

Severe headache associated with occupational exposure to Stoddard solvent

T. S. Prince and S. E. Spengler

University of Kentucky, College of Medicine, Department of Preventive Medicine and Environmental Health, Chandler Medical Center, Lexington, KY, USA

We report a case of recurrent headaches in a woman with a workplace exposure to airborne (misted) lubricating fluid containing Stoddard solvent. For 2 months, the employee was seen by her family physician, a neurologist and an ophthalmologist. All attempted to diagnose the cause of and treat her headaches. Despite extensive testing, no etiology was discovered. Her headaches continued despite the use of medications. The employee, suspecting an occupational connection, changed the lubricating fluid at her workstation to a non-Stoddard solvent. Within 2 days, she reported the complete resolution of her headaches with no further recurrences. A thorough occupational history and literature review supported exposure to Stoddard solvent as the probable source of her headaches.

Key words: Mineral spirits; solvent exposure and headache; Stoddard solvent.

Received 22 May 2000; revised 15 August 2000; accepted 4 December 2000

Introduction

Stoddard solvent, also known as mineral spirits or white spirits, is used extensively in industrial settings for paint and wax diluents, dry cleaning agents and pesticides, and as a cleaning agent/degreaser in mechanical shops. In 1990, the volume of Stoddard solvent produced in the USA was 38 million pounds [1]. Furthermore, between 1981 and 1983 an estimated 1.9 million employees in 404 plants were potentially exposed to Stoddard solvent in the workplace [2]. The composition of the man-made, organic solvent is straight and branched chain paraffins, naphthenes and aromatic hydrocarbons. Limited scientific studies suggest minimal risk for serious health effects from inhalation, the most common exposure route, at levels commonly found in industrial settings. However, case reports and cross-sectional studies do contain reports of acute central nervous system complaints in workers with exposure to Stoddard solvent [3].

Case report

A 33-year-old woman was seen by her family physician in late September 1999 for a complaint of recent severe headaches unresponsive to over-the-counter non-steroidal anti-inflammatory drugs. The headaches were largely bilateral and bifrontal/bitemporal in location. They were accompanied by dizziness, nausea and photophobia, and were so severe that the woman often needed to lie down in a darkened room. She denied both scotoma and seeing flashing lights with her headaches. She was initially treated with toradol for a diagnosis of 'migraine headache with tension component'. The following day, she required demerol and phenergan for the pain and nausea, and was treated for possible sinusitis. Despite this, she did not improve. A CT scan of the head and sinuses was normal. Blood work, including thyroid function, complete blood count and chemistry, was unremarkable. She was referred to an ophthalmologist and a neurologist. Glaucoma was ruled out and an electroencephalogram was normal. Other medications were tried, including vicodin, amitriptyline for long-standing complaints of disturbed sleep and serzone for possible depression. Her headaches continued unabated.

Correspondence to: T. S. Prince, MD, Department of Preventive Medicine and Environmental Health, 1141 Red Mile Road, Suite 201, Lexington, KY 40504-9842, USA. Tel: +1 859 323 5166; fax: +1 859 323 1038; e-mail: tprince@pop.uky.edu

Approximately 6–7 weeks after her headaches began, the patient noticed that, after being symptom-free in the morning, she developed a headache while at work. She began to suspect an occupational exposure as the cause of her headaches and reported this to her family physician. He then referred her to an occupational medicine clinic. Before her appointment there, the patient, suspecting exposure to a new lubricating fluid used in her workplace as the cause, removed the substance from her workstation. Within 2 days, her headaches resolved completely.

As part of her occupational medicine consultation, a thorough occupational and environmental history was obtained. The woman had worked for 8 years at her current employer as a set-up operator making metal parts. The parts were mist-sprayed with lubricating fluid contained in a 25 gallon tank at her workstation. No respiratory protection was provided or used, and she wore cloth gloves that were often soaked with solvent by the end of her shift. In late September of 1999, a new product, later determined to be essentially standard Stoddard solvent, was introduced as the misted lubricant. (The prior lubricant was a mixture of propylene glycol and hexylene glycol.) At the time of the changeover, she noted a new odor. She began to have headaches about the same time and was headache-free for only ~1–1.5 h after waking up in the morning. She also noted that her headaches improved through the course of the weekend. The patient denied any other suspect exposures during this time: no water changes (city water user), fumigations, construction, new hobbies, pets, travel, etc. She denied having headaches in recent years, but said that as a teenager she had suffered from headaches that had resolved on their own.

