Anesthesia and ECT

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3 pages on anesthesia - together
ECT – anesthesia:

1. Typical anesthetic drugs (p, e, m, t)
   a. Ketofol – two stones to catch one bird?

2. Some typical problems / solutions …
   a. ASTI
   b. Oxygen
   c. PAS / PIA
   d. Cardiac
<table>
<thead>
<tr>
<th>substance</th>
<th>typical dose range (mg/kg)</th>
<th>anticonvulsive effect (relative)</th>
<th>remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>methohexital</td>
<td>0.75-1.0</td>
<td>1-2</td>
<td>former gold standard, cardiovascular depression</td>
</tr>
<tr>
<td>thiopental</td>
<td>2-5</td>
<td>2</td>
<td>cardiovascular depression</td>
</tr>
<tr>
<td>propofol</td>
<td>1-2</td>
<td>3</td>
<td>shorter seizures, higher seizure threshold</td>
</tr>
<tr>
<td>etomidate</td>
<td>0.2-0.3</td>
<td>0</td>
<td>myocloni</td>
</tr>
<tr>
<td>S-ketamine</td>
<td>0.5-1.5</td>
<td>0</td>
<td>low doses pro-psychotic, higher blood pressure</td>
</tr>
<tr>
<td>alfentanil</td>
<td>0.01-0.015</td>
<td>0</td>
<td>longer time of apnoe, cardiovascular depression</td>
</tr>
<tr>
<td>remifentanil</td>
<td>0.001-0.008</td>
<td>1</td>
<td>similar to alfentanil ?</td>
</tr>
</tbody>
</table>

* Hikma Pharmaceuticals stopped the production of methohexital 2019
** Good Manufacturing Practice (GMP) problems at Lampugnani Pharmaceutici SPA
*** In Germany: more or less obsolete for critically ill patients and for non-single induction use

Adapted from:
Swartz CM Electroconvulsive and neuromodulation therapies. 2009 Cambridge Univ, Cambridge New York Melbourne
ECT – anesthesia:

- thiopental 3-5 mg/kg
- methohexital 50-120 mg
- etomidate 0.15-0.3 mg/kg
- propofol 1-2 mg/kg
Etomidate:
to use or not to use for endotracheal intubation in the critically ill?
Smischney NJ, Kashyap R, Gajic O.

This debate is ongoing …
… and of course there are no long term studies in ECT patients…

The theoretical problem of a cumulative risk remains, because of a „chronic“ HPA suppression with repeated use of etomidate. At least in Germany anesthesiologists become more and more „careful“ with the use of etmidate in ECT.
ECT – anesthesia:

- thiopental: 3-5 mg/kg
- methohexital: 50-120 mg
- etomidate: 0.15-0.3 mg/kg
- propofol: 1-2 mg/kg
<table>
<thead>
<tr>
<th></th>
<th>Treatment</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>lorazepam</td>
<td>up to 10mg i.v.</td>
</tr>
<tr>
<td>2</td>
<td>phenytoin</td>
<td>up to 20 mg/kg i.v.</td>
</tr>
<tr>
<td>or</td>
<td>phenobarbital</td>
<td>20 mg/kg i.v.</td>
</tr>
<tr>
<td>3</td>
<td>thiopental</td>
<td>5 mg/kg i.v.</td>
</tr>
<tr>
<td></td>
<td>vs. propofol</td>
<td>2 mg/kg i.v.</td>
</tr>
<tr>
<td></td>
<td>vs. midazolam or valproate or levetiracetam</td>
<td></td>
</tr>
</tbody>
</table>

* German Association for Neurology
ECT – anesthetia: Thiopental

32 years, “always had good seizures”, now 250 mg propofol
Anesthesia beyond the usual suspects:

Propofol:

A Bayesian framework systematic review and meta-analysis of anesthetic agents effectiveness/tolerability profile in electroconvulsive therapy for major depression.

The aim of this study was to assess the efficacy and tolerability/acceptability of 6 anesthetic agents in ECT for depressive disorders. We systematically reviewed 14 double-blind randomized controlled trials (610 participants). Efficacy was measured by the mean scores on validated depression scales at 6 ECT (or the nearest score if not available), number of responders at the end of treatment and seizure duration. The acceptability was measured by the proportion of patients who dropped out of the allocated treatment, and the tolerability by the number of serious adverse events and post-treatment cognition assessment.

