Cognitive effects of organophosphorus insecticide poisoning studied using reaction time, evoked potentials and event-related potentials

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MBBS (Sri Lanka)

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A thesis submitted to the Faculty of Medicine in fulfilment of the requirements for the Degree of

Master of Philosophy

of the

University of Peradeniya

Sri Lanka

June 2007

Abstract

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Introduction: Research over last few decades suggests that acute organophosphorus (OP) poisoning may lead to long-term cognitive impairment. However neurobehavioural and symptomatic manifestations reported in previous studies show numerous inconsistencies, highlighting the need for more objective and quantitative measures of cognitive functions.

Objectives: The aim of the present study was to determine whether acute OP insecticide poisoning leads to impairment of cognitive processing. The specific objectives were to compare objective and quantitative psychophysiological parameters of cognitive processing of visual information and auditory information, 1) between patients who recovered from cholinergic phase of OP insecticide poisoning and matched controls and 2) between immediate post-cholinergic phase and six months after poisoning in the patients.

Methodology: The first part of this research was a case-control study where patients recovered from cholinergic phase of OP poisoning (n=44) were compared with two age- and sex-matched controls groups, viz. healthy controls (n=43) and patients with

paracetamol overdose (n=11). The second part was a prospective study where the OP poisoned patients were followed up after six months of poisoning and the findings were compared with their immediate post cholinergic phase measurements. The tests used to assess visuomotor information processing were simple visual reaction time, recognition visual reaction time, visual evoked potentials (VEP) and motor evoked potentials. The term "cognitive processing time (CPT)" was used to denote the time taken from initial cortical perception of a stimulus to initiation of descending motor impulses. CPT of each type of visual reactions was calculated by subtracting the sum of the visual impulse duration and the motor impulse duration from reaction time (CPT = Reaction time – (P100 VEP latency + total motor conduction time)). Auditory P300 cognitive event-related potential (ERP) was recorded, quantified and analysed to assess cognitive processing of auditory information.

Results: Patients with OP insecticide poisoning showed significant delays in CPT of simple visual reactions (CPT_{SVR}) (p=0.037), CPT of recognition visual reactions (CPT_{RVR}) (p=0.024) and P300 latency (p=0.003) compared to healthy controls. The patients also had a significant impairment in CPT_{SVR} (p=0.017), CPT_{RVR} (p=0.002) and P300 latency (p=0.009) compared to the control group with paracetamol overdose. Comparison of the initial and follow-up findings of the patients revealed that the impairment in CPT_{SVR} (p=0.527) and P300 latency (p=0.867) remained unchanged even six months after poisoning. However, CPT_{RVR} showed a significant improvement (p=0.012). Visual and motor conduction latencies or P300 amplitude were similar between the groups and between the two assessments of the patients with OP poisoning.

Conclusions: OP insecticide poisoning appears to slow the cognitive processes involved in visuomotor information processing and auditory stimulus evaluation. These effects persist beyond the clinical recovery from the cholinergic phase, and the deficits in auditory stimulus evaluation and cognitive processing in simple visual reactions appear to be persistent even six months after exposure. These findings are compatible with the cognitive deficits observed in some previous human studies. The neural substrates of the affected cognitive processes are largely compatible with the topography of the neuropathological lesions that have been observed in OP exposed experimental animals.