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Epidemiologic Notes and Reports

Occupationally Related Neurologic Abnormalities – Massachusetts

A recent study sponsored by the National Institute for Occupational Safety and Health (NIOSH) and conducted by the Occupational Health Program, Harvard School of Public Health, shows that some workers, who were found in 1978 to have bladder neuropathy as a result of occupational exposure to dimethylaminopropionitrile (DMAPN), continue to have persistent neurologic abnormalities.

In 1978, Harvard and NIOSH evaluated workers who had developed bladder neuropathy at a plant in Marblehead, Massachusetts, that produced polyurethane-foam products (*1,2*). A similar outbreak was investigated by NIOSH at a plant in Baltimore, Maryland, which used the same chemical process (*3,4*).

Symptoms of urinary tract dysfunction at the Marblehead plant were associated with the introduction of a catalyst containing DMAPN, and most workers' symptoms resolved rapidly after the catalyst was removed from the process. Three months after the catalyst was removed, 14 (13%) of the 104 workers originally diagnosed as having DMAPN toxicity were found to have persistent symptoms. Eleven of these 14, as well as 2 other workers who were subsequently identified as having persistent symptoms, participated in the follow-up study in June 1980. This study included questionnaire evaluations of urinary symptoms and sexual history; neurologic examinations; urologic evaluations, including cystometrography; electromyography of the anal sphincter and measurement of sacral latency time; nerve conduction studies of the right peroneal and right sural nerves; and studies of visual- and auditory-evoked responses. Results were compared, when possible, with those of the 1978 study.

Although the overall prevalence of urologic and other symptoms was considerably lower than in 1978, a high proportion of the group examined continued to report symptoms of urologic and sexual difficulties. Of the 11 workers evaluated in both studies, 10 (90%) still had symptoms. Seven (64%) still reported urinary hesitancy and 6 (55%), incomplete bladder emptying. Five (45%) reported sexual difficulties (loss of libido or impaired sexual function) as compared with 3 (27%) in 1978. Three of the 10 symptomatic individuals had abnormalities on general neurologic examination—1 with lower extremity sensorimotor neuropathy; 1 with hyper-reflexic knee jerks and ankle clonus; and 1 with a right lower extremity radiculopathy. Three of 4 workers examined in both studies had objective neurologic findings in 1978 which were not present in this recent study. Electromyographic abnormalities were present in 3 of 10 workers; visual- and auditory-evoked responses, sacral latency, and sphincter electromyograms were normal in all 10. Two individuals were found on cystometrography to have the first sensation that they needed to void only when abnormally large volumes of urine were in

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the bladder. The persons most severely affected in 1978 were most likely to have persistent neurologic abnormalities at the time of the recent study.

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Editorial Note: This investigation documents the persistence of neurologic abnormalities in a small group of workers exposed more than 2 years ago to DMAPN. While some symptoms improved considerably after removal from exposure, many symptoms and neurologic abnormalities persisted, and symptoms of sexual dysfunction appeared to become more prominent. The biphasic course of DMAPN toxicity observed in the Maryland plant (initial bladder symptomatology that resolved, followed by the appearance of lower extremity peripheral neuropathy) was not observed in the Marblehead group (*1,4*).

The neurologic syndrome associated with DMAPN differs significantly from previously reported occupational neuropathies in the predominance of genitourinary dysfunction. Although the neurotoxicity of DMAPN has been demonstrated in the laboratory (*5*), additional animal studies are needed to clarify the pathogenesis of this unique disorder. Follow-up studies of workers affected by other industrial neurotoxins (methyl-n-butyl ketone, n-hexane) are also needed to assess the rate of permanent disability in these workers.

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Lead Toxicity Secondary to Gasoline Sniffing among Navajos — Arizona

In the period July 1974-June 1980, 23 patients (19 males and 4 females) were admitted to Navajo Area Indian Service Hospitals for lead toxicity secondary to gasoline sniffing. Ages at diagnosis ranged from 10-20 years (mean 14.6); 8 patients had 3 or more separate admissions, and 15 had 1.

Table 1 summarizes the blood lead levels by clinical classification of these Navajo adolescents. Fifteen patients (65%) had lead encephalopathy when seen initially. Of 5 patients (22%) who were asymptomatic, 3 had blood-lead levels of $\geq$80 μg/dL. Of 3 patients (13%) who initially had focal neurologic signs, 2 had blood-lead levels of $\geq$80 μg/dL. Hematologic examination showed that 3 patients had basophilic stippling, and 2 had radiographic "lead lines" in the metaphyses and epiphyses of the radius and ulna. Free erythrocyte protoporphyrin (FEP) levels measured for 8 patients were not consistently elevated and did not correlate closely with blood-lead levels.

Patients were treated with chelation therapy, either ethylenediaminetetraacetate