After excluding the trials responsible for heterogeneity, depression scores of patients who were administered methohexital were found to be significantly more improved than those who received propofol (p = 0.001). On the contrary, those who were administered propofol had lower depression scores than those with thiopental at the end of treatment (p = 0.002).

Compared to propofol, methohexital was found to be significantly associated with higher seizure duration (p = 0.018). No difference was found for the acceptability profile (all p > 0.05).

In summary, ketamine and methohexital may be preferred to propofol or thiopental in regard of effectiveness in depression scores and increased seizure duration. Further studies are warranted to compare ketamine and methohexital.
ECT – anesthesia: All four gone?

- thiopental: 3-5 mg/kg
- methohexital: 50-120 mg
- etomidate: 0.15-0.3 mg/kg
- propofol: 1-2 mg/kg
ECT – anesthesia: All four gone?

- thiopental 3-5 mg/kg
- methohexital 50-120 mg
- etomidate 0.15-0.3 mg/kg
- propofol 1-2 mg/kg

What about ketamine?
1962  first synthesis of ketamine (Parke-Davis)
1964  „psychodelic“ potential w (EF Domino, U Michigan, in a self-experiment)
1965  „dissociative“ is introduced for ketamine
1966  patented
1970  FDA approval

synonyms: K, Kate, Ket, Keta, Kite, Kitty, Kiti, Special K, Vitamin K, Multiketamin, Fiction …
NMDA channel blockers are noncompetitive antagonists and generally lack subunit selectivity. In addition, known channel blockers have a wide range of off-target activities (e.g., D₂, 5-HT, GABA, μ-, κ-opioid, σ, mAChR) and block channels other than NMDA (Na, nAChR, HCN1, and K_ATP). Examples of high-affinity blockers are ketamine (4), phenylcyclidine (PCP, 5), and MK-801 (6), while memantine (7) and amantadine (8) are low-affinity blockers (Kᵢ > 1 μM).
ketamine in general anesthesia:

- is listed as an essential drug by the WHO
- often used in emergency medicine
- treatment of status asthmaticus
- analgesia of intubated patients
- preferred for child and ado’s
- still in use for general and regional anesthesia
  - alone and in combination with hypnotics
- off-label for chronic pain patients
Box 1 | Assessing altered states of consciousness

dose-effect relation

Glue et al., Biol Psychiatry, 2011

ketamine racemate in mg/kg bw i.m. as bolus
ECT and ketamine

pros:

1. Ketamine probably possesses a unique intrinsic antidepressive potential
2. Ketamine has no anticonvulsive action
3. Ketamine may posses neuroprotective properties as an NMDA-antagonist

cons:

1. Ketamine acts non-depressively on the cardio-vascular system (like e.g. barbiturates)
2. Ketamine dose-dependently induces psychiatric side-effects (basically derealisation and depersonalisation, which can lead to anxiety)
In a multiple logistic regression model, higher adequacy was significantly related with anesthesia (p<0.001) - favoring etomidate and ketamine over thiopental and propofol.
### Fig. 2. (A): Meta-analysis of depressive symptoms with ketamine at the end of ECT course.

<table>
<thead>
<tr>
<th>Study or Subgroup</th>
<th>Ket</th>
<th>Con</th>
<th>Std. Mean Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abdallah 2012</td>
<td>22.9</td>
<td>20.4</td>
<td>3.4%</td>
</tr>
<tr>
<td>Alizadeh 2015</td>
<td>16.27</td>
<td>14.77</td>
<td>6.82</td>
</tr>
<tr>
<td>Anderson 2017</td>
<td>17.2</td>
<td>15.4</td>
<td>10.4</td>
</tr>
<tr>
<td>Chen 2017</td>
<td>10.07</td>
<td>9.56</td>
<td>63</td>
</tr>
<tr>
<td>Fernie 2017</td>
<td>13.5</td>
<td>9.32</td>
<td>16</td>
</tr>
<tr>
<td>Jarventausta 2013</td>
<td>10</td>
<td>10.93</td>
<td>16</td>
</tr>
<tr>
<td>Kuscu 2015</td>
<td>4.5</td>
<td>2.58</td>
<td>38</td>
</tr>
<tr>
<td>Loo 2012</td>
<td>14.28</td>
<td>10.34</td>
<td>22</td>
</tr>
<tr>
<td>Rasmussen 2014</td>
<td>22.08</td>
<td>8.11</td>
<td>21</td>
</tr>
<tr>
<td>Rybakowski 2016</td>
<td>12.5</td>
<td>6.05</td>
<td>30</td>
</tr>
<tr>
<td>Salehi 2015</td>
<td>8.32</td>
<td>5.17</td>
<td>80</td>
</tr>
<tr>
<td>Yoosif 2014</td>
<td>17.2</td>
<td>2.46</td>
<td>17</td>
</tr>
<tr>
<td>Zhong 2017</td>
<td>15.55</td>
<td>7</td>
<td>43</td>
</tr>
<tr>
<td>Zhong 2016</td>
<td>6.55</td>
<td>1.34</td>
<td>60</td>
</tr>
</tbody>
</table>

