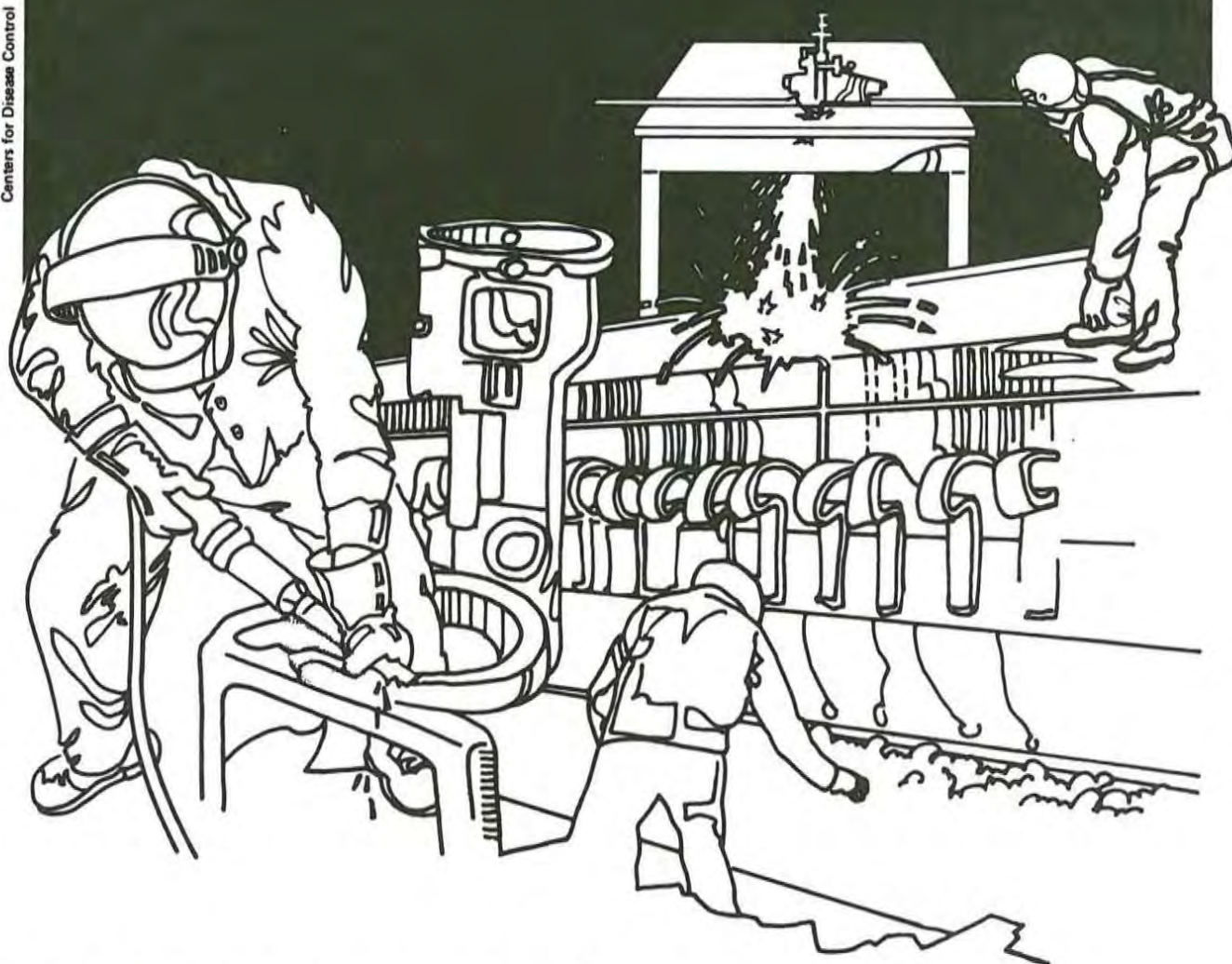


NIOSH



Health Hazard Evaluation Report

HETA 83-113-1470
RAYGO-WAGNER
PORTLAND, OREGON

PREFACE

The Hazard Evaluations and Technical Assistance Branch of NIOSH conducts field investigations of possible health hazards in the workplace. These investigations are conducted under the authority of Section 20(a)(6) of the Occupational Safety and Health Act of 1970, 29 U.S.C. 669(a)(6) which authorizes the Secretary of Health and Human Services, following a written request from any employer or authorized representative of employees, to determine whether any substance normally found in the place of employment has potentially toxic effects in such concentrations as used or found.

The Hazard Evaluations and Technical Assistance Branch also provides, upon request, medical, nursing, and industrial hygiene technical and consultative assistance (TA) to Federal, state, and local agencies; labor; industry and other groups or individuals to control occupational health hazards and to prevent related trauma and disease.

Mention of company names or products does not constitute endorsement by the National Institute for Occupational Safety and Health.

HETA 83-113-1470
MAY 1983
RAYGO-WAGNER
PORTLAND, OREGON

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I SUMMARY

In December 1982, the National Institute for Occupational Safety and Health (NIOSH) received a request from employees at Raygo-Wagner, Portland, Oregon, to determine if exposure to paint solvents was the cause of seizures experienced by three painters.

Between December 30, 1982, and June 15, 1983, a NIOSH medical officer administered a standardized questionnaire to 12 of the 14 current or former painters. NIOSH was unable to locate the other two painters. A follow-up environmental/medical survey occurred on August 18-19, 1983. Environmental air samples and personal breathing zone samples of two painters were collected during the spray painting operation. In addition, the purchase orders of paints, thinner, and paint remover were reviewed. NIOSH also interviewed a neurologist and a neuropsychologist who had seen four of the painters.

Due to changes in the composition and type of paints and solvents, the methods of paint preparation, and the use of personal protective equipment by the painters, it was not possible to predict/determine painters' exposures to solvents that occurred in the 1970's. These changes would have had the effect of reducing the painters' exposures to the solvent vapors. Air sampling under 1983 conditions showed one painter's solvent exposure was 1.1 times the 8-hour time weighted average criteria and 4.4 times the short-term criteria.

Nine of twelve (75%) interviewed painters reported trouble with memory or thinking ability. In addition, these painters reported a variety of other symptoms similar to those described in workers with solvent-associated chronic toxic encephalopathy.

Review of the questionnaires and medical records revealed five painters with significant neurological or neuropsychological problems. Of these five painters a detailed medical history, neurological examination, and neuropsychological testing was available for four. The medical history and the results of testing were compatible with the presence of chronic toxic encephalopathy in two painters and possibly a third. A fourth painter had abnormalities in neuropsychologic tests, but his history of encephalitis and seizures, which can produce the abnormalities seen, confounds the interpretation of the abnormalities. Finally, two painters developed complex partial seizures during employment at Raygo-Wagner, but NIOSH was unable to determine whether these were related to chemical exposures at Raygo-Wagner.

On the basis of the data collected from this evaluation, NIOSH has determined that the painters were exposed to paint and solvent vapors that could be the cause of 1) chronic toxic encephalopathy in two and possibly a third painter, and 2) subjectively reported decrements in memory in nine of twelve painters interviewed. This is based on the limited environmental sampling that indicates the environmental criteria could have been exceeded and the medical information acquired through personnel interviews, questionnaires and medical records. Since the plant has ceased production, there are no recommendations offered in this report.

