Physiological effects of ECT
This is where I work

Not where ECT is done usually
Broad Oak Hospital Liverpool (Mersey Care)

- Cottage hospital setting (no acute physicians or intensive care)
- At least 500 treatments a year
- Most done at Broadoak but some patients done at RLUH.
Aims of Today

- Physiological effects of ECT
- Physiological effects of Anaesthesia
- Effects that patient comorbidity has Broadly speaking)
- Effects that psychiatric drugs on Physiology (relevant to anaesthesia)
- What does the anaesthetist want to know about patient (what can the psychiatrist tell the anaesthetist)?
Scope of a problem

- ECT is inherently safe (1-3/100,000 mortality)

This does not mean that we should be complacent:

- ECT is more commonly prescribed in the older population
- Comorbidity is more common in older people
- Physiological extremes more likely in older people
- ECT is repeated up to 12 times (or more), therefore anaesthesia is repeated, therefore risk is repeated.
- Also Consider morbidity of ECT.
Function of Anaesthetist in ECT

- Provide anaesthesia: Amnesia
- Modify muscular component of seizure (clonic can be abolished almost completely. Tonic less so)
- Effective Oxygenation
- But also need to support changes in physiology that we measure
Anaesthetic drugs used

- Oxygen
- Induction agents: Propofol, Etomidate, Thiopentone, Ketamine
- Muscle relaxant: Suxamethonium, Rocuronium/sugamadex (expensive!)
- Analgesia: Alfentanil, Remifentanil (short acting)
- “Rescue drugs and fluids”
Physiological effects of Anaesthesia

**CVS**

- Hypotension (worse if dehydrated or cardiac disease)
- BP better maintained if using Etomidate (Ketamine)
- Possible Bradycardia

**RS**

- Lose control of airway
- Apnoea, hypoxia
- Hypoxia and compromised airway is more likely if patient is morbidly obese or has acute respiratory problem
Physiological effects of Anaesthesia

CNS

* Raise seizure threshold (Propofol). Use enough to produce anaesthesia
* Reduced cerebral oxygen consumption and blood flow CBF: Propofol, Etomidate
* Increased CBF and oxygen consumption (Ketamine)

other

* Muscle pain (suxamethonium)
* Rise in potassium level (Suxamethonium) ... Catatonia
* Increased IOP (Sux again!)
* Adverse Drug reaction
**Physiological Effect of ECT**

- Autonomic effects:
- Initial stimulus:
- Direct stimulation of the hypothalamus
- Parasympathetic outflow (Vagus)
- Bradycardia
- Asystole (more common in young and fit,)
- More likely to be persistent if no clonic phase or unilateral stimulus
- More common in first tx (dose titration)
- Hypotension due to anaesthesia may be attenuated at this point
- May also get a sudden decrease in heart rate at end of tonic phase
Autonomic effects of ECT

- Sympathetic NS stimulated directly during clonic phase (Via spinal cord).
- Increased NA and A levels
- Tachycardia
- Hypertension and increase in venous pressure
- Worse if using Etomidate or Ketamine
- Oxygen demand in heart increased and supply of oxygen needs to increase otherwise Ischaemia develops.
- Continues after ECT and into recovery phase
- Transient arrhythmia
- Piloerection and pupillary dilation
CNS effects of Seizure

- Increased oxygen consumption during seizure
- Increased cerebral blood flow and cerebral vasodilatation. Increased blood volume
- Skull has a fixed volume and therefore if blood volume increases, ICP increases. (Monroe Kellie doctrine)
- Brain compensates
- ICP increases more rapidly if ICP raised already increased (e.g., mass lesions)
- After ECT, CBF is reduced a while later (postulated mechanism of action)
- Increased intraocular pressure
- BUT induction agents reduce oxygen consumption and CBF
CNS effects of seizure