**Total (95% CI)**: 462 | 390 | 100.0% | -0.17 [-0.39, 0.06] |

Heterogeneity: $\tau^2 = 0.10$; $\chi^2 = 30.42$, df = 13 ($P = 0.004$); $I^2 = 57\%$

Test for overall effect: $Z = 1.47$ ($P = 0.14$)

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### Fig. 4. (A): Meta-analysis of depressive symptoms with ketamine alone at the end of ECT course.

<table>
<thead>
<tr>
<th>Study or Subgroup</th>
<th>Ket(alone)</th>
<th>Con</th>
<th>Std. Mean Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fernie 2017</td>
<td>13.5</td>
<td>8.41</td>
<td>16</td>
</tr>
<tr>
<td>Kuscu 2015</td>
<td>4.8</td>
<td>3.7</td>
<td>19</td>
</tr>
<tr>
<td>Rybakowski 2016</td>
<td>12.5</td>
<td>6.05</td>
<td>30</td>
</tr>
<tr>
<td>Salehi 2015</td>
<td>8.32</td>
<td>5.17</td>
<td>80</td>
</tr>
<tr>
<td>Yoosif 2014</td>
<td>17.2</td>
<td>2.46</td>
<td>17</td>
</tr>
<tr>
<td>Zhong 2016</td>
<td>6</td>
<td>0.7</td>
<td>30</td>
</tr>
</tbody>
</table>

**Total (95% CI)**: 192 | 176 | 100.0% | -0.27 [-0.83, 0.29] |

Heterogeneity: $\tau^2 = 0.40$; $\chi^2 = 29.77$, df = 5 ($P < 0.0001$); $I^2 = 83\%$

Test for overall effect: $Z = 0.93$ ($P = 0.35$)
Cognitive function outcomes.

