ANAESTHESIA AND ECT
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What are the current areas of interest in ECT anaesthesia? I suggest four. First, how should approach the current literature on ECT anaesthesia. Second is there a case to use either thiopentone or propofol as the drug of choice. Thirdly, what is the role of remifentanil in modern ECT and lastly can we do anything to help reduce the degree of post-operative cognitive impairment?

Literature analysis
What does the literature say about the selection of an induction agent for ECT? It is in fact a very difficult question since most of the studies are of limited benefit because of their study design in terms of outcome measurements. While seizure duration is helpful, it is by no means the best measure of ECT outcomes. Another problem is that most studies use methohexitone as the “gold standard” study drug - as it is still widely used in the US, but not readily available in Australia.

Choice of Induction agent
Thiopentone possesses significant anti-convulsant properties, yet remains the drug of choice in most centres for ECT, despite intermittent problems with supply issues. The alternate choice is propofol. Initially there was a suspicion that this agent could have pro-convulsant activity, as evidenced by the appearance of seizure like phenomena in some patients but this has been shown not to be the case. Almost all studies have shown that propofol produces seizures of shorter duration than thiopentone, but in some studies where other outcome measures were assessed, outcomes were similar, suggesting that a prolonged seizure duration was not an absolute necessity for successful ECT. Another attractive feature of propofol is the drugs propensity to blunt the cardiovascular response to ECT, due to either a direct myocardial depressant effect, vasodilator properties, or both (Avramov et al. 1995).

The place of remifentanil
The use of opioids in ECT anaesthesia is not new. However in recent times there has been a renewed interest in the use of remifentanil in ECT anaesthesia, primarily as a means to reduce the dose of the co-administered induction agent. In almost every study (and there have been at least ten), remifentanil, in doses of between 0.5-1 mcg/kg has been used to reduce the dose of induction agent in the order of 25%, which in turn has resulted in longer seizures. Opioids in general are not known to have epileptogenic activity in therapeutic doses, and so the most likely reason for this response is a reduced dose of induction agent, as opposed to any intrinsic effect of the remifentanil, although this question has not been definitively answered.

Sullivan et al. (2004) compared remifentanil (4-8 mcg/kg) and methohexitone alone (1.1 mg/kg), and found that cognition, measured by means of a Mini mental State exam that was “administered over the course of therapy”, was not different between the two groups. The motor and EEG seizures were significantly longer in the remifentanil group, although the production of such seizures required a lower total electrical charge. Whether differences are detected might be dependent on both the...
tests used to detect cognitive impairment and the timing and sensitivity of tests employed. Opioids generally are not known to have epileptogenic effects, so we are forced to conclude that they operate as agents that allow reduction of the standard induction agent.

**Cognitive Impairment and ECT**

**Ketamine**

Recently there has been a resurgence of interest in the use of ketamine anaesthesia for ECT. There have been two recent studies that have investigated the neuroprotective effects of ketamine in ECT. In a retrospective survey of 36 patients who switched from methohexital to ketamine anaesthesia for ECT because of insufficient seizure length, Krystal et al (2003) reported significantly shorter re-orientation times with ketamine. Post-treatment re-orientation time was assessed by questioning the patient with regard to age, date of birth, location and day of week. Re-orientation was assessed as complete when the patient scored 5/5. Time for re-orientation following ketamine anaesthesia was 50.2 min vs methohexital 63.00 min. Of interest, although systolic BP was increased in the ketamine group, as would be expected, the increase was only marginal (188.9 ± 22.1 mm Hg) in methohexitone and 199.4 ± 27.2 mm Hg in ketamine group.

There are several potential mechanisms by which ketamine may exert neuroprotective effects in ECT. Firstly, there is evidence that excitotoxic neuronal damage is mediated by glutamate (an excitatory amino acid) via effects on the NMDA receptor (Waxman et al. 2005) and that these effects can be mitigated by ketamine (Choi et al. 1989). The intense seizure activity elicited during ECT may give rise to reversible excitotoxic damage. It is known that higher stimulation doses lead to more intense seizures and greater cognitive side effects (Sackhein et al. 2000). Different testing methodologies makes assessing these studies difficult. Clearly the type of test employed and its timing in relation to ECT will be critical. Furthermore the reliability of some tests has been called into question. It has been suggested for example (Butterfield et al. 2004) that the widely used orientation questionnaire, while easy to administer, is a poor measure of cognition as it lacks the sensitivity to detect mild cognitive impairment. This is a developing area of ECT research (Loo et al. 2012).

**REFERENCES**


Butterfield, N; Graf, P, Macleod, B. Athanasios P. Propofol Reduces Cognitive Impairment after Electroconvulsive Therapy *The Journal ECT* 2004; 20(1);3-9


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