

Subacute neurocognitive impairment in organophosphate and carbamate insecticide poisoning

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Neurobehavioural studies suggest that organophosphate (OP) and carbamate insecticide poisoning could lead to subacute/chronic cognitive impairment. The factors that mediate this impairment are hitherto unknown. Using cognitive event related potentials (ERPs) as a neurophysiological marker, our objectives were to determine whether there is a subacute cognitive impairment in patients poisoned with Ops and carbamates, and to investigate the risk factors of cognitive impairment.

We recorded ERPs and reaction time (RT) data in 119 patients discharged from hospitals following OP/carbamate poisoning (Test Group) and 29 control subjects. P300 ERP waveforms were derived from averaged EEG recorded at FZ, CZ and PZ scalp sites during a standard auditory 'oddball' task where the subjects responded to target tones while ignoring the standard tones. RT, P300 peak latencies and amplitudes, were compared between two groups adjusting for confounding variables. The risk factors of cognitive impairment in the Test Group was analysed using multiple linear regression (MLR) models.

Once adjusted for other variables, mean P300 amplitude was 2.4uV smaller (~ 43% reduction from the mean of the controls) at FZ (P = 0.025) and 2.7uV (~ 40%) smaller at CZ (P = 0.025) in the Test Group. Once adjusted for other factors, the Test Group patients who developed hypoxia had a 49.4-ms delay in RT compared to those who did not develop hypoxia. Hypoxia also tends to increase the P300 latencies at fronto-central sites (a delay of 17.4ms at FZ and 20.4ms at CZ) but the effect was not statistically significant. Those who had a major psychiatric illness also showed delayed P300 latencies in fronto-central sites. Once adjusted for other variables, psychiatric diagnosis delayed P300 latency at CZ by 34.1ms (P = 0.031) and tended to delay P300 at FZ (P = 0.092).

Our findings indicate that acute poisoning of OP and carbamate insecticides may impair cognitive functions as indexed by RT and P300 cognitive ERPs. Hypoxia seems to play a role in this impairment. The neurocognitive impairment that outlasts clinical illness implies sub-acute effects of the insecticides on brain function.

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