"Say, are you psychiatrists still using ECT?"

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Editorial

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Résumé

DES MÉDECINS DE DISCIPLINES AUTRES QUE LA PSYCHIATRIE sont étonnés d'apprendre que les électrochocs sont encore répandus comme traitement sûr et utile contre les dépressions unipolaires et bipolaires graves. L'électrochoc consiste à faire passer un courant électrique au moyen d'électrodes fixées unilatéralement ou bilatéralement. Un traitement type comporte au total huit séances ou plus, administrées trois fois par semaine. Les effets secondaires sont minimes. Il ne faut pas considérer les électrochocs comme un traitement de dernier recours : c'est plutôt le traitement de choix chez les patients aux prises avec une dépression grave qui ne peuvent tolérer la pharmacothérapie ou qu'il est vital de faire réagir rapidement.

Psychiatrists are accustomed to the surprise that their colleagues in other disciplines sometimes express when they realize that electroconvulsive therapy (ECT) is still in common use. A few years ago an editorial in the *New England Journal of Medicine* was entitled "Electroconvulsive therapy — a modern medical procedure," the word "modern" implying the need to justify a treatment that is 90% effective and almost 100% safe.[1]

The primary indication for ECT is major unipolar or bipolar depression. Its efficacy is directly proportional to the severity of illness, especially as indicated by changes in psychomotor rate, sleep, appetite, weight, libido and the capacity to experience pleasure.[2] ECT is considered the treatment of choice for depression in the context of many neurologic and medical conditions, including Parkinson's disease, stroke and pregnancy. ECT is also efficacious in the treatment of acute mania and, in some cases, schizophrenia; it is relatively inefficacious, however, in the treatment of depressive episodes in patients with primary personality disorders.[3] It should be the first choice for patients who cannot tolerate pharmacotherapy and for those, such as actively suicidal patients, in whom a rapid response is needed.

In practice, ECT is most often used in patients who have not responded to treatment with 1 or more antidepressants. Contrary to this practice, the results of a multicentre study indicate that the outcome of ECT in medication-resistant patients may be inferior to that observed in patients without medication resistance.[4] However, a very recent study by Lam and his colleagues at the University of British Columbia, which involved a greater number of patients at a single centre, found a very high ECT response rate regardless of whether there was any history of medication resistance.[5] Although the issue is not resolved as to whether antidepressant resistance predicts inferior ECT response, there is no logical reason to consider ECT only after antidepressants have failed. In fact, informed consent requires that depressed patients be informed of ECT as one of the available therapeutic options.

Nondominant unilateral ECT (uECT), in which 1 electrode is placed on the nondominant temple and a second electrode is placed on the vertex, produces less memory impairment in the first 2 months after treatment than bilateral ECT (bECT), in which 1 electrode is placed on each temple. Unfortunately, 20% of patients do not respond to uECT. Most of these patients will still respond to bECT.[6] Many clinicians start patients with uECT and switch to bECT if no response is evident by the fourth treatment. This often means "starting from scratch" in terms of the number of additional treatments needed to obtain a full response. More recent research confirms that bECT is more efficacious than

uECT and suggests that bECT be tried first in most patients.[6] Another variable that may affect efficacy, especially for uECT, is the concomitant use of short-acting benzodiazepines intermittently or continuously during the course of ECT — even if seemingly adequate seizures are produced.[7] Benzodiazepines are commonly prescribed for depressed patients to alleviate anxiety, insomnia or agitation. Substituting buspirone (for anxiety), zopiclone (for insomnia) or a low-potency neuroleptic as needed (although this is controversial in view of the risks of side effects) may be effective against these symptoms without compromising the benefit of ECT.

