Electroconvulsive therapy

Electroconvulsive therapy (ECT) is a minimally invasive stimulatory biological treatment method for neuropsychiatric diseases, in which a modulated electric current is applied under general anaesthesia with scalp electrode muscle relaxation in order to influence the patient's psychopathology. In some indications, it is the only life-saving (ultimum refugium) treatment modality.

Today, they are successfully used in psychiatry mainly for the treatment of:

1. to treat resistant **moderate to severe depressive episodes** (major depressive disorder, organic depressive disorder);
2. for the treatment of resistant **depressive phase** (bipolar affective disorder, schizoaffective disorder);
3. for the treatment of resistant **manic phase** (bipolar affective disorder, schizoaffective disorder);
4. severe psychotic depression and mania (eg. stupor);
5. for the treatment of refractory psychosis (schizophrenia, organic psychotic disorders);
6. **catatonia**;
7. mania delirans;
8. suicidal tendencies otherwise unsolvable;
9. an agitated, aggressive patient who does not respond to other treatment;
10. neuroleptic malignant syndrome;
11. status epilepticus;
12. Parkinsonism with "on-off phenomena";
13. tardive dyskinesia;
14. delirium;
15. in case of failure of pharmacological treatment;
16. at the request of the patient in maintenance treatment.

Electroconvulsions are not effective: in anxiety disorders, obsessive compulsive disorders, post-traumatic stress disorder, personality disorders, eating disorders, addiction syndrome, and withdrawal, autism, mental retardation, and dementia.

General characteristics of ECT

This is a biological modality in treatment - the so-called **shock or convulsive method** = the principle is the induction of an epileptiform seizure (tonic phase - when an electric current passes through scalp electrodes and clonic phase - reminiscent clinically and on an EEG grand mal seizure). The first use dates back to the 1930s, when Ugo Cerletti and Carl Bini (Italians) successfully used ECT to treat a patient with catatonic schizophrenia.

- **Advantages:**
  - rapid onset of action (clinical effect visible after 3-4 applications);
  - unusual efficacy (almost 90% response to treat resistant depressive conditions);
  - high safety (mortality only 1-4 per 100,000 operations);
  - minimal complications (transient cognitive impairment, 7-10% delirium);
  - cognitive impairment as the main side effect is in 98% fully reversible within 6 months, in the vast majority of patients within 6 weeks detectable only by specialized tests;
  - does not affect brain morphology (no "scar" occurs, although it affects inflammatory mechanisms);
  - does not cause permanent personality changes (see CIA MkUltra (https://en.wikipedia.org/wiki/MKUltra) program in the 1950s);
  - affects amnesia on the exercise itself (short anterograde amnesia).

- **Disadvantages:**
  - Social stigmatisation (the view of the 'film spectator', eg. Forman's Flight over the Cuckoo's Nest or Requiem for a Dream);
  - views on the punishment of the sick;
  - the public believes that this is an obsolete method reserved for the "greatest fools".

Personnel and material equipment

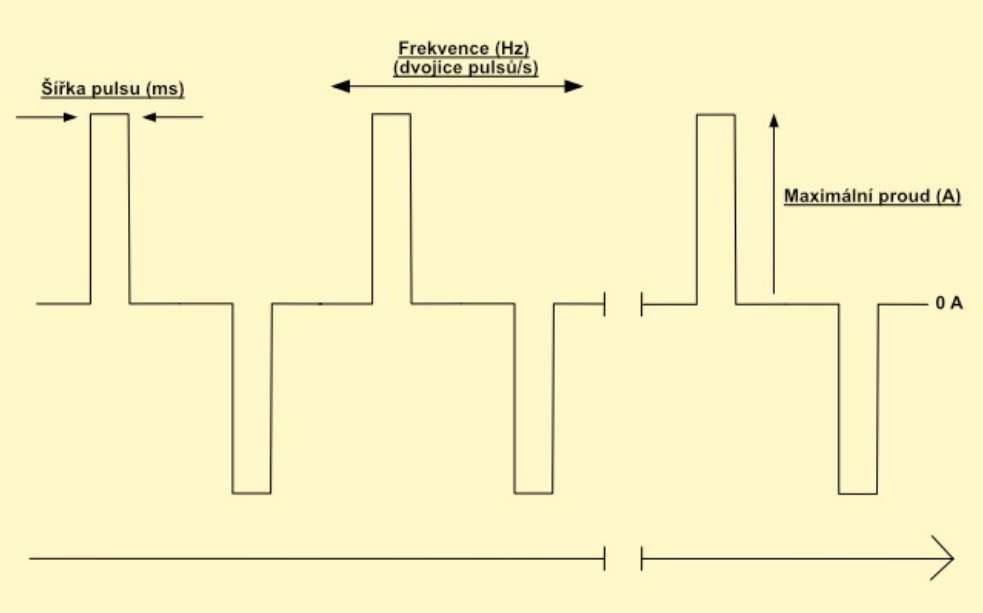
- inpatient (or outpatient) psychiatric facilities;
- anaesthesiologist (and an anaesthesiology nurse) - is responsible for the course of anaesthesia;
- trained psychiatrist (and an "electric" nurse, nursing staff) - responsible for performance;
- laboratory with anaesthesiology equipment, electroconvulsive device, dormitory with a monitor of physiological functions.