- Brain dependent on oxygen and glucose (limited anaerobic capacity)
- Brain maintains blood flow through a range of pressures to maintain oxygen delivery
- In normal circumstances, autoregulation is probably maintained during ECT unless severe hypo or hypertension (but we don’t know)

![Graph showing autoregulation of cerebral blood flow](image-url)
Effect of hyperventilation

- May prolong fit
- Anaesthetist can reduce EtCO₂ by hyperventilation. (if they have the means to do so)
- Reduces CBF and oxygen delivery.
- May not be a good thing if brain is relatively hypoxic
Neuro endocrine effects

Hypothalamic Pituitary axis

- CRF and ACTH increased with increased cortisol (about one hours) ? Stress related
- TSH increases after treatment (one hour)
- Prolactin (about one hour)
- But No effect on Growth Hormone , FSH or LH
- ? increase in 5HT pathway Serotonin
- Is it clinically relevant ?
- No evidence that increases in levels correlate with therapeutic response
Drugs used in psychiatry problems for ECT anaesthesia

Interaction with anaesthetic drugs or accentuate CVS/RS side effects of anaesthesia and ECT

- Anticholinergic side effects (TCAs, antipsychotics)
- Prolonged QTc (TCAs and antipsychotics)
- Cardiac toxicity (clozapine)
- Decreased T4 (lithium)
- Hepatotoxicity and interaction with drugs (MAOI)
- Hyponatraemia (SSRI)
- Weight gain (all)
Treatment of physiological compromise;

Cardiac most common problem

BRADYCARDIA

Give Atropine or Glycopyrollate

- If Bradycrdia persistent
- If rate of change is significant
- If previous hx of Bradycardia with ECT
- Sometimes use this on first treatment.
- No evidence that patients taking B blockers more likely to cause a Bradycardia that needs treating

HYPOTENSION

- Vasoconstrictors or sympathomimetic agents
- fluids
Cardiovascular effects: treatment

TACHYCARDIA AND HYPERTENSION

Treat if persistent or predictable

- Can treat before start of anaesthesia

- Use β blockade. Esmolol has been used and attenuates out hypertension/tachycardia but doesn't abolish it. Also may shorten fit which may reduce efficacy of ECT. Therefore don't use it routinely. We use Atenolol

- Remifentanil has been used again does not abolish. (Max 100Ug). If use more leads to prolonged recovery. And hypotension.

- Arrhythmias: most do not need treating
Who is more likely to be at risk of cardiac problems during ECT?

- Normally only 7.5% most do not need treating BUT….
- Recent cardiac event
- IHD LV dysfunction. ECT has been reported repeatedly as safe in patients with stable IHD.
- Valvular disease: Aortic stenosis
- Effect of depression on cardiac function (unstable comorbidities, dehydration)
- Effect of antidepressants on cardiac function
Respiratory problems; Most at risk?

- Acute problem (URTI, unstable COPD may cause airway sensitivity (laryngospasm and wheeze)
- Obese. (also more likely to aspirate)
- Airway problems.
CNS compromise; who is more likely to be at risk?

- Mass lesions (increased ICP)
- Recent stroke (autoregulation)
- Cerebral aneurism (?)
- Most common is agitation or memory issue (dose titration)... Can we predict?

We do not routinely measure effects of cerebral blood flow in ECT (unlike cardiac monitors so we do not treat problems directly and we have limited drugs to treat problems.

Many anecdotal case reports where ECT has been done in CNS compromised patients
Reducing the risk; Role of Psychiatrist

- Some Knowledge of what increases risk of morbidity in ECT
- Importance of psychiatrist being able to obtain clinical information from relatives, other hospitals and GP (deteriorating comorbidity)
- And communicating them to anaesthetist: Usually in the form of a questionnaire to fill in before ECT commences. This is usually delegated to junior doctor.
- Sometimes, verbal communication is best!
- Direct Access to physicians in medicine/ cardiologists to stabilise where appropriate.
- Family should be aware that although ECT carries a small risk, this is increased if they have severe depression, have unstable or severe comorbidity and are dehydrated.
Reducing the risk
Preanaesthetic assessment
History from patient/nurses/psychiatrists history

