criteria for a recommended standard....

OCCUPATIONAL EXPOSURE TO METHYL ALCOHOL

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
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where, in 1913, Baskerville was able to collect several hundred such case reports from various medical periodicals. Baskerville felt that these cases represented a small percentage of the total number because many physicians did not report cases in the scientific press and many others failed to recognize the industrial and occupational diseases of chronic methyl alcohol poisoning. [12] For an extensive summary of numerous poisoning cases from drinking wood alcohol or inhaling its vapor, the reader is referred to the Baskerville review. [12]

One of the earliest case reports of methyl alcohol poisoning in an occupational setting was by De Schweinitz [13] in 1901. He described the case of a 39-year-old man who suddenly became totally blind after a brief illness. The patient had been employed intermittently (3-4 days at a time) for 3 years as a painter and varnisher. The varnish was dissolved in methyl alcohol, and the patient stated that he generally used methyl alcohol to clean the varnish off his hands and arms, and sometimes off his face. He denied drinking the alcohol. During these 3 years, he had several times become dizzy when varnishing the insides of small articles of furniture or closets on hot days. For 2 months prior to the onset of blindness, he had worked every day as a varnisher in a shop. This was the longest period of uninterrupted exposure to the varnish during the 3-year period. He frequently noted attacks of what he called "misty vision," which disappeared 10-15 minutes after he left work. The day prior to his loss of sight, the patient was unable to work because of chills, numbness, and shooting pains in his lower extremities, and he returned home and went to bed. When he awoke the following morning, he was totally blind. Although treated by a physician, the blindness persisted for 2 weeks

In 1912, Tyson [17] described a case of methyl alcohol poisoning in a worker who was involved in varnishing the inside of beer vats. Work was commenced on December 3, 1911, and continued on the following day with no medical complaints. On December 5, the worker experienced headache, vertigo, unsteady gait, nausea, vomiting, and acted as if intoxicated; consequently he did not work on this day. The author did not state if the subject worked on December 6. On December 7, the worker began having visual disturbances. At this time, he consulted a physician who diagnosed methyl alcohol poisoning. On December 12, an ophthalmologist made the following observations: the pupils were practically nonreactive to light, there was retinal edema, and initial vision (eccentric) was right 1/200 and left 2/200. In three weeks, his vision had improved to 20/30 in each eye. Six to 7 months later, with no additional methyl alcohol exposure, visual acuity remained stable, while the pupillary response to light remained sluggish. In addition, the author described a progressive contraction of the visual fields during the entire period of observation. Tyson also indicated that the progressive constriction of visual fields corresponded to degenerated bundles of fibers and groups of ganglion cells becoming confluent as the degenerative process spread. He also concluded that this case was produced solely by inhalation of methyl alcohol vapor. The airborne concentration of methyl alcohol to which the worker was exposed was not determined.

In a review article published in 1912, Wood [18] commented on 4 workers (one of which was the case previously described by Tyson [17]) poisoned while varnishing beer vats. Methyl alcohol was reported as a constituent of the varnish. All 4 workers had been involved in varnishing

Drager respiratory filters which were impermeable to methyl alcohol. The next filter used was a Drager Type K-90, which was permeable to methyl alcohol. The latter filters were changed 4 times since they became very wet within a period of 20-30 minutes. Occasionally during the first day of scraping the boiler, the worker suffered from vertigo. During break periods in fresh air, he saw colored rings. The first day's operation required about 5 hours. The next morning, the worker became nauseated upon entering the boiler room which had been used the preceding night. Despite the nausea, the worker emptied the boiler, liberating small quantities of methyl alcohol vapor. He then suffered visual disturbances for the rest of the second day, despite the fact that he underwent no further methyl alcohol exposure. On the third day, upon entering the boiler room, the worker suffered nausea and visual disorders and was then hospitalized. Ophthalmoscopic examination showed papilledema of both eyes that began to clear after a few days. After 5 weeks, full visual acuity returned. Blood, urine, and cerebrospinal fluid tests, as well as physical examination, disclosed no abnormal findings. Formic acid, found in the urine in the first 11 weeks following the initial examination, was no longer detectable after 11 weeks. The presence of formic acid confirmed the author's belief that the toxicity was due to methyl alcohol exposure. Questioning of the patient revealed that he was in the habit of frequently washing his hands with methyl alcohol. The author [26] therefore concluded that the exposure involved a single acute intoxication by inhalation superimposed upon a chronic condition resulting from percutaneous absorption of methyl alcohol along with inhalation of low concentrations of methyl alcohol over a period of years. In his theoretical discussion of

