Electroconvulsive Therapy: Pre-ECT Evaluation and Special Populations

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Objectives

- To review indications when selecting patients for ECT, with focus on special populations

- To review the pre-operative evaluation process
Disclosure: No conflicts
Toronto Star Stories (December 2012): “Electroshock therapy more prevalent in Ontario, but guidelines are minimal”

http://www.thestar.com/news/gta/2012/12/13/electroshock_therapy_more_prevalent_in_ontario_but_guidelines_are_minimal.html
Access to Electroconvulsive Therapy Services in Canada, Delva et al. J. ECT, Dec 2011

“Stigma, which was not specifically addressed in the questionnaire, was spontaneously identified as the most important single barrier by 6 of the 107 centers”
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Stigma Busters

- Kitty Dukakis

- Dr. Sherwin Nuland
  - www.ted.com
Guidelines and Policies
Guidelines and Position Papers


- Royal College (UK) of Psychiatrists Handbook of ECT, 2013

- BC ECT Guidelines 2002
<table>
<thead>
<tr>
<th>Year</th>
<th>Indication</th>
<th>Induction, Devices</th>
<th>Technique</th>
<th>Post-ECT Amnesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>1978</td>
<td>Severe depression, mania, psychosis, catatonia</td>
<td>GA, O₂, muscle relaxation. Sine wave or pulse wave devices</td>
<td>Favour UL over BL; no stimulus dosing noted</td>
<td>Retrograde (permanent and spotty) and anterograde</td>
</tr>
<tr>
<td>1990</td>
<td>Primary Mood Disorders, Schizophrenia</td>
<td>Strongly favour brief pulse device</td>
<td>Therapeutic window; fixed vs titrated dose</td>
<td>“1 in 200 report severe problems that remain for months or even years”</td>
</tr>
<tr>
<td>2001</td>
<td>Primary and secondary indications</td>
<td>“Sine wave device is not justified”</td>
<td>2.5-6X RUL, 1.5-2.5X BL. Favour titration</td>
<td>“spottiness in memory..may extend back several years or more”</td>
</tr>
<tr>
<td>Primary Indications</td>
<td>APA/BC Guidelines</td>
<td>AACAP Guidelines</td>
<td></td>
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<tr>
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<tr>
<td>Adolescents ECT</td>
<td>Mood Disorder, Schizoaffective, Sz</td>
<td>Severe, persistent, significantly disabling.</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Refractory to 2 med trials*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Secondary Indications</td>
<td>Catatonia, NMS, etc.</td>
<td>Catatonia, NMS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Screening</td>
<td>No routine</td>
<td>Pregnancy Test in all females; consider CT head</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Second Opinion</td>
<td>Mandatory by child or adolescent</td>
<td>Mandatory by independent practitioner</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>psychiatrist</td>
<td>knowledgeable in ECT</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Consent</td>
<td>Review every 15 treatments</td>
<td>“6-15 treatments” (12 tx.)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Electrode placement</td>
<td>No preference stated</td>
<td>Unilateral preferred; bilateral if urgent</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Seizure Threshold</strong></td>
<td>Dose titration or age-based</td>
<td>Lack evidence in either</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Except 1) medication intolerant, 2) unable to take medication, or 3) waiting for a response to a psychopharmacological treatment may endanger the life of the adolescent.

**Adolescents have very low seizure thresholds generally, so start as low as 1-2 %
“Are these just guidelines, or are they actual new policies?”
7.2 Are any particular sets of published guidelines closely followed in the delivery of ECT?

☐ No

☐ Yes

If the answer is “yes,” please specify: (check all that apply)

☐ ECT Guidelines for Health Authorities in BC (Mheccu)

☐ American Psychiatric Association (APA) Task Force Report

☐ U.K. Royal College of Psychiatrists

☐ Other (specify): ____________________________
Figure 1: The percentage of facilities with written policies/procedures for different aspects or components of ECT (n=89 responding sites)
Information
Figure 2: How ECT Information was provided to patients and families (n=106 responding sites)
BC ECT (2002) Video Website

- www.canects.org
- www.canects.org/patients.php
Electroconvulsive Therapy

Electroconvulsive therapy (ECT) is an effective psychiatric treatment in which seizures are electrically induced in anesthetized patients in order to achieve therapeutic benefits. ECT is most often recommended for use as a treatment for patients who are severely depressed, suicidal or have bipolar disorder with depression or mania.

The decision to use ECT as a treatment typically comes after discussion and exploration of other therapies. In order to inform discussion and decision making, the BC Ministry of Health has funded the creation of a 20-minute video guide for patients and families.

