

Long-lasting effects of acute organophosphorus insecticide poisoning on cognitive processing of visual information

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Objective: Acute organophosphorus (OP) poisoning may lead to long-term neuropsychological impairment (1,2,3). However, the manifestations reported in literature show numerous inconsistencies, highlighting the need for more objective and quantitative measures of cognitive functions. This study was conducted to determine whether acute OP insecticide poisoning leads to long-lasting impairment of objective parameters of cognitive processing visual information.

Methods: The first part of this research was a case-control study where a group of patients who had recovered from the cholinergic phase of OP poisoning (n=44) were compared with two age- and sex-matched control 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 6 months of poisoning and the findings were compared with their initial 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 visual reaction time ($CPT = \text{visual reaction time} - (\text{P100 VEP latency} + \text{total motor conduction time})$) (Figure 1). **Results:** OP poisoned patients showed significant delays in CPT of simple visual reactions (CPT_{SVR}) and CPT of recognition visual reactions (CPT_{RVR}) compared to both control groups. Comparison of the initial and follow-up findings of the patients revealed a sustained impairment in CPT_{SVR} and an improvement in CPT_{RVR} . Visual and motor conduction latencies were similar between the groups and between the two assessments of the OP intoxicated patients (Table 1). **Conclusions:** OP insecticide poisoning appears to slow the cognitive processing of visual information. These effects persist beyond cholinergic phase of poisoning, and the deficits in cognitive processing in simple visual reactions appear to be persistent even 6 months after poisoning. Being an objective measure of cognitive processing, CPT could also be used to assess neurocognitive deficits that follow other intoxications or disease states. **References:** 1. Savage EP, et al. Chronic neurological sequelae of acute organophosphate pesticide poisoning. *Arch Environ Health* 1988;43:38-45. 2. Rosenstock L, Keifer M, Daniell WE, McConnell R, Claypoole K. Chronic central nervous system effects of acute OP pesticide intoxication. *Lancet* 1991;338:223-227. 3. Steenland K, et al. Chronic neurological sequelae to organophosphate pesticide poisoning. *Am J Public Health* 1994; 84:731-736.