- Patient can’t always give a coherent history (depression, dementia etc)
- Patient doesn’t always arrive at ECT with anyone who knows about their condition. Psychiatric history very important!
- Access to relatives very useful
- Clues from drug history
- Results from routine screening helps with decision making process
- Problems more likely if patient is prepared for ECT urgently, but this is when patients are most unfit.
Reducing the risk
Decisions for anaesthetist and Psychiatrist

- Is comorbidity stable?
- Is comorbidity likely to cause physiological problems?
- Have I got the bigger picture? Is ECT in patients best interests?
- Refer patient to acute care physician to treat acute problems (e.g. renal failure, dysrythmia). AN EXPERT
- ? Defer ECT until stable (risk vs. benefit)
- ? Treat in acute care setting
- Terminal illness is not a reason not to go ahead

Decision made on a CASE BY CASE BASIS

- Patient Centred Care
- Remember that ECT needs to be repeated.
Conclusion

- Anaesthesia for ECT is inherently safe; short procedure, no fluid shifts.
- Physiological changes need to be monitored where possible. Treated if necessary.
- Most problems with ECT relate to CVS compromise.
- Anaesthetic and psychiatric assessment is aimed at reducing the risk with the knowledge of physiological impact on patient.
- Need to repeat to produce clinical effect.
- Treat patients on a case by case basis (and relatives).
- Communication and a good working relationship with the ECT team is essential to ensure that risks of ECT are minimised.
Acute care setting?

- Access to ICU and AMU
- May delay treatment
- If decision is made to start treatment in an acute care setting, this will need careful organisation
- Can reassess the need for acute care setting after a few treatment sessions
Reducing risk on the day

- Pre treatment assessment (nurse lead) including NBM. Any change form last time?
- AAGBI monitors
- Records of previous anaesthetic and communication
- Skilled staff (inc. appropriate dose titration)
- Recovery
So what?

Risk reduction

* Choice of patients, stable co-morbid state
* Monitors (AAGBI) to treat side effects' of physiology
Assessing the risk
Should we do ECT?

Absolute contraindications are few: Risk benefit assessments

E.g. Recent MI, Severe Aortic stenosis, recent CVA, raised ICP

Having terminal illness does not necessarily preclude treatment

IMPORTANT. Do patient and/or relatives understand risk?
Incidents in ECT

Higher incidence if

* Dehydrated

* Unstable CVS especially; Aortic stenosis, untreated heart failure, uncontrolled hypertension, uncontrolled dysrythmia

* Unstable RS (i.e. acute problem)

* Unstable endocrine; Diabetes, thyroid

* Difficult airway: Morbid obesity, SOL in mouth, neck
Cardiovascular response; treatment

- Often no treatment needed
- Can sometimes preempt problems
- Atropine/Glycopyrollate
- Ephedrine
- Fluids
- Atenolol (B blockers)
Other effects

- Succinylcholine
- Hyperkalaemia (care in catatonia).
- Intragastric pressure (due to fir) and succ
- Intraocular pressure (due to fir as well) and succ
Drugs used in psychiatry

- Effects that can cause problem for patient and anaesthetic during ECT
- NMS, Serotonin syndrome
- QT prolongation and arrhythmia
- Tachycardia (anticholinergic side effects)
- Electrolyte disturbances (hyponatraemia)
- Thyroid and renal function (Lithium):
- MAOI (we cannot give certain drugs)
Common Acute changes which may need treatment

Autonomic

- Bradycardia more likely if no clonic seizure (anticholinergic agents)
- Tachycardia and hypertension (B Blockers)
- Dysrhythmia usually resolves but may need treating
- Hypotension (Fluids, sympathomimetic agents)
- Salivation not usually a problem

RS

- Acute Wheeze

CNS

- Delirium may need treating (midazolam)