Click here to watch the English version
Click here to watch the French version
Click here to watch the Punjabi version
Click here to watch the Cantonese version
Websites

- Canadian Psychiatric Association Position Paper
  - www.cpa-apc.org


- Mayo Clinic Web Site:
  - www.mayoclinic.com/health/electroconvulsive-therapy/MY00129

- ECT Accreditation Service in UK (ECTAS)
  - www.rcpsych.ac.uk/quality/qualityandaccreditation/ectclinics/ectas.aspx
ECT Consultation

- **Indication?**
  - Primary vs. Secondary

- **Contraindications or Risks?**
  - Medical vs. Psychiatric
  - Lab Investigations

- **Concurrent Medications?**
  - Discontinue, Hold, Taper

- **Consent?**
  - Involuntary or Voluntary
  - Capable or Incapable
ECT Consultation (cont’d)

Outpatient Considerations

- Behavioural restrictions
  - Fasting
  - Not driving

- Logistical issues
  - Transport, and responsible person to pick up and monitor
  - Duration and frequency of treatment
ECT Response Rates

- Generally: 75-85%
- Elderly: 80-90%
- Med-resistant: 60-70%
- Clinical Practice in Elderly: >90%
ECT: The CORE Experience

- Multi-site, prospective, RCT for C-ECT vs. C-Meds
- N=253 MDD, 1.5T BL ECT Index Series, open label phase
- 86% completers, 80% response overall

- TRD was not a predictor of response

- Nonpsychotic (n = 176) vs. psychotic (n = 77)
  - Psychotic depression remitted faster

- Higher HRSD **remission** rates (overall 75%) with age:
  - 45 and under: 70%
  - 46-64 y.o.: 89.8%
  - 65 and over: 90%

- Median time for:
  - Response: 3 ECT’s
  - Relief from suicidal ideas: 4 ECT’s


- **Design**
  - 60 y and older eligible, multi-centred
  - Mean age 70, 10% psychotic depression
  - Phase 1: RUL ultrabrief pulse width ECT augmented with Venlafaxine; 81% pts seized @ 5%stim (ECT#1)
  - Phase 2: Remitters randomized to 6 month continuation ECT: Venlafaxine-Li or Venlafaxine-Li-ECT

- Phase 1 prelim data Feb 2010-Apr 2012 of index ECT’s:
  - n=121, 65% remitters, 28% dropout
ECT and Geriatric Depression

- Right unilateral and bilateral ECT can be effective

- Possible cognitive improvement when MMSE < 24, as symptoms remit (Tielkes 2008)
  - less improvement seen with BL ECT

- “Pseudodementia” from depression can respond well to ECT (Wagner 2011, Rapinesi 2013)

- Late onset geriatric depression is a predictor for developing dementia later (Barnes 2012, Byers 2012)
  - 2-4X risk

- Incorporate in consent procedure?
ECT and Dementia with Depression

- Mood disorders complicating dementia can respond well to ECT (Weintraub 2001; Rao 2001) BUT:
  - Non-specific effects of ECT on agitation
  - Higher risk for post-ECT Delirium and lingering cognitive disturbance—consider discussing during consent

- Only 5 prospective trials (Oudman, J ECT, Mar/12)

- Best to stay on ChEI’s during ECT (Hausner et al. J. Clin. Psychiatry 2011;76).
  - ChEI’s considered safe with ECT, though may enhance bradycardic response in a few.
Primary Indications

- Major Depressive Episode
- Mania
- Mixed States
- Schizophrenia
- Schizoaffective Disorder
1. Acute suicidality with high risk of acting out suicidal thoughts.

2. Psychotic features.

3. Rapidly deteriorating physical status due to complications from the depression, such as poor oral intake.

4. History of poor response to medications.
Primary Indications: Mood Disorders

5. History of good response to ECT.


7. The risks of standard antidepressant treatment outweigh the risks of ECT, particularly in medically frail or elderly patients.