<table>
<thead>
<tr>
<th>Study</th>
<th>Cognitive evaluation</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shams et al., 2015</td>
<td>“Cognitive Performance Recovery Time” after each ECT</td>
<td>Ketamine group had a shorter cognitive performance recovery time compared to propofol group. No significant differences between ketamine + propofol or thiopental and propofol or thiopental groups</td>
</tr>
<tr>
<td>Anderson et al., 2017</td>
<td>Hopkins Verbal Learning Test-Revised (HVLT-R-DR); originally Controlled Oral Word Association Test (COWAT); Autobiographical Memory Interview-Short Form (AMI-SF); Medical College of Georgia Complex Figure Test (MCGFCT); Oral digit span forwards and backward; Self-reported Global Self Evaluation of Memory (GSE-My)</td>
<td>MMSE score was significantly lower in the ketamine group compared to the propofol group; WMS-RC score was significantly lower in propofol group compared to ketamine + propofol group No significant differences between ketamine + thiopental and thiopental groups</td>
</tr>
<tr>
<td>Chen et al., 2017</td>
<td>MMSE; Wechsler Memory Scale-Chinese Revision (WMS-RC)</td>
<td>Ketamine group showed a lower degree of executive cognitive impairment compared to the ketamine + propofol and propofol groups</td>
</tr>
<tr>
<td>Loo et al., 2012</td>
<td>Medical College of Georgia Complex Figure (CFT); Hopkins Verbal Learning Test (HVLT); Controlled Oral Word Association Test (COWAT); Symbol Digit Modalities Test (SDMT); Woodcock Johnson Cross-Out Test; Autobiographical Memory Interview—short form (AMI-SF),</td>
<td>No significant difference was found on the MCCB between the propofol group and the ketamine plus propofol group</td>
</tr>
<tr>
<td>Zhong et al., 2016</td>
<td>The Word Fluency Test; Symptom Checklist (SCL); Wisconsin Card Sorting test (WCST); the Tower of Hanoi; the Trail Making Test (TMT); the Visual Regeneration Test.</td>
<td>No significant difference was found on the CANTAB SRM between the propofol group and the ketamine group A significantly better cognitive performance was evident in ketamine-receiving group No significant difference was found in MMSE</td>
</tr>
<tr>
<td>Zhang et al., 2017</td>
<td>Speed of Processing (SoP), Attention/Vigilance (AV); Working Memory (WM); Verbal Learning (Vrlb Lrng); Visual Learning (Vis Lrng); Reasoning and Problem Solving (RPS); Social Cognition (SC)</td>
<td>No significant difference was found in the test of visual-spatial function. Impairment of verbal memory and verbal fluency were greater with ketamine.</td>
</tr>
<tr>
<td>Femie et al., 2017</td>
<td>Cambridge Automated Neuropsychological Test Battery Spatial Recognition Memory Task (CANTAB SRM)</td>
<td>No significant difference was found on the CANTAB SRM between the propofol group and the ketamine group</td>
</tr>
<tr>
<td>Yoosefi et al., 2014</td>
<td>Mini-Metal State Examination (MMSE)</td>
<td></td>
</tr>
<tr>
<td>Rasmussen et al., 2014</td>
<td>Mini-Mental State Examination (MMSE)</td>
<td>No significant difference was found in MMSE</td>
</tr>
<tr>
<td>Ray-Griffith et al., 2017</td>
<td>Mini-Mental State Examination (MMSE)</td>
<td>No significant difference was found in MMSE</td>
</tr>
<tr>
<td>Rybakowski et al., 2016</td>
<td>Tests assessing visual-spatial function Tests assessing verbal auditory function Tests assessing working memory and executive function</td>
<td>No difference was found in the test of visual-spatial function. Impairment of verbal memory and verbal fluency were greater with ketamine.</td>
</tr>
</tbody>
</table>

To conclude so far, ketamine is

- Probably not as side effectively as it was feared

- Probably not more, but definitely not less effective as the grand old four (metho, thio, propo and eto)

- Our experience is that we need less charge for ketamine which explains no difference in response rates, but still could lead into less cognitive side effects (still has to be verified)

- no study has controlled for mean charge so far
ECT – anesthesia:

- thiopental: 3-5 mg/kg
- methohexital: 50-120 mg
- etomidate: 0.15-0.3 mg/kg
- propofol: 1-2 mg/kg

- ketamine (instead-of OR also an add-on?)
ketofol !
propofol plus ketamine = **ketofol**:

Mind the order:

**ketofol**: first propofol – followed by ketamine!
Anesthesia beyond the usual suspects:

- Ketofol (=> reduce k and p, not just add-on !)

Ketofol in electroconvulsive therapy anesthesia: two stones for one bird.

<table>
<thead>
<tr>
<th>Incident</th>
<th>Propofol group (n = 30)</th>
<th>Ketamine group (n = 30)</th>
<th>Ketofol group (n = 30)</th>
<th>p (ANOVA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Motor seizure (s)</td>
<td>29.3 ± 5.1</td>
<td>37.2 ± 3.2*</td>
<td>34 ± 5.8*</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Spontaneous breathing (s)</td>
<td>252 ± 13.1</td>
<td>266.6 ± 11.5*</td>
<td>260.7 ± 8.3</td>
<td>0.001</td>
</tr>
<tr>
<td>Open eyes (s)</td>
<td>413.1 ± 19.8</td>
<td>538.8 ± 43.2*+</td>
<td>436.2 ± 32.1</td>
<td>&lt;0.001, &lt;0.001</td>
</tr>
<tr>
<td>Obey commands (s)</td>
<td>514.3 ± 38.7</td>
<td>576.5 ± 37.6*+</td>
<td>519.9 ± 31.1</td>
<td>&lt;0.001, &lt;0.001</td>
</tr>
</tbody>
</table>

* p < 0.001 (post hoc Bonferroni) compared with group propofol
+ p < 0.001 (post hoc Bonferroni) compared with group ketofol
Own experiences with ketofol:

- anesthesiologists are excited
- less time in recovery room
- less side effects regarding post ictal agitation and psychomimetic problems
- propofol is still critical regarding seizure threshold/induction

- But how to mix propofol and ketamine ???
ECT – anesthesia: propofol+ketamine 1 : 1 ?