While symptomatic, the patient was missing from 1 to 3 days of work each week. After switching back to the original lubricating fluid, the patient was symptom-free and did not miss any work. The patient provided the material safety data sheet for the lubricating fluid (Stoddard solvent) as well as those for the prior/replacement fluid (propylene glycol and hexylene glycol). These were reviewed and the manufacturers were contacted for additional information.

Discussion

It has been estimated that millions of industrial workers have been exposed to Stoddard solvent over time [2]. The medical and scientific data from controlled human studies suggest that there is minimal risk for serious health effects from inhalation at commonly occurring industrial levels [3–5]. The most commonly encountered symptoms from inhalation are eye, skin and throat irritation. The low toxicity for mammals is demonstrated by controlled animal studies. In one study, the LC_{50} for rats was not reached despite an 8 h exposure to air

concentrations of 1400 p.p.m. [4]. A current recommended average allowable airborne level for Stoddard solvent in the workplace is 100 p.p.m., based on the data on toxicities of the major components [5]. Although few data are available on actual workplace concentrations of Stoddard solvent, a survey of dry-cleaning plants in Michigan found an average concentration of 65 p.p.m., with maximum average ambient air concentrations of 135–200 p.p.m. at a particular plant [6]. No air sampling data could be obtained in our patient's setting.

Despite a lack of controlled human studies on neurotoxic effects, there do exist case reports and cross-sectional epidemiological studies in the literature of acute central nervous system symptoms, including headache, dizziness and fatigue, in cases of occupational exposure to Stoddard solvent [4,7–9]. These symptoms, consistent with acute central neurotoxicity, have generally been reported to clear on weekends, at night and with time away from the solvent [10].

The persistence of headaches in our patient into the weekend may be explained by the finding that although solvents are partly excreted within a few hours after exposure, they may linger in expired air for several days. In addition, chronic exposure to organic solvents has been reported to result possibly in an organic brain syndrome characterized by decreased memory, fatigue, headache and dizziness [7]. In the current case, the patient was exposed for only ~8 weeks and had no persistent symptoms.

Perhaps most significantly demonstrated by this case is the importance of obtaining an occupational and exposure history. According to the patient and records reviewed, an exposure history was not taken until the patient was referred to our office. Had this been done earlier in the course of her exposure, symptoms could have been relieved sooner, saving significant medical resources and lost productivity, as well as reducing the risk of chronic toxicity.

References

1. Environmental Protection Agency. *Toxic Substances Control Act Inventory 1990* [Chemical Update System Database]. Washington, DC: US Environmental Protection Agency, Office of Pollution Prevention and Toxics, Information Management Division, 1992.
2. US Department of Health and Human Services. *National Occupational Exposure Survey*. Cincinnati, OH: US Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, Division of Surveillance, Hazard Evaluations and Field Studies, 1992.
3. US Department of Health and Human Services. *Toxicological Profile for Stoddard Solvent*. Chapel Hill, NC: US Department of Health and Human Services, Public

- Health Service, Agency for Toxic Substances and Disease Registry, June 1995.
4. Carpenter CP, Kinkead ER, Geary DL Jr, Sullivan LJ, King JM. Petroleum hydrocarbon toxicity studies: III. Animal and human response to vapors of Stoddard solvent. *Toxicol Appl Pharmacol* 1975; **32**: 282–297.
 5. Stoddard solvent. In: *Documentation of Threshold Limit Values and Biological Exposure Indices*. Cincinnati, OH: American Conference of Governmental Industrial Hygienists, Inc., 1991, p. 31.
 6. Oberg M. A survey of the petroleum solvent inhalation exposure in Detroit dry-cleaning. *Am Ind Hyg Assoc J* 1968; **29**: 547–557.
 7. Arlien-Söberg P, Bruhn P, Gyldensted C, Melgaard B. Chronic painters' syndrome: chronic toxic encephalopathy in house painters. *Acta Neurol Scand* 1979; **60**: 149–156.
 8. Baker EL. Solvent neurotoxicity: the current evidence. *J Occup Med* 1986; **28**: 126–129.
 9. Hane M, Axelson O, Blume J, Hogstedt C, Sundell L, Ydreborg B. Psychological function changes among house painters. *Scand J Work Environ Health* 1977; **3**: 91–99.
 10. Daniell WE, Couser WG, Rosenstock L. Occupational solvent exposure and glomerulonephritis: A case report and review of the literature. *J Am Med Assoc* 1988; **259**: 2280–2283.