CONTEMPORARY UNDERSTANDING OF ECT ANAESTHESIA

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WHAT WILL YOU LEARN FROM THIS PRESENTATION?

- Why anaesthesia is an essential part of modern ECT
- How to deliver high-quality ECT anaesthesia
- What are the major challenges and pitfalls

ABOUT THE SPEAKER

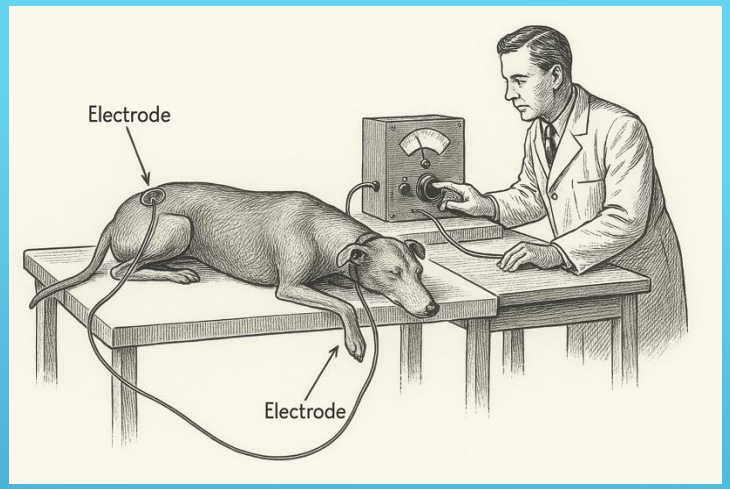
- **Saara Huoponen, MD. PhD.**
  Specialist in Anaesthesiology and Intensive Care
  Special Competence in Medical Education
- Department of Anaesthesiology, Turku University Central Hospital
- Working at hospital's Neuromodulationunit since 2018, when the unit was established
- Doctoral dissertation **"Anaesthesia for electroconvulsive therapy in Finland – Variation in clinical practices and the management of adverse effects"**

- No commercial or political conflicts of interest in terms of today's topics

CONVULSIVE THERAPY – THE PREDECESSOR OF ECT

- Developed by Ladislas Meduna in the 1930s in Hungary
- Made an observation that epilepsy and schizophrenia rarely coexisted
- Induced seizures using camphor and later cardiazol (metrazol)
- Treatment was effective but it caused extreme fear and physical risks to the patients
- These findings inspired Italian doctors Ugo Cerletti and Lucio Bini to seek an alternative method to induce seizures

EARLY EXPERIMENTS AND SAFETY DEVELOPMENT



- 1930s: Ugo Cerletti and Lucio Bini conducted animal experiments
- Electrodes were initially placed on the mouth and rectum of the animal to induce seizures
- This setup caused cardiac arrhythmias and occasional cardiac arrest
- Led to development of bilateral temporal electrode placement
  - Electrical current went through the brain, but not the heart
  - Improved safety