KEYWORDS: SIC 3531 (Construction, Mining, and Materials Handling Machinery and Equipment) Paint Solvents, Glycol Ethers, Painting, Seizures, Neurological Symptoms, Chronic Toxic Encephalopathy.

II INTRODUCTION

On 16 December, 1982, the National Institute for Occupational Safety and Health's (NIOSH) Region X Office received a letter from a painter previously employed at the Raygo-Wagner (R-W) Company of Portland, Oregon. The painter described health problems in three R-W painters, including himself, which he attributed to solvent exposures experienced during work at R-W. A request for a Health Hazard Evaluation, submitted by the local Painter's Union steward on December 30, 1983, addressed these same concerns.

NIOSH conducted an initial survey in February 1983. Between December 30, 1982, and June 15, 1983, a NIOSH medical officer administered a standardized questionnaire/interview to 12 of the 14 current or former Raygo-Wagner painters identified by NIOSH. NIOSH has been unable to contact the two remaining painters.

A follow-up medical and environmental survey was conducted on August 18-19, 1983. The NIOSH medical officer met with plant management, worker representatives, four of the painters, and several of the painters' physicians. NIOSH also reviewed purchase orders for products purchased from 1977 through 1983 from a local supplier in Portland, Oregon (R-W's principle supplier of solvents), and a paint company in Portland, Oregon (R-W's principle supplier of paint until 1981). The NIOSH industrial hygienist conducted personal breathing-zone sampling for solvents on two painters on August 19, 1983.

At a second follow-up medical survey on October 20, 1983, NIOSH interviewed a neurologist and psychologist who had seen four of the painters.

III BACKGROUND

The Raygo-Wagner Company, Portland, Oregon, manufactured large log handling, piggyback truck handling and ship container handling equipment. The initial step in the manufacturing process was the production and subassembly of parts in the metal working, welding and machining areas of the plant. These parts were cleaned by shot blasting, moved into the large paint spray booth where they were sprayed with a primer paint coat. Some of the parts also received a top coat at this time. The parts were then assembled to make the final configuration. The units were steam cleaned and moved into the paint booth where they were sanded, spots of residual oil removed with solvent wetted rags and painted with an enamel top coat. During the painting and/or parts cleaning, the painters wore coveralls, head covers, gloves and supplied-air half-face respirators. The paint booth, located in the main assembly building, is approximately 20 feet wide, 20 feet high and 60 feet long. The larger manufactured units would just fit in the booth and some had to be turned around in order to complete the painting.

Raygo Wagner, a 25-year-old firm, moved to its present location in 1961. Since then, many changes have occurred in the number of employees, paint materials used, paint preparation, and personal protective equipment. Employer and employee interviews supplied the following historical information.

A. Number of Employees

In February 1983 there were 95 employees, including three painters. During the period of peak employment, there were 350 employees and up to nine painters.

B. Materials Used

1. The composition of the products used by painters at R-W is given in Table 1. Use of some of these products, as determined from purchase orders, is shown in Table 2 (Dates are based on the month in which R-W received the product).
2. The paint thinner (products C and D) used from 1961 to 1975 was not available after July, 1975. At that time, Raygo-Wagner began using a similar paint thinner produced by a local firm (Product J).
3. Prior to 1980, the primer paint and top coat paint were lead based paints. The yellow top coat paint used on over 98% of the equipment was a lead chromate paint. Since 1980 the paints have been lead free.
4. In 1978 and/or 1979 the regular paint used on one unit did not cure properly and had to be removed using a paint remover (Product B). The paint remover contained methylene chloride. Some small parts were stripped inside the booth. The larger pieces were stripped outside of the building.
5. The paints used since 1969 were heated just prior to entering the spray gun to permit the application of thicker paint with a lower solvent content.

C. Paint Preparation

Prior to painting the metal had to be clean. Until 1978 the metal parts were sanded and hand wiped with solvent wetted rags prior to painting. In mid-1978 a shot blast facility was installed. In April, 1981, an internal plant memo indicated that selected parts, i.e., hydraulic cylinder heads, were still being hand wiped with solvent-soaked rags. After final assembly the units were steam cleaned; any rust spots sanded; occasionally oil and grease residues wiped off with paint thinner; and the top coat applied.

D. Personal Protective Equipment

From 1961 to 1979 half-face cartridge type respirators for use with organic vapors and paint mist were used during spray painting. A cotton respirator sock was usually placed over the edge of the respirator face piece. The use of these socks will prevent a good seal between the respirator and the face. Before 1978 respirators were sometimes not worn when hand wiping the parts with solvent. After 1978 their use was mandatory. The use of respirators approved for dust was optional during sanding operations.

In June, 1979 supplied air respirators were supplied to the painters; however, their use was optional until 1980. In 1981 two additional supplied air respirators were purchased. During the NIOSH visit in August, 1983, the supplied air respirators were worn during the sanding and painting operations.

Rubber gloves were made available in approximately 1974. Prior to that a protective lotion was available when hand wiping parts or when painting.

The large units contained a large built in storage box on either side. Spray painting the inside was done by placing the arm and spray gun inside the box and spraying. For degreasing, though, painters had to climb inside the boxes to hand wipe the surfaces with solvents. The area under the chassis where the engine and drive train were located formed a partial enclosure. Respirators were worn when painting in this area but not when solvents were used to hand wipe the surface prior to painting.

E. Health Effects

The painters reported that exposures to the solvents and paints sometimes resulted in lightheadedness and the sensation of being drunk. Because of this, painters were allowed to take 10-15 minute "fresh air" breaks.

IV EVALUATION DESIGN

A. Environmental

Exposures that the painters received during the 1970's are difficult to estimate. Some of the reasons for this difficulty are:

1. Painters conducted several different operations in the past and used other materials:

a) During paint preparation metal parts were hand wiped with solvent wetted rags.

b) The painters did not wear rubber gloves or respirators during hand wiping and, therefore, were exposed to solvents through both inhalation and skin absorption.

c) Until 1979 half-face cartridge type respirators were used when painting, but were usually not worn when hand wiping with solvent wetted rags. The cartridges were changed only once or twice a month. Since the respirators probably had a functional life of four to eight hours in the environment as described, during most of their use they would have been ineffective in removing the organic vapor from the breathed air since the organic vapor cartridges would have been saturated with solvent.

d) An exact composition of all the material used over the past years could not be confirmed.

2. Since work was conducted in the paint booth, exposures would have varied depending on work location (up wind or down wind) relative to the air flow. In enclosed and semi-enclosed areas of a unit (e.g., in tool boxes or around the drive train the exposures would have been higher than when working on open surfaces.

Due to the changes in the preparations for painting (hand wiping with solvent changed to shot blasting) and type of paint thinner and paints used, NIOSH was unable to duplicate the exposures to solvent and paint vapors that occurred during the painting of a unit prior to 1983. Nevertheless, NIOSH hoped that sampling during the painting of a unit in 1983 would provide information from which past exposures could be estimated. Personal samples were collected in the breathing zone of the painters as they sprayed the enamel top coat on a large log handler. Samples were scheduled to be collected during the application of the primer coat on another piece of equipment; however, the company was sold in September, 1983, and production terminated prior to the scheduled sampling date.