Modified ECT

Currently, the so-called **modified electroconvulsive therapy** is used. Before its introduction, there were many complications and side effects (a sinusoidal current of 50 Hz from the mains socket has a neurotoxic effect). Epileptiform paroxysm is induced under general anaesthesia and partial muscle relaxation by the application of a modulated (ultrashort pulse method) alternating current between two scalp electrodes (located unilaterally above

the non-dominant hemisphere, bifrontally or bitemporally) with a current of 0.5–0.9 A with a frequency of 20–120 Hz, pulse width 0.25–1.5 ms, for 0.2–8 s. The voltage depends on the dynamic impedance (generally the resistance of the alternating current during the passage through the conductor). Most of the charge passes through the place of least resistance (locus minoris resistentiae) - the scalp.

Ultrashort pulses are the result of zero-mean AC modulation into sinusoidal rectangular pulses **with a base width ≤ 0.3 ms**. In general, pulse width correlates with cognitive side effects (shorter pulse leads to less disability).



Ultrashort pulse method

Interesting fact: the total discharge energy is low. At the **maximum dose** of a modern device with stimulation parameters: frequency 120 Hz, delivery time 8.0 s, pulse width 0.75 ms at a current of 0.8 A, the size of the delivered charge is **1152 mC**. At an impedance of 1000 Ω, the voltage is 400 V and the power of the device is 320 W, but the total energy is only 460 J, ie the energy needed to heat 108 g of water by 1 ° C, or 5 kg of water by 0.02 ° C.

There is no evidence of thermal changes in brain tissue following a safety protocol. There is also no electrolysis due to the nature of the alternating current.

The lowest dose to induce tonic-clonic seizures is **6-19 mC**.

Energy titration

The most common side effects of treatment are transient and reversible cognitive impairments - memory impairment (especially autobiographical), dyssexecutive syndrome and pseudo-frontal syndrome. In an effort to minimize these side effects, the ultrashort pulse method was invented. Another way to improve the procedure is the so-called energy titration, which is the procedure of lege artis and "state of the art" within the individualised EBM. The lowest effective discharge energy is sought to induce sufficient convulsion to minimise cognition failure and other complications.

Factors affecting the threshold for triggering an epileptiform seizure

Factors	Raising the Threshold	Lowering the Threshols
Age	eldery	younger
Sex	men	women
State	dehydration hyposaturation	hypocapnia
Brain Disease Brain Area	non-irritating diffuse frontal	irritating limbic
Medication	anesthetics benzodiazepens antiepiletics barbiturates beta-blockers	pentylenetetrazol vasopressing tricyclic AD pheothiazines clazapine lithium reserpine theofylin withdrawal state(alcohol, bzd)
Electrode Placement	bitemporal (bifrontal)	unilateral
Static Impedance Dynamic Impedance	high (bad contact) 0	low (good conductivity) reduces proportionally
Pulse width	longer (>0.5ms)	shorter (ultra short <0.3ms)
Seisure Frequency	during the last days/months	

