

Pharmaceuticals

- Ketamine (IV, intranasal)
- Neurosteroids (IV, oral)
- Psilocybin (oral)
- Botox (IM)
- Clinical Trials

Least invasive

Technical consideration:
Uses electrical stimulation applied to the scalp or via implantation of electrodes

Most invasive

Technical consideration:
Requires special expertise, monitoring, and in-office application
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-Interventional Psychiatry Service (IPS)
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
Yale-Interventional Psychiatry Service (IPS)

Unique Patients Treated Per Month

Patient Count

ECT

Ketamine

R² = 0.1575

R² = 0.5658
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
Logistical wisdom 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)
Yale-Interventional Psychiatry Service (IPS)

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

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...
“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

Surgeon and author Sherwin Nuland discusses the development of electroshock therapy as a cure for severe, life-threatening depression — including his own. It’s a moving and heartfelt talk about relief, redemption and second chances.
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

ECT

MDD 159 (60%)

Bipolar Disorder 62 (23%)

Psychosis 37 (14%)

Catatonia 7 (3%)

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

<table>
<thead>
<tr>
<th>Trial</th>
<th>Number of participants</th>
<th>Standardised effect size (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wilson 1963&lt;sup&gt;10&lt;/sup&gt;</td>
<td>12</td>
<td>-1.078 (-2.289 to 0.133)</td>
</tr>
<tr>
<td>West 1981&lt;sup&gt;11&lt;/sup&gt;</td>
<td>25</td>
<td>-1.255 (-2.170 to -0.341)</td>
</tr>
<tr>
<td>Lambourn 1978&lt;sup&gt;15&lt;/sup&gt;</td>
<td>40</td>
<td>-0.170 (-0.940 to 0.600)</td>
</tr>
<tr>
<td>Freeman 1978&lt;sup&gt;12&lt;/sup&gt;</td>
<td>40</td>
<td>-0.629 (-1.264 to 0.006)</td>
</tr>
<tr>
<td>Gregory 1985&lt;sup&gt;13&lt;/sup&gt;</td>
<td>69</td>
<td>-1.418 (-2.012 to -0.824)</td>
</tr>
<tr>
<td>Johnstone 1980&lt;sup&gt;14&lt;/sup&gt;</td>
<td>70</td>
<td>-0.739 (-1.253 to -0.224)</td>
</tr>
<tr>
<td>Pooled fixed effects</td>
<td></td>
<td>-0.911 (-1.180 to -0.645)</td>
</tr>
<tr>
<td>Pooled random effects</td>
<td></td>
<td>-0.908 (-1.270 to -0.537)</td>
</tr>
</tbody>
</table>

ECT for Treatment Refractory Depression

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<th>Trial</th>
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</thead>
<tbody>
<tr>
<td>Steiner 1978</td>
<td>12</td>
<td>0.369 (−0.840 to 1.578)</td>
</tr>
<tr>
<td>Wilson 1963</td>
<td>12</td>
<td>−0.513 (−1.663 to 0.637)</td>
</tr>
<tr>
<td>Davidson 1978</td>
<td>19</td>
<td>−1.389 (−2.449 to −0.328)</td>
</tr>
<tr>
<td>McDonald 1966</td>
<td>22</td>
<td>−0.930 (−1.813 to −0.047)</td>
</tr>
<tr>
<td>Gangadhar 1982</td>
<td>32</td>
<td>1.287 (0.406 to 2.169)</td>
</tr>
<tr>
<td>MacSweeney 1975</td>
<td>27</td>
<td>−0.714 (−1.492 to 0.065)</td>
</tr>
<tr>
<td>Dines 1989</td>
<td>30</td>
<td>−0.196 (−0.926 to 0.534)</td>
</tr>
<tr>
<td>Janekiramiah 2000</td>
<td>30</td>
<td>−1.095 (−1.863 to −0.328)</td>
</tr>
<tr>
<td>Folkerts 1997</td>
<td>40</td>
<td>−1.336 (−2.032 to −0.640)</td>
</tr>
<tr>
<td>Herrington 1974</td>
<td>43</td>
<td>−1.497 (−2.174 to −0.821)</td>
</tr>
<tr>
<td>Stanley 1962</td>
<td>47</td>
<td>−1.342 (−2.047 to −0.638)</td>
</tr>
<tr>
<td>Medical Research Council 1965</td>
<td>204</td>
<td>−0.559 (−0.883 to −0.234)</td>
</tr>
<tr>
<td>Greenblatt 1964</td>
<td>242</td>
<td>−1.683 (−2.020 to −1.346)</td>
</tr>
</tbody>
</table>

Pooled fixed effects: −1.010 (−1.170 to −0.856)
Pooled random effects: −0.802 (−1.290 to −0.259)
ECT for Treatment Refractory Depression

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

*Remission was defined as having a score \( \leq 10 \) 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
FIGURE 3. Speed of Remission Among Remitted Patients (N=148) in a Study of ECT and Venlafaxine in Geriatric Depression

ECT for Geriatric Depression

Kellner C et al., 2016
# Clinical Predictors of ECT Response for Depression and Mania

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

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

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

Ward HB et al., *Psychiatry Research*, 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

- 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

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; persistent memory complaints are more common in patients with BL ECT.
Impact of lead placement on cognitive side-effects

Caveats:
Significant interactions with LP, # of treatments, premorbid IQ, HRSD at 6 month f/u, and baseline test score!!!
Impact of lead placement on cognitive side-effects

Caveats:
Significant interactions with LP, # of treatments, premorbid IQ, HRSD at 6 month f/u, and baseline test score!!!
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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  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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Opportunities for CME and CEU’s in ECT:
Yale-IPS Mini-Fellowship (2.5 day experience)
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