Breathing zone air samples for solvent vapors were collected during the spray painting operation using activated charcoal tubes at a flow rate of 200 cc per minute. Bulk air samples were analyzed by mass spectrometry and gas chromatography to identify the compounds present. The individual samples were then analyzed by NIOSH Method P&CAM 127.

B. Medical

Painters were identified by means of job descriptions, supplied by the company or by recollection of other painters. A NIOSH medical officer administered a standardized questionnaire either in person or by telephone to two current and ten former R-W painters. The questionnaire requested information about (1) current and previous occupations, (2) time spent painting while at R-W, (3) other chemical exposures that might have caused neurological illness, (4) work hygiene and use of personal protective equipment, (5) smoking and alcohol consumption, (6) neurological history, and (7) general health history.

NIOSH obtained medical records from private physicians and consultants, when possible, to verify the nature and course of illnesses disclosed by the questionnaires. This included the results of neurological exams and neuropsychological testing. In 1981 R-W Company provided electroencephalograms (EEG) to painters on a voluntary basis. NIOSH reviewed the EEG reports that were available.

In addition, NIOSH reviewed orders for materials purchased from 1977 through 1983 from a local solvent supplier in Portland, Oregon (R-W's principle supplier of solvents), and a paint company in Portland, Oregon (R-W's principle supplier of paint until 1981).

V. EVALUATION CRITERIA

A. Environmental Criteria

As a guide to the evaluation of the hazards posed by workplace exposures, NIOSH field staff employ environmental evaluation criteria for assessment of a number of chemical and physical agents. These criteria are intended to suggest levels of exposure to which most workers may be exposed up to 10 hours per day, 40 hours per week for a working lifetime without experiencing adverse health effects. It is, however, important to note that not all workers will be protected from adverse health effects if their exposures are maintained below these levels. A small percentage may experience adverse health effects because of individual susceptibility, a pre-existing medical condition, and/or a hypersensitivity (allergy).

In addition, some hazardous substances may act in combination with other workplace exposures, the general environment, or with medications or personal habits of the worker to produce health effects even if the occupation exposures are controlled at the level set by the evaluation criteria. Also, some substances are absorbed by direct contact with the skin and mucous membranes, and thus potentially increase the overall exposure. Finally, evaluation criteria may change over the years as new information on the toxic effects of an agent becomes available.

The primary sources of environmental evaluation criteria for the workplace are: 1) NIOSH Criteria Documents and recommendations, 2) the American Conference of Governmental Industrial Hygienists' (ACGIH) Threshold Limit Values (TLV's), 3) the U.S. Department of Labor (OSHA) occupational health standards, and 4) the Oregon State Standards. Often, the NIOSH recommendations and ACGIH TLV's are lower than the corresponding OSHA standards. Both NIOSH recommendations and ACGIH TLV's usually are based on more recent information than are the OSHA standards. The OSHA standards also may be required to take into account the feasibility of controlling exposures in various industries where the agents are used; the NIOSH-recommended standards, by contrast, are based primarily on concerns relating to the prevention of occupational disease. In evaluating the exposure levels and the recommendations for reducing these levels found in this report, it should be noted that industry is legally required to meet only those levels specified by an OSHA or state standard.

A time-weighted average (TWA) exposure refers to the average airborne concentration of a substance during a normal 8- to 10-hour workday. Some substances have recommended short-term exposure limits or ceiling values which are intended to supplement the TWA where there are recognized toxic effects from high short-term exposures.

<u>Substance</u>	<u>NIOSH and/or ACGIH Recommended Criteria 10 Hr TWA</u>	<u>Oregon State or (OSHA) Standard 8 Hr TWA</u>	<u>Health Effects</u>
Aliphatic naphtha	75 ppm	200 ppm	dizziness, drowsiness, headache, irritation of eyes, nose, throat, dry skin
2-butoxy-* ethanol (skin)	25 ppm (ACGIH)	50 ppm	irritation of eyes, nose, & throat, skin absorption, hemolytic anemia
N-butyl alcohol (skin)	50 ppm ceiling (ACGIH)	100 ppm	irritation of eyes, nose & throat, drowsiness, blurred vision, dry skin, corneal inflammation
2-ethoxyethyl* acetate (skin)	50 ppm (ACGIH)	100 ppm	eye and nose irritation
Isopropyl alcohol	400 ppm	400 ppm	skin irritation, headache fatigue, conjunctivitis
Toluene (skin)	100 ppm	100 ppm	headache, dizziness, fatigue weakness, dermatitis
Xylene	100 ppm	100 ppm	headache, dizziness, nausea irritation of eyes, nose & throat, dermatitis

NOTE: When there are two or more substances present that have similar health effects, their exposure levels are combined and an equivalent permissible exposure level determined for each exposure.

*See last paragraph of the toxicity section which follows.

B. Toxicity

Aromatic Hydrocarbon Solvents

Acute Toxicity

The acute toxicity of aromatic hydrocarbon solvents (e.g., toluene, xylene) is well known. At low exposures, symptoms may include headache, lassitude, nausea, and irritation of eyes and mucous membranes. As concentration increases, impairment of coordination, momentary loss of memory, and anorexia result. At even higher concentrations palpitation, extreme weakness, pronounced loss of coordination and impairment of reaction occur. This may be followed by narcosis and coma. In addition, abnormalities in renal and liver function may be seen (8-11).

Chronic Toxicity

Chronic toxicity is less well understood. In the past there was some concern that toluene and xylene might be myelotoxic as is benzene. However, at present it appears that these reports of myelotoxicity were probably due to benzene contamination of these other solvents. Apart from this possible effect, it was felt that chronic exposure to toluene and xylene was without harmful effect.

Within the past ten years, however, reports of chronic neuropsychological effects in workers exposed to these solvents have appeared, principally in the Scandinavian literature. The spectrum of these effects has been termed "chronic toxic encephalopathy". As described, the symptoms of chronic toxic encephalopathy are of a nonspecific, neurasthenic nature and include fatigue, loss of appetite, and memory loss, especially short-term memory. Physical examination is usually normal, although a mild peripheral neuropathy may be present. In addition, many of the authors report decrements in neuropsychological tests, especially those which measure short-term memory, attention span, and visuomotor coordination. Electroencephalograms may also be abnormal. One author who has called this spectrum of neuropsychological problems "Chronic Painter's Syndrome" described cerebral atrophy (as determined by CT scan) as an additional component. Several retrospective studies of pension records have shown an increase in the diagnosis of presenile dementia in painters, which may be a manifestation of these same toxic effects of solvents. (12-37).

Seizures

Seizures have only rarely been associated with aromatic hydrocarbon exposure. Most of the reported cases are examples of substance abuse, most frequently "glue sniffing". The seizures have commonly been of the generalized convulsive type and follow acute, very high dose exposures to solvents.