Anaesthesia for electroconvulsive therapy - new tricks for old drugs: a systematic review.

Stripp TK, Jorgensen MB, Olsen NV.

OBJECTIVE:
The objective of this review is to investigate existing literature in order to delineate whether the use of anaesthesia and timing of seizure induction in a new and optimised way may improve the efficacy of electroconvulsive therapy (ECT).

METHODS:
PubMed/MEDLINE was searched for existing literature, last search on 24 June 2015. Relevant clinical studies on human subjects involving choice of anaesthetic, ventilation and bispectral index (BIS) monitoring in the ECT setting were considered. The references of relevant studies were likewise considered.

RESULTS:
Propofol yields the shortest seizures, etomidate and ketamine the longest. Etomidate and ketamine+propofol 1 : 1 seems to yield the seizures with best quality. Seizure quality is improved when induction of ECT is delayed until the effect of the anaesthetic has waned - possibly monitored with BIS values. Manual hyperventilation with 100% O2 may increase the pO2/pCO2-ratio, which may be correlated with better seizure quality.

CONCLUSION:
Etomidate or a 1 : 1 ketamine and propofol combination may be the best method to achieve general anaesthesia in the ECT setting. There is a need for large randomised prospective studies comparing the effect of methohexital, thiopental, propofol, ketamine, propofol+ketamine 1 : 1 and etomidate in the ECT treatment of major depressed patients. These studies should investigate safety and side effects, and most importantly have antidepressant efficacy and cognitive side effects as outcome measures instead of seizure quality.
ECT: a new look at an old friend

Pavan Kumar Kadiyala\textsuperscript{a} and Lakshmi Deepthi Kadiyala\textsuperscript{b}

KEY POINTS

- ECT is improving into a new form that may be perceived with a lower degree of social stigma.

- Anesthesia and augmentation strategies have a significant influence on clinical efficacy and tolerability of ECT. Etomidate, or a ketamine-propofol combination, may be the first choice. Dexmedetomidine or remifentanil may be added in selected patients.

- Hyperventilation protocols and ASTI influence the clinical outcome of ECT.

- Refinements in stimulus parameters and electrode placements leading to increased focality have led to a reduction of cognitive adverse effects. RUL brief pulse ECT represents an acceptable first-line form of ECT.

- EEG ictal indices (specifically mid-ictal amplitude, postictal suppression) during ECT procedure should be monitored for therapeutic adequacy of seizure.

propofol+ketamine 1 : 1 ?

Own experiences with ketofol (unpublished):

- 52 patients treated with ketofol, 912 ECT sessions included:

<table>
<thead>
<tr>
<th></th>
<th>mean</th>
<th>min</th>
<th>max</th>
</tr>
</thead>
<tbody>
<tr>
<td>propofol</td>
<td>0.54 mg/kg</td>
<td>10 mg</td>
<td>100 mg</td>
</tr>
<tr>
<td>S-ketamine</td>
<td>0.72 mg/kg</td>
<td>30 mg</td>
<td>90 mg</td>
</tr>
</tbody>
</table>

- mean ratio of propofol + S-ketamine = 1 : 1.5

- this corresponds to a ratio of propofol + ketamine = 1:3 !