Epidemiologic Studies

In 1912, Tyson [17] described a factory in New York City in which 25-30 young women worked in a 20 x 50 foot room polishing wooden lead pencils with a varnish solution containing methyl alcohol. During damp or cold weather, the windows of this room remained closed in order to maintain the quality of the finished pencils. All of the women in the room experienced headaches and an unspecified number exhibited what the author termed gastric disorders. One woman missed 8 weeks of work because of chronic gastritis. Two cases from the same work area were reviewed by Tyson. The initial symptoms of a 30-year-old woman described in the first case were headache, vertigo, weakness (unspecified), and nausea without vomiting. She also had dizziness and obscuration (sic) of vision while working. The woman stated that the symptoms occurred principally during the day when the windows were closed. After working about 3 hours, she experienced blurring of vision, changes in color perception, and the symptoms mentioned previously. After half an hour in fresh air, the symptoms subsided. The same condition then occurred in the afternoon. Upon examination, her optic discs were hyperemic, the edges were blurred, and the veins were dilated. The other case was similar in that, approximately 3 hours after beginning work, the woman would on certain days experience frontal headache, dizziness, and nausea. At times, she experienced what she called a mist before her eyes. She was examined initially because of failing vision. The eye examination showed pallor, blurring, and edema of the discs, as well as dilated retinal veins. Upon questioning, both patients stated that they used methyl alcohol on occasion to cleanse their skin. The author suggested that the visual disturbances or loss of function were related to

nature. [24] This conclusion was supported by the findings of later studies on rabbits, [50] which showed that methyl alcohol was a mild eye irritant.

Many of the signs and symptoms of intoxication attributed to either the ingestion, inhalation, or percutaneous absorption of methyl alcohol are not specific to methyl alcohol. Thus, for example, headache, dizziness, nausea and other gastrointestinal disturbances, weakness, vertigo, chills, behavioral disturbances, and neuritis can be caused by a wide range of chemical and physical stresses on the organism. Therefore, these signs and symptoms may be of little use in diagnosing methyl alcohol poisoning. The characteristic signs and symptoms of methyl alcohol poisoning in humans, then, are the various visual disturbances and severe metabolic acidosis which appear to result from overexposure to methyl alcohol by any route. Chronic exposure at relatively low levels of methyl alcohol may have effects other than those resulting from acute exposure; however, no studies have been found that would support this speculation.

The presence of a characteristic asymptomatic latent period following ingestion of methyl alcohol, prior to the development of acidosis and/or visual disturbances in humans and in some nonhuman primates, suggests that these effects are caused by a metabolite of methyl alcohol rather than by the alcohol itself. Evidence for a metabolite of methyl alcohol acting as the proximal toxic agent is the fact that toxic manifestations can be attenuated by the administration of ethyl alcohol, [29] a compound that has been shown to inhibit the oxidation of methyl alcohol in vivo. [30,31,37]

As a result of the critical role which the metabolism plays in the course of human methyl alcohol intoxication, it is clear that factors which

affect that metabolic pathway will also affect the severity and course of the methyl alcohol intoxication. The amelioration of methyl alcohol poisoning by ethyl alcohol [29] is one example. The individual variations in activity of the alcohol dehydrogenase systems probably account for the variation in the individual responses observed with methyl alcohol poisoning. In their study of an epidemic of methyl alcohol poisoning, Bennett et al [40] noted what they called an extreme variation in individual response to a given amount of methyl alcohol in that one individual died after ingesting approximately 15 ml of a 40% methyl alcohol solution and another survived after ingesting 500 ml of this same solution. This wide variability in individual susceptibility to ingested methyl alcohol has also been noted by others, [11] and reviewed by Cooper and Kini. [44]

Although not as clearly documented, there appears to be a similar individual variability among persons exposed to methyl alcohol by inhalation or percutaneous absorption, both in the type of symptoms manifested and in their severity. For example, Wood [18] described the cases of 4 men who were employed together as varnishers of beer vats. One felt dizzy after the first day and could not continue past the second day. Another did not develop symptoms until the third day. The remaining 2 worked through the third day but subsequently died without returning to work. In Tyson's study of the pencil-varnishing operation, [17] all the women in the room presumably had similar exposures but only 2 sought medical treatment for visual disorders. The results of one inhalation study [47] using rhesus monkeys revealed individual susceptibility differences in that one animal died during exposure to 1,000 ppm methyl

hand, occupational exposures at air levels of 25 ppm [38] during an 8-hour working day apparently may be endured without harmful effects.

No human or experimental mammalian studies have been found to evaluate the possible mutagenic, teratogenic, or carcinogenic effects of methyl alcohol. In a study [54] in grasshoppers, Oxya velox Fabricius, 0.3% methyl alcohol injected in the vicinity of the testes produced an incidence of 3.5% chromosomal aberrations in testicular tissue, but examination of the stages of spermatogenesis was not performed.

No aberrations were observed in grasshoppers injected with distilled water. Saha and Khudabaksh [54] did not report any evidence for the induction of permanent aberrations in germ cell lines or for the inheritability of the observed abberations. In view of the fundamental differences in genetic mechanisms, the utility of the grasshopper in quantitatively predicting inheritable germinal or somatic mutations in humans is questionable.

In Feb. 1981, the National Institute for Occupational Safety and Health (NIOSH) received a request to det. if a potential health hazard existed to employees from exposure to MeOH [67-56-1] while operating a spirit duplicator and to the chem. by-products produced when using an electronic stencil maker. NIOSH conducted an environmental evaluation on Apr. 1, 1981. NIOSH detd. that a health hazard existed due to excessive exposure to MeOH. This is based on the MeOH concns. measured, eye irritation experienced by the operator, and exposure levels similar to those found in a large study involving use of spirit duplicators which resulted in operators experiencing blurred vision, headache, nausea, and dizziness. The by-products identified from the stencil cutter during operation were present in very low concns. Adverse health effects are usually not found with these low exposures. Recommendations to reduce the exposure to MeOH vapors are presented with examples of suggested local exhaust ventilation designs.