One study demonstrated the onset of seizures in cats following exposure to either benzene or toluene. Benzene provoked generalized convulsive seizures, whereas toluene was described as producing seizures resembling the complex partial type. One case of seizure associated with xylene exposure has been reported (42-44).

Seizures have been reported following exposure to methyl chloride, but not methylene chloride (1-7).

Glycol Ethers

NIOSH recommends that 2-methoxyethanol (2ME) and 2-ethoxyethanol (2EE) be regarded in the workplace as having the potential to cause adverse reproductive effects in male and female workers. These recommendations are based on the results of several recent studies that have demonstrated dose-related embryotoxicity and other reproductive effects in several species of animals exposed by different routes of administration. Of particular concern are those studies in which exposure of pregnant animals to concentrations of 2ME or 2EE at or below their respective Occupational Safety and Health Administration (OSHA)

Permissible Exposure Limits (PEL's) led to increased incidences of embryonic death, teratogenesis, or growth retardation. Exposure of male animals resulted in testicular atrophy and sterility. In each case the animals had been exposed to 2ME or 2EE at concentrations at or below their respective Occupational Safety and Health Administration (OSHA) Permissible Exposure Limits (PEL's). Concern also extends to structurally related glycol ethers, such as 2-butoxyethanol and 2-ethoxyethanol, that have not been tested adequately to assess fully their potential for causing reproductive effects. Preliminary test results of some structurally related glycol ethers indicate that they also have the potential for causing adverse reproductive effects similar to 2ME and 2EE (49).

VI RESULTS

A. Environmental

The composition of the solvents used by painters at R-W is given in Table 1. Use of these solvents and paints, as determined from purchase orders, is shown in Figure 1 and Table 2 (Dates are based on the month in which R-W received the product). The results of the air samples conducted during the spray painting of the enamel top coat on a log handling unit are shown in Table 3. There were seven solvents that were present in sufficient amounts to quantify. They were aliphatic naphtha, 2 butoxyethanol, N-butyl alcohol, 2-ethoxyethyl acetate, isopropyl alcohol, toluene and xylene.

There were two painters who conducted the spray painting during the sampling period. First they spray painted the front portion of the unit since only part of the unit could fit into the spray booth at a given time. This took 55 minutes. This portion had been prepared (sanded and masked) the previous day. The unit was then turned around, the remaining portion was sanded by the painters, residual oil and grease were removed, and the rear portion was masked. These steps took 240 minutes. The rear portion was then painted, which took 95 minutes. During 20 minutes of the 95 minute period, the area under the unit which surrounded the drive train, was painted and a simultaneous sample was collected to obtain the peak exposure. The painters wore supplied air respirators while painting and sanding; consequently, the samples represent an exposure that would have occurred if respirators were not worn or if the cartridges in a cartridge type respirator were spent or used.

The following table gives the ratio of the measured exposure to the standard or criteria used. Each solvent is compared to its individual allowable exposure criteria. However, when there are two or more substances present that have similar health effects, their exposure levels are combined and an equivalent permissible exposure level determined for each sample. Their combined potential exposure is compared to both the recommended evaluation criteria available and the current Oregon State Standards.

390 Minute TWA Exposure VERSUS

	Recommended TWA Evaluation Criteria	Oregon State TWA Standard
Painter A	0.60	0.35
Painter B	1.09	0.60

1.0= equivalent permissible TWA exposure level determined for each sample.

20 minute exposure while painting area under unit by the drive train VERSUS

	Recommended Short-Term Evaluation Criteria	Oregon State Long Term Standards
Painter A	3.0	1.5
Painter B	4.4	2.3

1.0= equivalent permissible short term exposure level determined for each sample.

The time weighted average for painter A was 0.6 (60%) of the criteria while painter B was 1.09 (109%) of the criteria. This difference shows the variability of exposure that the painter can receive. The 20 minute exposure while painting the underside of the unit, were 3.0 and 4.4 times the short term exposure criterion which is 1.5 times the 8 hours TWA.

These limited data suggest that exposure to solvent vapors during spray painting had a large variability; they could have exceeded the TWA criteria; and high peak exposures could have occurred when painting in semi-confined areas. It is anticipated that exposure while hand wiping parts with solvent soaked rags would have been at least as high as when spray painting. If the painters did not wear respirators or rubber gloves when conducting this job, the health effects included headache, nausea, tiredness, eye, nose and throat irritation, would have been likely to occur.

B. Medical

Review of the company personnel records, NIOSH questionnaires and personal interviews with the painters revealed the following: 13 men and possibly a 14th man had worked at R-W as painters or painter's helpers between 1965 and 1983. At the time of the hazard evaluation only three of these were current employees of R-W. Two of the three still worked as painters. The third worked in the sandblasting area. Of the twelve painters interviewed, the mean time spent at R-W as painter or painter helper was 78 months (range 17-204 months). The current mean age of the painters is 37 years (range 29-51 years). The responses of the painters on the neurological history portion of the questionnaire are summarized in Table 4. The most frequently reported symptom was trouble with memory or thinking ability (75%).

Review of the questionnaires and medical records turned up five painters with significant neurological or neuropsychological problems:

Case #1

This former painter was a full-time painter at R-W in late 1978 when he noted the onset of memory problems and was told by another worker that he was "acting strange." He reported periods of being "spaced out" and fading off" lasting 5-10 minutes which occurred over the course of several years, apparently beginning in either late 1978 or 1979. These episodes were generally preceded by an aura and have been diagnosed by three neurologists as representing complex partial seizures. In addition to these complex partial seizures, he reported two grand mal seizures, one in January 1979 (while blowing on a fire he was trying to start) and one in November 1981. Following the first grand mal seizure, he had an EEG and CT scan in January 1979, which were reported as normal, and was started on antiseizure medication. None of these seizures had been observed by a physician. He has been treated for these seizures (with apparently poor to fair response) with several drugs and has reported some improvement in his symptoms. He had two neuropsychological evaluations in 1982, initially reported as abnormal. However, a review of the 1982 neuropsychological tests showed that these tests may have been improperly interpreted. An EEG, done in August, 1982, was reported as abnormal and suggestive of a temporal structural lesion. A CT scan in November 1982 was reported as normal. This painter had a history of a head injury at age seven with possible loss of consciousness, and several other head injuries prior to work at R-W but no history of seizures prior to 1978.

Case #2

This former painter had a history of seizures (complex partial) starting in early 1981. At the time he was a full-time painter at Raygo-Wagner. None of his seizures have been observed by a physician, and several physicians have stated that his problems were not epileptic but rather functional. Nevertheless, he was treated with antiepileptic medication. Following self-termination of his medication on two separate occasions, he reported having seizures. He has been evaluated by three neurologists. Two of them have felt that the patient had historical evidence for a seizure disorder best characterized as complex partial. The third felt that he had a "history of syncopal episodes and possible seizure states that are best typified as complex partial" but "unexplained as to etiology." He had two awake and asleep EEGs in 1981: one, performed in May, was reported as abnormal; and one, performed in July, was reported as normal. He had a CT scan in July 1981 reported as normal. He had neuropsychological testing in 1983, reported as abnormal.