SHORT HISTORY OF ECT

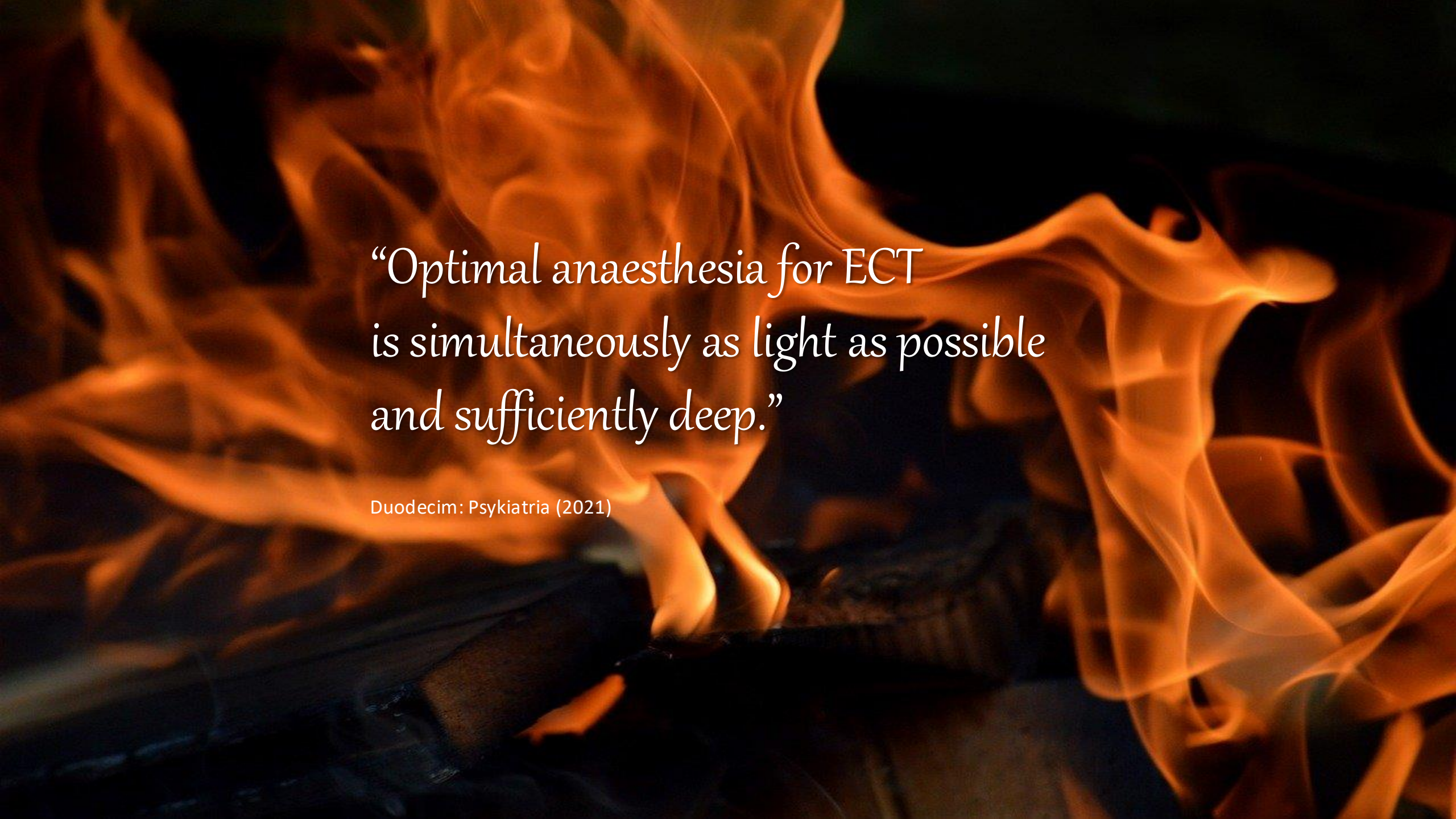
- 1938: First ECT was given to a human by Cerletti and Bini
- Although ECT was effective, the motor seizures were intense, leading to musculoskeletal injuries, fractures, and dental trauma in up to 50% of patients.
- 1951: Introduction of curare (neuromuscular blocking agent)
- Later in the1950s: Suxamethonium replaced curare
- 1960s: General anaesthesia became standard care in many ECT units

- Modern ECT: brief, controlled, safe, humane procedure

WHY DO WE NEED ANAESTHESIA IN ECT?

- Enables the use of neuromuscular blocking agents (NMBAs) for muscle relaxation
  - NMBAs prevent injuries during convulsions

- Ensures unconsciousness and amnesia
- Reduces fear and anxiety
- Reduces cardiovascular stress


“Optimal anaesthesia for ECT
is simultaneously as light as possible
and sufficiently deep.”
Duodecim: Psykiatria (2021)

WHY DOES THE DEPTH OF ANESTHESIA MATTER?

- All anesthetic agents affect the therapeutic component of ECT—the convulsion.
  - They increase seizure threshold and shorten seizure duration
- If anesthesia is too deep → short seizure (or no seizure at all)

Which medications can be used to induce anesthesia for ECT?

Drug	Plusses / main effects	Minuses	Practices at TYKS Neuromodulation unit
Methohexital (GABA-A – agonist barbiturate)	Fast recovery (4–7 minutes), less bradycardia and extrasystole than with sodium pentothal	Availability is occasionally limited, requires dilution. Limited familiarity among anaesthesiologists.	Primary choice
Propofol (likely complex GABA-A –mediated effects)	Familiar to all anaesthetists, decreases post-ECT hypertension, prevents and treats agitation, fast and short acting → fast cognitive recovery. Fast to prepare for use.	Shortens EEG-seizures, more energy is needed to achieve quality seizures which may increase memory side effects. Injection pain.	If more seizure suppression is needed or the patient is very restless and/or aggressive after ECT or if they present with intense PENV despite prophylaxics
Propofol & esketamine (NMDA-receptor antagonist) aka "ketofol"	Smaller doses of propofol → less depression of breathing and less hypotension plus less suppression of seizures. Relaxation of smooth muscle in airways. Increased antidepressive effect?	esketamine: hallucinations, increased salivation, can increase QT interval pre-disposing to arrhythmias?, increases cardiac oxygen consumption, more PENV, vertigo.	Selected patients with depression, currently a rare choice at TYKS
Ethomidate (nonbarbiturate GABA-A - agonist)	Suppresses seizures the least — good when struggling to achieve sufficient seizures.	Myoclonus, slower recovery and more tachycardia, hypertensions and PENV than with methohexital. Suppression of cortical adrenal hormone production, though likely not an issue with ECT. Injection pain.	For patients in whom methohexital suppresses seizures too much
Thiopental (complex GABA-A –agonist barbiturate)	Recovery as fast as with propofol, suppresses seizures less than propofol (but more than methohexital)	Requires dilution. Releases histamine. Onion taste in mouth.	Used to be the primary choice, now used only if methohexital is temporarily unavailable on the market