Anesthesia for ECT is usually induced with thiopental sodium or methohexital (propofol shortens ECT seizure length and may compromise efficacy), followed by succinylcholine to produce virtually complete muscle paralysis. Monitoring of cardiac rhythm, oxygen saturation and blood pressure helps to ensure safety. Stimulation with newer brief-pulse machines significantly reduces confusion and memory impairment compared with induction of seizures with older sine-wave machines, even with bECT.[8] These advances have allowed ECT to become an outpatient procedure for most patients.

A typical course of ECT requires a total of 8 or more treatments, usually given 3 times weekly; improvement is usually evident by the fourth treatment. Because the benefits of ECT in acute depression persist for only 2 to 3 months after completion of the course,[2] the risk of relapse is high unless adequate maintenance therapy is provided. Full-dose antidepressant therapy after successful ECT is a common strategy but may not provide adequate protection against relapse in patients who did not respond well to antidepressant therapy before ECT.[9] Maintenance ECT, in which treatment is given every 2 to 4 weeks, has been suggested as giving better protection against relapse in such patients.[9] Randomized controlled trials to compare ECT with antidepressant therapy as maintenance strategies are needed.

Although many patients experience headache and jaw stiffness immediately after ECT sessions, amnesia and confusion are more significant potential side effects.[2] Most patients who receive bECT experience permanent memory gaps for some events occurring during and up to a few weeks after a course of ECT, as well as shorter periods of retrograde amnesia. Confusion and disorientation are less common, as is delirious euphoria (as distinguished from mania). All of these side effects are far less common with uECT. Most patients, including those treated with bECT, recover normal memory and other cognitive functions within weeks of receiving ECT.[10] However, a few patients (especially those with pronounced global cognitive impairment before ECT) demonstrate persistent retrograde amnesia after ECT, even when there is an improvement in many other aspects of cognitive function.[10]

Considerable improvements have been made in the delivery of ECT, but the mechanism of its action remains unclear.[2] It is known that successful ECT correlates with many of the neurotransmitter and receptor changes in the catecholamine and serotonin systems in the hypothalamus and left frontal cortex (thought to be key sites in the pathophysiology of depression) that occur with antidepressants. Whether such changes are integral to the therapeutic process remains to be determined. What *is* clear is that variables in stimulus intensity, seizure threshold and seizure duration have an important impact on the efficacy and side effects of ECT. The production of bilateral generalized seizures appears necessary but not sufficient to achieve an antidepressant effect. Stimulation at or barely above the seizure threshold is less therapeutic than stimulation at higher levels, even though the seizure duration itself may be the same in both instances.[8] This is especially true for uECT. Because patients vary markedly in seizure threshold, and because this threshold is generally lower in uECT than in bECT, individualizing the "dose" of electrical stimulation is important. The threshold can be determined empirically for each patient at the time of the first uECT. In bECT, a formula based on the patient's age may offer a means of predicting seizure threshold, although this issue is controversial.[11] In uECT, optimal efficacy (with minimal side effects) is correlated with an electrical dose 2.5 times that necessary to reach seizure threshold.[8] Higher doses do not enhance efficacy but do increase cognitive side effects. Over a course of ECT the seizure threshold tends to rise and the seizure duration to shorten. Raising the stimulus intensity to correct this is not usually helpful in lengthening seizure duration. If the seizure duration falls below 10 seconds, the benefit may be lost. In such instances caffeine (500–1000 mg) given orally before treatment often restores seizure length to more than 20 seconds, along with a corresponding benefit, and may also reduce cognitive side effects.[12] However, this strategy requires further evaluation; the results of recent experiments on rats suggest that caffeine augmentation of ECT may be associated with hippocampal neuronal damage.[13]

ECT is a valuable technique in the treatment of major depression and should be better understood by generalist physicians. ECT is neither obsolete nor a treatment of last resort. It is in fact the treatment of choice for patients in whom pharmacotherapy is contraindicated or poorly tolerated, and it can be a crucial intervention for patients who are actively suicidal. Physicians should be aware, however, of the high relapse rate after ECT in patients with acute depression and tailor maintenance strategies accordingly.

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