Side effects

- **formerly common** long bone fractures, vertebral compression fractures, temporomandibular joint dislocation and large joints (premedication: curare - had serious respiratory side effects, succinylcholine is now used successfully and safely;
- **before the introduction of ultrashort pulses** - the sinusoidal current from the network has a neurotoxic effect, ultrashort pulses on the contrary have a neuroprotective effect;
- complications due to general anesthesia (propofol, thiopental, etomidate or metohexital);
  - respiratory arrest, apnea pause, cardiovascular failure;
  - aspiration of secretions from the upper respiratory tract (prevention is premedication with **atropine**);
- cognitive disorders (memory disorders, acute confusion = delirium, dyssexecutive syndrome) - fully reversible in 98% of patients within 6 months, in the vast majority of patients within 6 weeks; the vast majority of cognitive domain disorders are detectable only in specialized neuropsychological tests;
- prolonged seizure (longer than 60 s - must be stopped by i.v. application of benzodiazepines);
- transient mild headache, transient confusion (delirium);
- **if the safety procedures are not followed**, there is a risk of burns (low or high "resistance"), fractures (in case of insufficient muscle relaxation), bruises during the patient's massive grip, nail damage and tongue bites during the tonic phase, etc. ***(very rare)***

Contraindications

- **Absolute** - Some authors state that ECT has no absolute contraindications.
  - Severe intracranial hypertension (suspected ophthalmic consultation - papillary oedema).
- **Relative** - does not hinder the performance itself, special caution is needed (to illustrate the load on the body is very similar to getting up to the second floor faster).
  - conditions excluding general anaesthesia (ASA classification);
  - severe cardiovascular disease (arrhythmias, severe heart failure, severe valve defects, unstable angina, myocardial infarction in the last 3 months, aortic aneurysm);
  - a recent (within three months) head injury;
  - fresh intracranial haemorrhage;
  - fresh cerebral infarction and cerebral oedema;
  - general condition (coagulation disorder, severe liver disease, pheochromocytoma, severe respiratory insufficiency);
  - severe osteoporosis and osteopenia, fractures;
  - implanted pacemaker (can be temporarily switched off in cooperation with cardiologists).

- The following are **not contraindicated**: epilepsy, older or adolescent age, pregnancy (there is a need for an obstetrician with a cardiograph before and during the procedure, fetal lethality is 7%).
- An **alternative to ECT** is the use of repetitive transcranial magnetic stimulation (rTMS), which does not require general anaesthesia but is not as effective.

The course of the modified ECT

- **The patient goes to ECT:**
  - correctly indicated without contraindications;
  - after undergoing "preoperative internal examination" before general anesthesia, ev. with additional conciliar examinations (neurological, ophthalmological, radiodiagnostic);
  - informed and educated;
  - with signed informed consent (except for the so-called vital indications - conditions where it is a life-saving procedure or conditions where the patient is unable to understand the importance of treatment);
  - from midnight prepared hungry, non-smoking and premedicated before the procedure (atropine, possibly internal medication - insulin, antihypertensives, levothyroxine, etc.);
- **Method:**
  - the patient is on an empty stomach from midnight before the procedure, receives his chronic (non-psychiatric medication) in the morning and is given **atropine**;
  - the patient is taken by staff from the ward to the premises of the Electroconvulsive Treatment Laboratory;
  - pre-oxygenation and connection of monitoring (ECG, EEG, saturation - values are recorded during the whole procedure) and stimulation electrodes;
  - securing venous access;
  - administration of anesthetic i.v. (thiopental, propofol, abroad also etomidate or metohexital), no intubation is required, the whole procedure does not take more than 2 minutes;
  - muscle relaxants (succinylcholine, or suxamethionium) State Office for Drug Control: suxamethionium;
  - muscle relaxation is expected (manifested by muscle twitches - fasciculations - with rostrocaudal propagation);
  - static impedance range control (200-3000 Ω);
  - discharge application according to selected parameters;
    - there are types of access (two types of device) - one has a **single optional parameter - energy** (given in percent, where the maximum dose corresponds to 550 mC; measures only static impedance) and more sophisticated device with greater variability of selected parameters with regard to minimising side effects and maximising safety patient - **energy titration** (pulse width, pulse frequency, pulse amplitude, application length; periapplication dynamic impedance monitoring);
  - paroxysm itself;

1. during the passage of electric current (max. 8 s) **tonic phase** (sometimes deep exhalation, bite of jaws, face flushes, mydriatic pupils, body tense - lower limbs - flexion, upper limbs - extension);
2. then the **clonic phase** (resembling a grand mal seizure);
3. subsequently the effect of anesthesia subsides, the patient breathes spontaneously and consciousness returns slowly → to sleep;
4. also one-hour post-exercise observation (dormitory) with monitoring of physiological functions;

- **The patient returns accompanied by staff to the ward** fully oriented, cardiopulmonary stabilised, normosaturated, where they eat breakfast and take morning medication.