8. Catatonia
Primary Indication: Mania

- Features as per Major Depression
- Extreme agitation
- “Manic Delirium”
Primary Indication: Schizophrenia and related Disorders

- i. Positive symptoms with abrupt or recent onset.
- ii. Catatonia.
- iii. History of good response to ECT.
- (refractory psychosis)
Secondary Indications

- Catatonia
- Parkinson’s Disease
- Neuroleptic Malignant Syndrome (NMS)
- Mood Disorder Secondary to Physical Conditions
- Delirium
- Intractable Status Epilepticus
Catatonia

Hawkins et al 1995
- N=178, 1985-1994 published cases
- Benzo’s effective in 70% (Lorazepam)
- ECT effective in 85%
- Antipsychotics effective in 7.5%, or may even worsen symptoms (“neuroleptic-induced catatonia”)

Taylor MA, Am J Psych 2003
- In the modern era, the most likely psychiatric cause for catatonia is Mood Disorder:
  - Especially Mania in Adults and more likely when severe mania
  - Kahlbaum, Bleuler, Kraepelin all noted mood disturbance preceding catatonia

Review: Daniels, J. Neuropsych Clin Neurosci 2009
ECT Risks

- Headaches
- Musculo-skeletal pain
- Nausea, vomiting
- Cardiovascular complications
- Mortality: 1/80,000 individual ECT’s = approx. 1/10,000 index courses
- Anterograde amnesia
- Retrograde amnesia
- Dental Injury: covered well in UK guide
Mechanisms
J. ECT, June 2014
Monoamines
- Noradrenaline:
  - Down-regulate B-receptors, decreased $\alpha_2$ adrenoreceptors
- Serotonin:
  - Up-regulate $5HT_{1A}$ receptors in the hippocampus
  - Up-regulate $5-HT_{2A}$ receptors in prefrontal regions
  - Decrease $5HT_{1A}$ and $5-HT_{2A}$ receptor binding in humans
- Dopamine:
  - Sustained increase in DA transmission and metabolites
  - Increase $D_1$ receptor binding in striatum, nuc accumbens
  - Increase $D_3$ receptor binding in striatum

Others
- GABA:
  - Sustained increase, increase GABA receptors
- Glutamate:
  - Increase in glutamate receptor expression (NMDA)
Other Theories

- **Brain-derived neurotrophic factor (BDNF) and Neurogenesis**
  - Limbic BDNF levels diminish in those with depression correlating with decreased hippocampal and prefrontal volumes in the brains of depressed patients (Duman, Biol Psychiatry 2006).
  - Antidepressant meds can restore BDNF, esp. in hippocampus, with accompanying dendritic arborization, as can ECT and rTMS.

- **Restoring central diencephalic (hypothalamic) function**
  - Haskett, J ECT, 2014

- **Alterations in cerebral metabolic activity and blood flow**
  - Bolwig, J ECT 2014

- **Connectivity Hypothesis**
  - Farzan, J ECT 2014

- **Inflammatory (cytokines) modulator?**
  - Guloksuz, J ECT 2014
Neurogenesis, neuroplasticity, and dendritic sprouting in key areas such as the hippocampus in animal models involving electroconvulsive stimulation (ECS), which can translate into changes in monoamine transmission (Wahlund, Neuropsychopharm. 2003)

The rise in BDNF with chronic ECS has been the most studied to date (Taylor, J ECT. 2008), with alterations noted in different brain regions such as hippocampus, ventral tegmental area (Taliaz 2012, Kyeremanteng 2012, Segawa 2013)

ECT can give a rise in plasma BDNF levels which correlate with clinical response (Stelzhammer 2012, Hu 2010, Marano, 2007) but not always (Fernandes 2009, Gronli 2009)
Cerebral Metabolic and Connectivity Alterations

- Some studies have shown a course of ECT will reduce regional cerebral blood flows (rCBF) or cerebral metabolic rates in medial PFC and ACC, and increase blood flow in thalamus (Nobler, Am. J. Psychiatry 2001; Takano Br J Psych 2007). This is an inconsistent finding though (Yatham, J. ECT. 2000).
  - Indirect evidence from rTMS and DBS also.

- Changes in Connectivity
  - Hyperconnectivity Hypothesis: Evidence for ECT producing...
    - Reduced left DLPFC “functional connectivity” (Perrin 2012)
    - Increased ACC functional connectivity to right DLPFC and PCC (Beall 2012)
    - Reduced deactivation of orbitofrontal cortex to negatively valenced emotional stimuli (Beall 2012)
  - Connectivity Resetting Hypothesis (Farzan): Evidence for ECT producing...
    - “resetting aberrant neural connectivity, likely mediated through activating thalamocortical pathways and central inhibitory mechanisms, and increasing the possibility of formation of newer and healthier connection by promoting neurogenesis”
    - Changes in cortical oscillations with ECT of delta, theta, alpha waves (EEG)
Reviews

- Kellner, ECT for TRD. Am J. Psychiatry Dec 2012

- Tess, “Medical Evaluation of Patients Undergoing ECT”, NEJM, April 2, 2009

- Verwijk, “Neurocognitive effects after brief pulse and ultrabrief pulse unilateral electroconvulsive therapy for major depression: A review”, J. Affective Dis 140 (2012)