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GENDER BUT NOT STIMULUS PARAMETERS INFLUENCE PROLACTIN RESPONSE TO ELECTROCONVULSIVE THERAPY*

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SUMMARY

The post-ictal prolactin (PRL) response represents one of the most consistent findings of electroconvulsive therapy (ECT), but correlates variably with the gender of the patient, ECT stimulus waveform, dose and electrode placement. Forty patients with endogenous depression (29 drug-naïve) received either high-energy (240 mC) or low-energy (60 mC) bilateral brief-pulse ECT once or three times a week. The PRL and growth hormone (GH) levels were estimated using double antibody radioimmunoassay. The average post-ECT PRL levels differed significantly from the pre-ECT levels, with a seven- to nine-fold increase in PRL at each week of treatment. No such difference was observed in the GH levels. All patients showed an increase in PRL levels, whereas 42% failed to show an increase in GH levels. The delta PRL response (difference between post-ECT and pre-ECT serum hormone levels) was not significantly different between the drug-naïve and medicated patients nor between the high-energy and low-energy groups at first ECT. Similarly, no difference was observed between the once-weekly and thrice-weekly groups at the third week of ECT. At each week of treatment, the delta PRL was significantly higher in females than in males, unlike the GH response. Electroencephalographic (EEG) seizure duration did not correlate with either delta PRL or delta GH at first ECT and third week ECT. Apart from gender, none of the variables, such as age, baseline severity of illness, presence of psychotic symptoms, drug-naïve status, stimulus dose, seizure duration, seizure strength, pattern and symmetry, frequency of ECT and degree of improvement predicted the delta PRL response. Neither stimulus energy nor frequency of ECT had a significant effect on PRL response. Gender differences in PRL response to ECT merit further investigations.
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*The study was conducted such that all procedures were carried out with the adequate understanding and written consent of the patients.

INTRODUCTION

Electroconvulsive therapy (ECT) produces a rapid and large increase in prolactin (PRL) secretion (Arato et al., 1980; Deakin et al., 1983; O'Dea et al., 1978; Ohman et al., 1976; Skrabanek et al., 1981; Whalley et al., 1982), whereas simulated ECT produces either a non-significant PRL increase (Aperia et al., 1985a; Arato et al., 1980) or a much smaller response than "real" ECT (Deakin et al., 1983). PRL levels rise after spontaneous generalized seizures in epileptics but not after pseudo-seizures (Trimble, 1978). In contrast, the response of other pituitary hormones such as luteinizing hormone releasing hormone (LHRH), thyrotropin releasing hormone (TRH), thyroid stimulating hormone (TSH), growth hormone (GH) and cortisol to ECT is markedly variable and/or inconsistent (Deakin et al., 1983; Weizman et al., 1987; Whalley et al., 1982, 1987). Whalley et al. (1982) observed that whereas rapid increments in PRL and neurohypophysin occurred following ECT, such increases in other pituitary hormones did not occur. These observations suggest that PRL release is specific to ECT rather than a non-specific result of stress and/or anesthesia.

ECT-induced PRL response provides an indirect measure of hypothalamic stimulation, reflecting the aggregate of hypothalamic neurotransmitter release, which in turn depends upon the local electrical or seizure activity as well as seizure-induced changes in other parts of the brain (Swartz and Abrams, 1984). The measurement of pituitary hormones in the plasma may thus provide an important insight into the mechanism of action of ECT (Whalley et al., 1987).

Reviewing the literature on the factors influencing the PRL response (Table I), it becomes evident that no single factor has consistently emerged. The PRL response neither correlates with the degree of clinical improvement, nor with the speed of recovery (Deakin et al., 1983; Huskett et al., 1985), although other studies have reported contradictory findings. There was no correlation between the PRL response and the severity of depression (Aperia et al., 1985a; Huskett et al., 1985). Although Aperia et al. (1985a) reported that electrode application did not influence the hormone release, with PRL levels being the same after unilateral (UL) and bilateral (BL) ECT, most other studies (Abrams and Swartz, 1985; Papakostas et al., 1984; Swartz and Abrams, 1984) report that PRL response was higher with BL ECT than with UL ECT. Regarding the effect of stimulus energy on hormonal release, the study by Zis et al. (1993) found that the PRL increase in response to high-energy ECT was significantly larger compared with low-energy ECT. Robin et al. (1985) found that the ECT-induced PRL release was highest when a sinusoidal waveform stimulus was used and lowest in response to ultra-brief pulse, low-energy ECT; whereas high-energy, wider pulse ECT produced intermediate increments. Furthermore, they reported a high positive correlation between the PRL ratio (post:pre PRL) and the measures of seizure intensity. The reports regarding the relationship between PRL response and seizure duration have been conflicting, with some studies (Aperia et al., 1985a; Scott et al., 1992; Swartz, 1985; Swartz and Abrams, 1984) reporting no significant correlation, whereas Robin et al. (1985) and Zis et al. (1993) report a significant positive correlation between seizure duration and the PRL response. No correlation between the PRL response and either gender or age has been reported (Aperia et al., 1985b; Scott et al., 1992), whereas Huskett et al. (1985) found a larger delta PRL response only in female patients, and Robin et al. (1985) reported a positive correlation between the PRL ratio and the age of the patient. The relationship between the postictal PRL response and the baseline PRL level is unclear, with a positive correlation being reported by some (Swartz, 1985) and no correlation being found by others (Abrams and Swartz, 1985).

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