Case #3

This current employee but former painter had a history of poor memory, change in personality, and feelings of unreality. The worker stated that he felt he had had the problems with concentration and memory for several years prior to an examination in 1983 (onset approximately late 1960's or early 1970's). Neither

neurologist that has seen him feels that his history is consistent with a seizure disorder. He has a history of significant hypertension, probably essential, first diagnosed, according to available records, in 1981, which has been poorly controlled. He also has a past history of heavy alcohol intake of more than 4 beers per day. An EEG, dynamic and static brain scans, and an echoen- cephalogram, all performed in May 1981, were reported as normal. He had neuropsychological evaluations in August 1982 and October 1983, both reported as abnormal.

Case #4

This former painter was hospitalized on several occasions in 1981 with grandmal seizures. Multiple diagnostic studies were conducted including CT scan, EEG, CSF analysis, and a left hemispheric brain biopsy. Nevertheless, the etiology of his seizures could not be confirmed. The diagnosis made was probable encephalitis. At the time of onset of his seizures, he was working at R-W as a mechanic and had not worked for R-W as a painter for approximately one year. However, he may have worked as a substitute painter intermittently during 1980. Both the neurologist and neuropsychologist who saw him felt that the course and nature of his neurological problems was fundamentally different than the cases described above and was probably not related to solvent exposure at R-W.

Case #5

This former worker had an EEG in the summer of 1981, reported as abnormal. NIOSH has been unable to contact this worker. Thus, there is no medical and only limited work history available.

Other Painters

In addition to these cases, there were reports of four painters passing out on the job from solvent fume exposure, (workers #2, 5, 13, 14 - See Figure 2), necessitating visits to local emergency rooms.

The other eight painters who completed interviews reported no seizures. Except for EEGs, which several painters had in 1981, no neurological or neuropsychological testing has been done on any of these eight men. Two workers (#6, 8) reported having EEGs in 1981, but reports were not available for NIOSH review.

VII DISCUSSION

The preparation and painting processes at R-W changed significantly over time. In addition, there had been marked improvements in the personal protective equipment used by the painters. These included the introduction of supplied air respirators in 1979 and regular use of these respirators beginning in 1981, which reduced the inhalation exposures of painters to solvents and paints, and the installation in 1978-1979 and full use in 1981 of the shot blast facility, which reduced the amount of hand wiping and thus the solvent exposure associated with hand wiping. Thus, it is not possible to determine full exposure levels that may have been routinely encountered in the past. However, the following body of evidence persuasively argues that exposures in the past to solvents were considerably higher than recent exposures:

1. the reports of repeated episodes of euphoria, light-headedness, appetite loss, and incoordination while at work at R-W;
2. the four reports of painters passing out on the job necessitating visits to local emergency rooms;
3. fresh air breaks being taken during the workday by the painters;
4. the use during painting of cloth socks with cartridge respirators and the probable use of respirator cartridges that were spent or fully used.
5. the practice of hand-wiping of parts and machines without rubber gloves or respiratory protection.
6. sampling data from the present study which show (under improved conditions and using a paint that contains less solvent than in the past) variable levels of exposure to solvent vapors during spray painting; solvent levels at or exceeding the TWA criteria; and high peak exposures occurring during painting in confined areas.

This evidence is compatible with significant solvent exposure for the painters at R-W and the presence of acute health effects resulting from those exposures.

Chronic solvent exposure has recently been described to produce a chronic toxic encephalopathy consisting of incoordination, short-term memory loss, and decrements in neuropsychological testing. Regrettably, medical history, and neuropsychological testing were not available for ten of the 14 painters. However, of the four R-W painters with test results and medical histories available, the questionnaire responses, medical history, and neuropsychological testing are compatible with the presence of chronic toxic encephalopathy in two (cases #2, 3) and possibly a third (case #1). A fourth painter (case #4) had abnormalities in neuropsychological tests but his history of encephalitis and seizures which can produce the abnormalities seen, confounds the interpretation of the abnormalities. With respect to a case #3, while hypertension can cause decrements in neuropsychological scores, the changes in his behavior and memory predate the onset of his hypertension and coincide with his work at R-W. This suggests that his problems may be attributable to his solvent exposure.

In addition, several other current or former workers reported symptoms similar to those described in the Scandinavian literature among workers with chronic toxic encephalopathy.

There is a temporal association between work as a painter at R-W and the onset of seizures or seizure-like states in two (cases 1, 2) of the three painters (cases #1, 2, 4) with a history of seizures or seizure-like states. With the exception of case 4 above, these reported seizures resemble complex partial seizures.

Exposure to solvents of the same type as those present at R-W has not been reported to be associated with seizures, except for toluene in high doses (glue sniffers and cats) and one equivocal case report of xylene-induced seizures (42-44). The seizures associated with glue sniffing differ from those reported by the R-W painters, both in their type of and their temporal relationship to solvent exposure. Glue sniffing cases are of the generalized convulsive type and immediately follow very high solvent exposure, whereas R-W cases #1 and #2 reported complex partial seizures which did not immediately follow solvent exposure. In contrast to the experience of glue sniffers, toluene-produced seizures in cats have been described as complex partial, although the latency period between exposure and seizure onset was short (minutes) for the cats.

In this group of R-W painters, there is no evidence of exposure to known convulsants (methyl chloride, hydrazines, nicotine, etc). However, the lack of prior reports in the medical literature does not preclude that some of the solvents used at R-W may have contributed to the development of seizure disorders or states resembling seizures. Also, it is possible that one of the solvents or paints used could have been contaminated with a known convulsant (e.g., methyl chloride contamination in a methylene chloride paint stripper. Regrettably, apart from Material Safety Data Sheets, there is no information about the composition of the products either to support or refute the possibility.

A final possibility is that the lapses in attention and memory, whether they are complex partial seizures or some other type of interruption of cognitive function, may be a further progression of "chronic toxic encephalopathy which has not been previously described.

VIII RECOMMENDATIONS

1. Since production has ceased permanently at the R-W company, NIOSH has no specific recommendations.

IX REFERENCES

Chlorinated Methanes

1. Ahlstrom, RC and Steele, JM. Methyl Chloride. From Kirk Othmer, Encyclopedia of Chemical Technology. Vol 5: 677-685, 1978.
2. American Conference of Governmental Industrial Hygienists. Methylene Chloride. Documentation of TLV's. page 275-276, 1982.
3. Anthony, T. Methylene Chloride. From Kirk Othmer, Encyclopedia of Chemical Technology. Vol 5: 686-693, 1978.
4. Oettingen, WF. Methyl Chloride. From The Halogenated Aliphatic, Olefinic, Cyclic, Aromatic, and Aliphatic-Aromatic Hydrocarbons Including the Halogenated Insecticides, their Toxicity and Potential Dangers. DHEW (PHS) Publication No. 414: 1-15, 1955.

5. Ott, MG, Skory, LK, Holder, BB, Bronson, JM and Williams, PR. Health Evaluation of Employees Occupationally Exposed to Methylene Chloride. *Scand J Work Environ & Health* 9 (Suppl 1): 1-38, 1983.
6. Scharnweber, HC, Spears, GN, Cowles, SR. Chronic Methyl Chloride Intoxication in Six Industrial Workers. *Jour Occup Med* 16(2): 112-113, 1974.
7. Skrabalak, DS and Babish, JG. Safety Standards for Occupational Exposure to Dichloromethane. *Regulatory Toxicology and Pharmacology* 3: 139-143, 1983.