NEUROMUSCULAR BLOCKING AGENTS (NMBAS)

- Suxamethonium
  - Depolarizing NMBA
  - Rapid onset (30–60 s) and short duration (~5 min)
  - Contraindications: hyperkalaemia, neuromuscular disorders, prolonged immobilization or chronic bed rest…
  - Adverse effects
    - Bradycardia
    - Fasciculations and muscle rigidity → postoperative myalgia
    - Risk of malignant hyperthermia
    - Increased plasma potassium

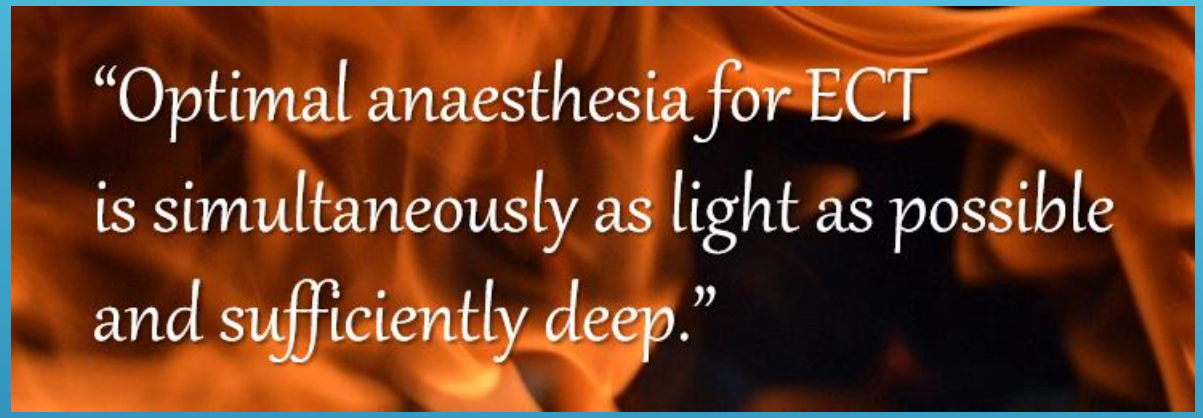
NEUROMUSCULAR BLOCKING AGENTS (NMBAS)

- Rocuronium
  - Non-depolarizing NMBA
  - In the context of ECT, reversal with sugammadex is required
  - The depth and recovery of neuromuscular blockade needs to be monitored!!!
  - Costlier alternative
- Good option when suxamethonium is contraindicated

ADJUVANTS IN ECT

- Glycopyrrone for prevention of bradyarrhythmias and excessive salivation
- 5HT blockers/betamethasone/DHBP for prevention of PENV
- NSAID/paracetamol for prevention of myalgia and headache
- Labetalol for prevention of hypertension
- ECT as a procedure is painless – opioidanalgesia is not needed

WHICH ANESTHETIC AGENT SHOULD BE USED IN ECT?



Use whatever you like—as long as the anesthesiologist knows the goal!

WHY IS THIS SO DIFFICULT?

- Light anesthesia goes against the principles anesthesiologists are trained to follow
- Neuromodulation units lack a dedicated anaesthesia team
- Anesthesiologists may not be aware that inappropriate anesthesia can reduce the effectiveness of ECT

The challenge is to find a dedicated anesthesiologist!

WHAT ARE THE PITFALLS?

- Light anaesthesia → How can we tell if the patient is awake?
- One size rarely fits for all when it comes to anaesthetics
  - Adverse effects and patient characteristics should guide the choice
- ECT is fast-paced, quick turnaround: risk for medication errors
  - Up to 15 patients in 3-4 hours

CONCLUSIONS

- Anaesthesia is an essential part of ECT: it improves the safety of the treatment and helps manage adverse effects.
  - But it can also compromise the treatment if we're not careful.
- To really get the benefits, we need dedicated anesthesia staff and close collaboration with psychiatry.

Let's spread the message: ECT anaesthesia matters!