ECT applications

1. **Titration phase** - minimal energy is applied, which triggers a clonic seizure with corresponding ictal activity on the EEG (20–60 s), during one session it is possible to repeat the application 3-5 times a day; (The second less suitable is the empirical titration of energy according to the patient's age).
2. **Acute phase** - classically **3 times a week** (Monday - Wednesday - Friday) to a **total of 8-20 applications** (usually 12); rarely **cumulative** (daily in life-threatening conditions of lethal catatonia, mania delirans, neuroleptic malignant syndrome).
3. **Continuation phase** - after the end of the acute phase, 14 days after the next application (the series should not be terminated abruptly, it reduces the relapse rate).
4. **Maintenance phase** - at various intervals (month, 3 months) outpatient application (the patient leaves home in the afternoon).

Mechanism of action of ECT

Among the hypotheses that, although partially, explained clinical effectiveness, or which continue to co-explain it in the light of the science of the beginning of the 21st century, we can state:

1. "Convulsive" (epilepsy that eliminates melancholy, psychosis);
2. "Anticonvulsant;"
3. classic monoamine (affecting the neurotransmitters dopamine, serotonin and noradrenaline);
4. changes in acetylcholine transmission;
5. inhibitory (gamma-aminobutyric acid);
6. excitatory (glutamate);

7. affecting the endogenous opioid system;
8. affecting tryptophan-kynurenine metabolism;
9. neuroendocrine (thyrotropin releasing hormone, neuropeptide Y, substance P);
10. endocrine (growth factor, cortisol, prolactin, vassopressin);
11. immune (affecting the neuropil - glia or acute phase reactants, leukocyte migration, mast cells, changes in the blood - brain barrier);
12. neuroprotective effects - changes in subclinical inflammatory processes in the central nervous system (pancreatic polypeptide, C-peptide, natriuretic peptide B, leaching interleukin 6, tumor necrosis factor alpha; suppressed interleukin 8 and 13, endothelial growth factor, insulin-like growth factor I, myeloperoxidase, sortilin-1, interferon gamma);
13. neuroneogenetic (demonstrated in localizations: hippocamus, habenula, amygdala, anterior cingulum, temporal and frontal neocortex, hypothalamus; mediated by brain growth factor, vascular and glial growth factor leaching, p11 protein deregulation);
14. induction of molecules preventing the deterioration of immature neuronal tissue (activated T-lymphocytes);
15. hypotheses including synaptic and dendritic plasticity, influence on arborization;
16. affecting the interconnection of the (pre) frontal and temporal cortex;
17. changes in cortico-subcortical communication and plasticity;
18. neurophysiological changes (cordance in frontal and cortico-subcortical areas);
19. changes in global brain metabolism (hypermetabolic state followed by functional suppression, changes in regional blood flow and glucose consumption);
20. diencephalothamalamic changes;
21. early response gene activation hypotheses;
22. activation of genes directly responsive to the passage of electric current through the cell (Bienenstock-Cooper-Munro theory) with influence on antiapoptotic processes;

And many others. It turns out that genetic polymorphism also plays an important role in the effectiveness and tolerability of treatment, the occurrence of side effects. Electroconvulsive therapy has its irreplaceable place in the psychiatric armament and the current modifications make it one of the most effective and safest methods of treatment in medicine.

Conclusion

The paradigm still applies: Without motor manifestations of tonic-clonic seizures (which are not only affected by energy dose, anaesthetic dose, use of benzodiazepines, anti-epileptics and other drugs and depth of muscle relaxation), are electroconvulsions abortive and have no clinical effect? Is it worth monitoring the single-channel EEG of both hemispheres and marking a paroxysm shorter than 20s as abortive?

Will research by FEAST and MST, rTMS and tDCS and other biological modalities in conjunction with state-of-the-art knowledge not only of neuroimaging disciplines lead to the sought-after "unified theory of the mechanism of electromagnetism on human psychopathology"?

Links

Related articles

- Biological treatment methods in psychiatry

Source

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