Neurological Effects of Solvent Exposure

A. Acute Effects

8. Acute Behavioral Effects of Solvent Exposure. *Acta Neurol Scand* 66 (Suppl 92): 117-129, 1982.
9. American Conference of Governmental Industrial Hygienists. Toluene. Documentation of TLV's. page 400-401, 1982.
10. American Conference of Governmental Industrial Hygienists. Xylene. Documentation of TLV's. page 439-440, 1982.
11. Craft, BF. Solvents and related compounds. From *Environmental and Occupational Medicine*, edited by Rom, WN. Boston: Little, Brown and Co, 1983: 511-528.

B. Chronic Effects

12. Arlien-Soborg, P, Bruhn, P, Glydensted, D and Melgaard, B. Chronic painter's syndrome: Chronic toxic encephalopathy in house painters. *Acta Neurol Scand* 60: 149-156, 1979.
13. Arlien-Soborg, P, Zilstorff, K, Grandjean, B and Pedersen, LM. Vestibular dysfunction in occupational solvent intoxication. *Clin Otolaryngol* 6: 285-290, 1981.
14. Axelsson, O. Epidemiology of Solvent-Related Neuropsychiatric Disorders. From Seminar at the TUC Centenary Institute of Occupational Health. London School of Hygiene and Tropical Medicine, April, 1982. Proceedings in press.
15. Axelsson, O, Hane, M and Hogstedt, C. A Case-Referent Study on Neuropsychiatric Disorders among Workers Exposed to Solvents - A Review and Some Further Aspects. From International Symposium on the Control of Air Pollution in the Working Environment. The Work Environment Fund, 1978. 103-111.
16. Axelsson, O, Hane, M and Hogstedt, C. A case-referent study on neuropsychiatric disorders among workers exposed to solvents. *Scand J Work Environ & Health* 6: 239-273, 1980.

17. Bruhn, P, Arlien-Soborg, Glydensted, C and Christensen, EL. Prognosis in chronic toxic encephelopathy: A two-year follow-up study in 26 house painters with occupational encephalopathy. *Acta Neurol Scandinav* 64: 259-272, 1981.
18. Elofsson, SA, Gamberale, F, Hindmarsh, T, et al. Exposure to organic solvents: A cross-sectional epidemiologic investigation on occupationally exposed car and industrial spray painters with special reference to the nervous system. *Scand J Work Environ & Health* 6: 239-273, 1980.
19. Escobar, A and Aruffo, C. Chronic thinner intoxication: clinicopathologic report of a human case. *J Neur Neurosurg Psych* 43: 986-994, 1980.
20. Hane, M, Axelson, O, Blume, J, Hogstedt, C, Sundell, L, Ydreborg, B. Psychological function changes among house painters. *Scand J Work Environ & Health* 3: 91-99, 1977.
21. Hanninen, H, Eskelinen, L, Husman, K and Markku, N. Behavioral effects of long-term exposure to a mixture of organic solvents. *Scand J Work Environ & Health* 4: 240-255, 1976.
22. Hogstedt, C, Hane, M and Axelson, O. Chapter 12: Diagnostic and Health Care Aspects of Workers Exposed to Solvents. *From Developments in Occupational Medicine*, Ed by Zenz, C. Chicago: Year Book Medical Publ, 1980: 249-257.
23. Husman, K. Symptoms of car painters with long-term exposure to a mixture of organic solvents. *Scand J Work Environ Health* 6: 19-32, 1980.
24. Husman, K and Karli, P. Clinical neurological findings among car painters exposed to a mixture of organic solvents. *Scand J Work Environ Health* 6: 33-39, 1980.
25. Juntunen, J, Hernberg, S, Eistola, P and Hupli, V. Exposure to Industrial Solvents and Brain Atrophy: A Retrospective Study of Pneumoencephalographic Findings among 37 Patients with Exposure to Industrial Solvents. *Eur Neurol* 19: 366-375, 1980.
26. Juntunen, J Hupli, V, Hernberg, S, and Luisto, M. Neurological Picture of Organic Solvent Poisoning in Industry: A Retrospective Clinic Study of 37 patients. *Int Arch Occup & Environ Health* 46: 219-231, 1980.
27. King, MD, Day, RE, Oliver, JS, Lush, M and Watson, JM. Solvent Encephalopathy. *Br Med J* 283: 663-665, 1981.
28. Knox, JW and Nelson, JR. Permanent Encephalopathy From Toluene Inhalation. *NEJM* 275(26): 1494-1496, 1966.
29. Lindstrom, K. Psychological performances of workers exposed to various solvents. *Work-envirom.-hth* 10: 151-155, 1973.

30. Lindstrom, K. Changes in Psychological Performances of Solvent-Poisoned and Solvent-Exposed Workers. *Am J Ind Med* 1: 69-84, 1980.
31. Lindstrom, K. Behavioral Effects of Long Term Exposure to Organic Solvents. *Acta Neurol Scand* 66(Suppl 92): 131-141, 1982.
32. Mikkelsen, S. A Cohort Study of Disability Pension and Death among Painters with Special Regard to Disabling Presenile Dementia as an Occupational Disease. *Scand J Soc Med Suppl* 16: 34-43,
33. Neurophysiological Findings in Workers Exposed to Organic Solvent. *Acta Neurol Scand* 66(Suppl 92): 109-116, 1982.
34. Seppalainen, AM, Lindstrom, K and Martelin, T. Neurophysiological and Psychological Picture of Solvent Poisoning. *Am J Ind Med* 1: 31-42, 1980.
35. Spradlin, JB. Neurotoxic Effects of Long-Term Exposure to Organic Hydrocarbon Solvents: Epidemiologic Aspects. *Developments in Toxicology and Environmental Science* 8: 307-317, 1980.
36. Struwe, G, Mindus, P and Jonsson, B. Psychiatric Ratings in Occupational Health Research: A Study of Mental Symptoms in Lacquerers. *Am J Ind Med* 1: 23-30, 1980.
37. Voights, A and Kaufman, CE. Acidosis and Other Metabolic Abnormalities Associated With Paint Sniffing. *South Med J* 76(4): 443, 1983.

Epilepsy

38. Delgado-Escueta, AV, Trieman, DM and Walsh, GO. The Treatable Epilepsies. *NEJM* 308(25,26): 1508-1514, 1576-1584, 1983.
39. Porter, RJ. Etiology and Classification of Epileptic Seizures. From *Epilepsy Updated: Causes and Treatment*, Ed by Robb, P Year Book Medical Publ, Chicago, 1980: 1-10.
40. Porter, RJ and Theodore, WH. Nonsedative Regimens in the Treatment of Epilepsy. *Arch Intern Med* 143: 945-947, 1983.
41. Schomer, DL. Partial Epilepsy. *NEJM* 309(9): 536-539, 1983.