- less seizure quality was predicted by age and dose of propofol
ketofol:

- reduce dose of k and dose of p
- apply p and then k
- use $p : k = 1 : 3$ (OR $p : S-k = 1 : 1.5$)
ECT – anesthesia:

1. Typical anesthetic drugs (p, e, m, t)
   a. Ketofol – two stones to catch one bird ?

2. Some typical problems / solutions …
   a. ASTI (anesthesia-to-stimulation time interval)
   b. Oxygen
   c. PAS / PIA
   d. Cardiac
ECT – anesthesia: Dosing or Timing?

bispectrum (BIS) as a surrogate of the depth of the induced anesthesia
ECT – anesthesia: Dosing

- dose = 0 (at unmodified ECT) results in post ictal agitation (PIA) rates of 10-50% 
  (Andrade, Shah, Tharyan et al., Indian J Psychiatry. 2012)

- PIA is in an individual patient perfectly predicted by BIS
  (Kranaster, Janke, Hoyer, Sartorius, J ECT. 2012)
  (Janke, Hambsch, Bumb, Kranaster, Thiel, Sartorius, Aksay, ANIN 2017)

⇒ lower doses of anesthetic are not a good solution
ECT – anesthesia: Timing !!!

- ECT anesthesia: the lighter the better?
  Sartorius A, Muñoz-Canales EM, Krumm B, Krier A, Andres FJ, Bender HJ, Henn FA.

- The Anaesthetic-ECT Time Interval in Electroconvulsive Therapy Practice--Is It Time to Time?

- The influence of the anesthesia-to-stimulation time interval (ASTI) on seizure quality parameters in electroconvulsive therapy.
  Jorgensen A, Christensen SJ, Jensen AEK, Olsen NV, Jorgensen MB.
ECT – anesthesia: ASTI

propofol / thiopental / methohexital

succinylcholine

1-2 mins

2 - 3 mins

> 4 mins
ECT – anesthesia: Oxygen

What’s the difference between grand mal seizures and ECT?
ECT – anesthesia: Oxygen

35-year-old patient with refractory temporal lobe epilepsy.

MR shows subtle hyperintensity of the left hippocampus on the axial FLAIR (blue arrow) and atrophy of the left hippocampus on coronal images (yellow arrow).
ECT induced grey matter volume increase

Electroconvulsive therapy induced gray matter increase is not necessarily correlated with clinical data in depressed patients.

Volume of the Human Hippocampus and Clinical Response Following Electroconvulsive Therapy.
ECT induced grey matter volume increase

Sartorius A, Demirakca T, Böhringer A, Clemm von Hohenberg C, Aksay SS, Bumb JM, Kranaster L, Ende G.
Electroconvulsive therapy increases temporal gray matter volume and cortical thickness.
Eur Neuropsychopharmacol. 2016 Mar;26(3):506-17

Volume of the Human Hippocampus and Clinical Response Following Electroconvulsive Therapy.
Figure 2: Mean time course of transcutaneously measured pCO₂ and pO₂ level. The mean onset of (pre-)oxygenation, muscle relaxation, start of ECT and 2 minutes post ECT are labeled.

New Evidence for Seizure Quality Improvement by Hyperoxia and Mild Hypocapnia.
Aksay SS, Bumb JM, Janke C, Hoyer C, Kranaster L, Sartorius A.
J ECT. 2014 Mar 12.
New Evidence for Seizure Quality Improvement by Hyperoxia and Mild Hypocapnia.
Aksay SS, Bumb JM, Janke C, Hoyer C, Kranaster L, Sartorius A.
J ECT. 2014 Mar 12.
ECT – anesthesia: Oxygen

win –win:

- $O_2$ makes the procedure safe
- $O_2$ lowers seizure threshold

Charles Kellner: “The green gas is the good one!”
ECT – anesthesia: PAS and PIA

Or problems with “movements” peri-ECT ...
Rare side effect of propofol:

A rare side effect of propofol: acute restless legs syndrome pre ECT
Also possible after flumazenil in the recovery room post ECT
Myocloni frequently seen with etomidate and sometimes even with S-ketamine (not a seizure – as formerly suspected!)
Typical fasciculations due to succinylcholine
post anesthetic shivering (PAS)
Shivering
Another shivering
snooring
Postanesthetic shivering (PAS) is shivering after anesthesia is not fasciculating, is not myocloni, is not restless legs!