Epilepsy and Solvents

42. **Allister, C, Lush, M, Oliver, JS, and Watson, JM. Status epilepticus caused by solvent abuse. Letter to the Editor. *Br Med Jour*. 1156, 1981.
43. Contreras, CM, Gonzalez-Extrada, T, Zarabozo, D and Fernandez-Guardiola, A. Petit Mal and Grand Mal Seizures Produced by Toluene or Benzene Intoxication in the Cat. *Electroencephalography and Clinical Neurophysiology* 46: 290-301, 1979.

44. Goldie, I. Can Xylene (Xylol) Provoke Convulsive Seizures? Industrial Medicine and Surgery Jan 1960: 35.

Neuropsychological Assessment

45. Baker, EL, Feldman, RG, White, RF, Harley, JP, Dinse, GE and Berkey, CS. Monitoring Neurotoxins in Industry: Development of a Neurobehavioral Test Battery. Jour Occup Med 25(2): 125-130, 1983.
46. Feldman, RG, Ricks, NL and Baker, EL. Neuropsychological Effects of Industrial Toxins: A Review. Am Jour Ind Med 1: 211-227, 1980.
47. Johnson, BL and Anger, WK. Behavioral Toxicology. From Environment and Occupational Medicine. Edited by Rom, WN. Boston: Little, Brown and Co, 1983: 329-350.
48. Lezak, MD. Neuropsychological Assessment. Second Edition. New York: Oxford University Press, 1983.

Glycol Ethers

- 49 NIOSH Current Intelligence Bulletin 39, May 2, 1983, Glycol Ethers (2-Methoxyethanol, 2-Ethoxyethanol).

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XI DISTRIBUTION AND AVAILABILITY OF REPORT

Copies of this report are currently available upon request from NIOSH, Division of Standards Development and Technology Transfer, 4676 Columbia Parkway, Cincinnati, Ohio 45226. After 90 days, the report will be available through the National Technical Information Service (NTIS), 5285 Port Royal, Springfield, Virginia 22161. Information regarding its availability through NTIS can be obtained from the NIOSH Publications Office at the Cincinnati address. Copies of this report have been sent to:

1. Raygo Wagner, Minneapolis, Minnesota.
2. Sign Painters, Paint Makers, and Allied Trades, Seattle, Washington.
3. Oregon State Accident Prevention Division, Salem, Oregon.
4. U. S. Department of Labor, Occupational Safety and Health Agency (OSHA), Region X, Seattle, Washington.

For the purpose of informing affected employees, copies of this report shall be posted by the employer in a prominent place accessible to the employees for a period of 30 calendar days.

TABLE 1

Products used by painters at Raygo-Wagner 1977-1981.
Composition based on MSDS data supplied by manufacturer.

RAYGO-WAGNER
PORTLAND, OREGON
HETA 83-113

Product A (Lacquer Thinner #2):	%
Paraffins (including naphthenes)	25
Aromatics (not benzene)	26
Oxygenated Solvents	49
Product B (Painter Remover)	
Methylene Chloride	76
Methanol	10
Toluene	3
Product C (Solvent)	
Isoparaffins	18
Naphthenes	9
Toluene	73
Product D (Solvent)	
Paraffins (including naphthenes)	22
Toluene	49
Ethyl Benzene	6
Xylene	23
Product E (Xylene)	
Paraffins (including naphthenes)	2
Benezene	1
Toluene	2
Ethyl Benzene	21
Xylene	74
Product F (Thinner #200)	
Paraffins (including naphthenes)	93
Benzene	1
Toluene	6

MSDA not available for: Product G (Stoddard Solvent), Product H (#325 Solvent), and Product I (Lacquer Thinner #3).

Table 2

Products received 1977 through 1981 by
R-W but not depicted in figure 1.

RAYGO-WAGNER
PORTLAND, OREGON
HETA 83-113

Product	Date of Receipt	Quantity (gallons)
B	9/77	10
	3/80	5
	2/81	5
	9/81	5
F	6/79	55
G	2/77	55
	5/77	55
	7/77	165
	11/77	76

TABLE 3
BREATHING ZONE AIR CONCENTRATIONS FOR
PAINT SOLVENTS
AUGUST 1983

RAYGO-WAGNER
PORTLAND, OREGON
HETA 83-113

Job	Sample Number	Sample time (min.)	Sample volume liters	Toluene ppm	Xylene ppm	2-Butoxy-ethanol ppm	2-Ethoxyethyl acetate ppm	N-butyl alcohol ppm	Isopropyl alcohol ppm	Aliphatic naphtha ppm
Painter A										
Paint front end	1	55	2.43	10	2	5	<1	3	5	18
Sand and mask back end	3	240	12.3	6	0.2	<1	<1	<1	<1	2
Paint back and underside	5	95	3.92	30	4	10	1	13	20	55
Paint underside only	7	20	3.65	50	8	18	4	32	47	176
Painter B										
Paint front end	2	55	2.55	27	4	9	1	9	14	50
Sand and mask back end	4	252	12.4	2	0.2	<1	<1	<1	<1	<1
Paint back & underside	6	95	5.02	49	6	15	3	27	38	128
Paint underside only	8	20	3.64	81	13	27	7	55	78	235
TWA - Painter A	1,3,5	390	18.6	13	1	3	<1	4	5	17
TWA - Painter B	2,4,6	402	20.0	17	2	5	1	8	11	37

Limits of detection - toluene - 0.01 mg; xylene - 0.01 mg; 2-butoxyethanol - 0.05 mg; 2-ethoxyethyl acetate - 0.01 mg; N-butyl alcohol - 0.01 mg; isopropyl alcohol - 0.01 mg; aliphatic naphtha - 0.1 mg.

Table 4.

Frequency of neurological symptoms with four or more positive responses as reported during questionnaire survey of current (N=2) and former (N=10) painters.

RAYGO-WAGNER
PORTLAND, OREGON
HETA 83-113

<u>Symptom</u>	<u>Number reporting symptom</u>
Trouble with memory or thinking ability	9
Drowsiness/sleepiness during day	7
Change in hearing	6
Ringing in ears	5
Nausea	5
Loss of balance or staggering	5
Dizziness	5
Nervousness	5
Sleep disturbance	5
Fainting/Black-out	4
Numbness/tingling in hands or feet	4
Incoordination	4
Frequent headaches	4

FIGURE 1

Solvent use at Raygo-Wagner Company, 1977-1981.
(Date corresponds to date of receipt of solvent at Raygo-Wagner.
Each block corresponds to one 55 gallon drum).

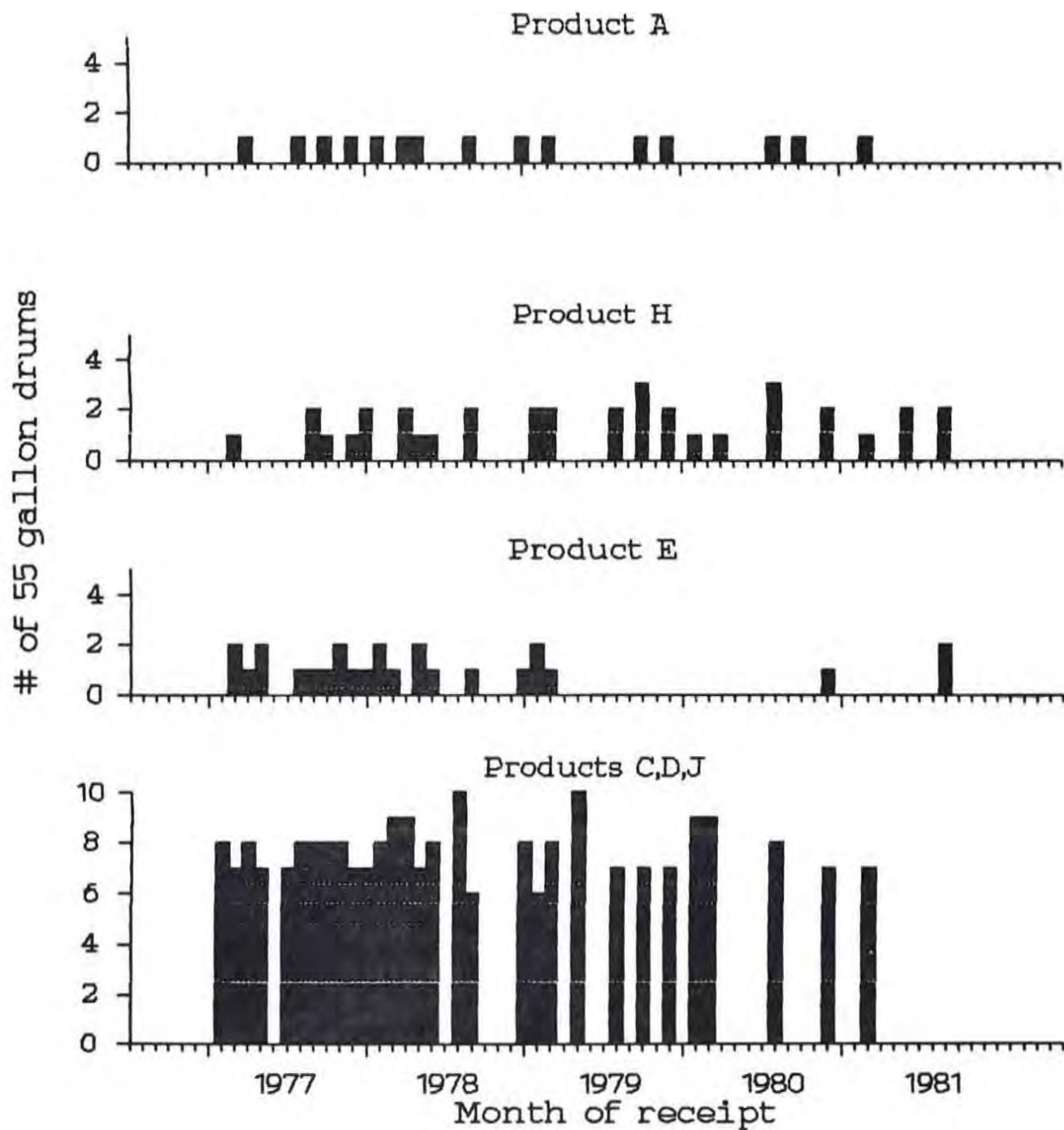
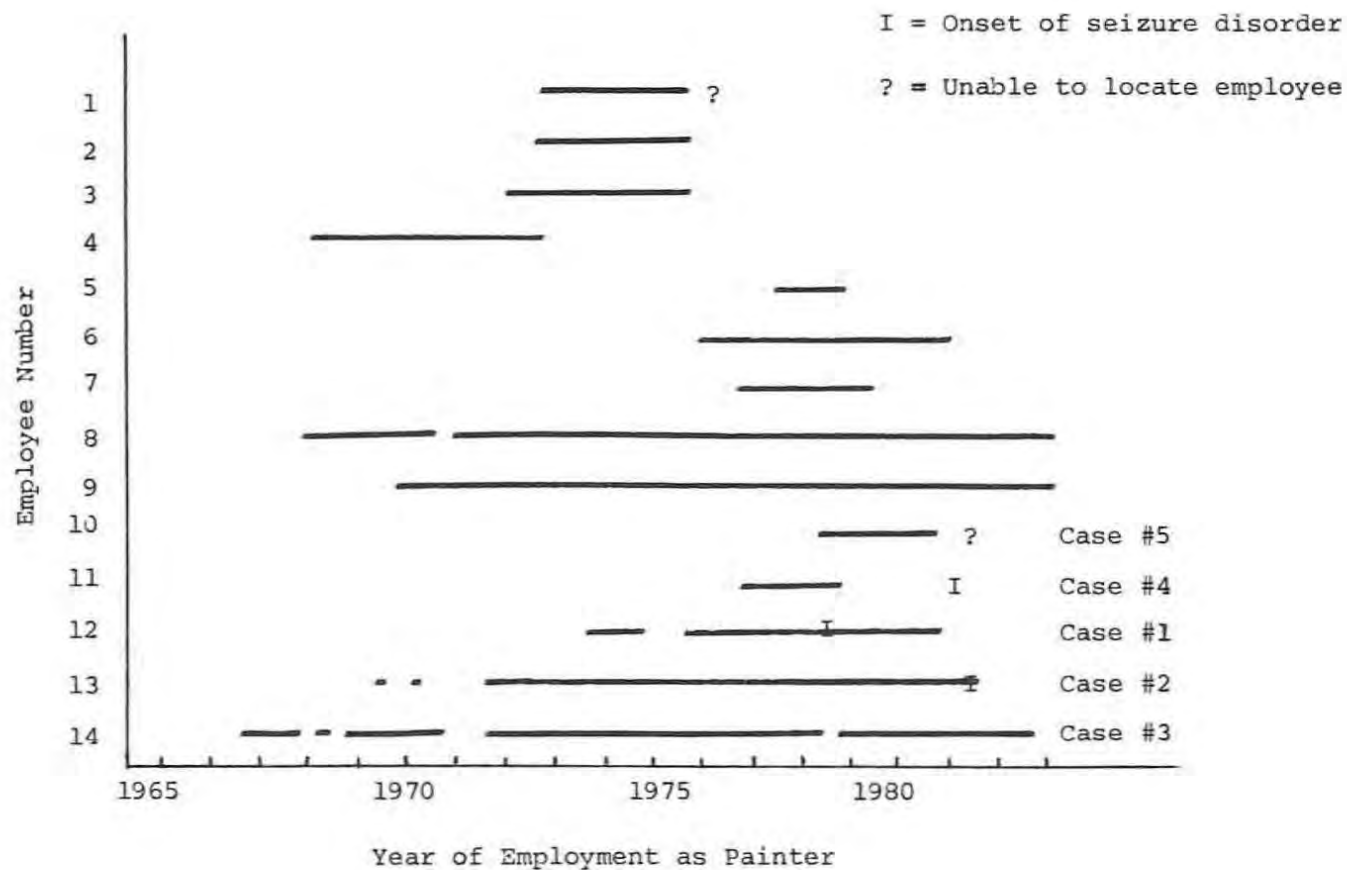


Figure 2. Employment as Painter at Raygo-Wagner.
Portland, Oregon, 1966-1983.



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