The intensity of PAS may be graded using the scale described by Crossley and Mahajan:

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No shivering.</td>
</tr>
<tr>
<td>1</td>
<td>No visible muscle activity, but one or more of piloerection, peripheral vasoconstriction or peripheral cyanosis (other causes excluded).</td>
</tr>
<tr>
<td>2</td>
<td>Muscular activity in only one muscle group.</td>
</tr>
<tr>
<td>3</td>
<td>Moderate muscular activity in more than one muscle group, but not generalised shaking.</td>
</tr>
<tr>
<td>4</td>
<td>Violent muscular activity that involves the entire body.</td>
</tr>
</tbody>
</table>

The intensity of postoperative shivering is unrelated to axillary temperature.
Crossley AW, Mahajan RP.
Anaesthesia. 1994 Mar;49(3):205-7
1. clonidine
2. dexmedetomidine
3. mivacurium instead of succinylcholine
4. probably more often with barbiturates / propofol and less with ketamine

What is the place of clonidine in anesthesia? Systematic review and meta-analyses of randomized controlled trials.
Sanchez Munoz MC, De Kock M, Forget P.

Systematic Quality Assessment of Published Antishivering Protocols.
Choi KE, Park B, Moheet AM, Rosen A, Lahiri S, Rosengart A.

Efficiency and safety of ondansetron in preventing postanaesthesia shivering.
He K, Zhao H, Zhou HC.

Effectiveness of dexmedetomidine use in general anesthesia to prevent postoperative shivering: a systematic review.
Hoffman J, Hamner C.
ECT – anesthesia: post ictal agitation (PIA)

- dose = 0 (unmodified ECT) results in post ictal agitation (PIA) rates of 10-50% (Andrade, Shah, Tharyan et al., Indian J Psychiatry. 2012)

- PIA is in an individual patient perfectly predicted by BIS (Kranaster, Janke, Hoyer, Sartorius, J ECT. 2012)
  (Janke, Hambsch, Bumb, Kranaster, Thiel, Sartorius, Aksay, ANIN 2017 )

⇒ lower doses of anesthetic are not a good solution
ECT – anesthesia: post ictal agitation (PIA)

- Do not restrain !!! (=> otherwise increase of PIA)

- Keep everything calm and use as less physical limitation as possible

- Self limiting in most cases within 20 mins

- Severe forms: Escalate staff

- Severe forms: Use i.v. diazepam e.g. 10mg

- Increase dose of anesthetic next ECT
ECT – anesthesia: cardiac
hypersalivation / sialorrhoë

- Former times: atropine, which is basically obsolete. Why?
- Today: glycopyrrolate as muscarinic receptor antagonist

- Both reduce hypersalivation (parasympatholytic)

- atropine reduces initial bradycardia, but increases ictal hypertension *

* Psychiatry Res. 2019 Jan;271:239-246
Electro convulsive therapy: Modification of its effect on the autonomic nervous system using anti-cholinergic drugs.
Christensen STJ, Staalsø JM, Jørgensen A, Weikop P, Olsen NV, Jørgensen MB.
ECT – anesthesia: cardiac

The Brady Bunch: A Montage of Typical Sinus Pauses in Electroconvulsive Therapy.

Kellner CH, Paparone P
Asystolia appears shorter in our printout (printout starts at the end of charge delivery!)
Incidence of post-stimulus asystole

- > 50% !!!

- conclusion:
  1. frequent! (probably very physiologic, low risk)
  2. BIL > RUL > BF
  3. age
The effect of electrode placement and pulsewidth on asystole and bradycardia during the electroconvulsive therapy stimulus

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\textsuperscript{2} The Northside Clinic & Wesley Hospital, Sydney, Australia
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\textsuperscript{4} Black Dog Institute, Sydney, Australia
\textsuperscript{5} Department of Anaesthesia and Pain Management, Royal North Shore Hospital, Sydney, Australia

Table 3. Asystole during ECT stimulus

<table>
<thead>
<tr>
<th>Covariate</th>
<th>Pulsewidth</th>
<th>Electrode placement</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B</td>
<td>S.E.</td>
</tr>
<tr>
<td>Electrode placement</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RUL vs. BF</td>
<td>5.334</td>
<td>1.333</td>
</tr>
<tr>
<td>RUL vs. BT</td>
<td>2.158</td>
<td>0.774</td>
</tr>
<tr>
<td>BT vs. BF</td>
<td>3.176</td>
<td>1.348</td>
</tr>
<tr>
<td>Pulsewidth</td>
<td>1.0 vs. 0.3</td>
<td></td>
</tr>
</tbody>
</table>
Pacemaker / intracardial defibrillator (ICD)
Pacemaker and stimulation (movie)
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