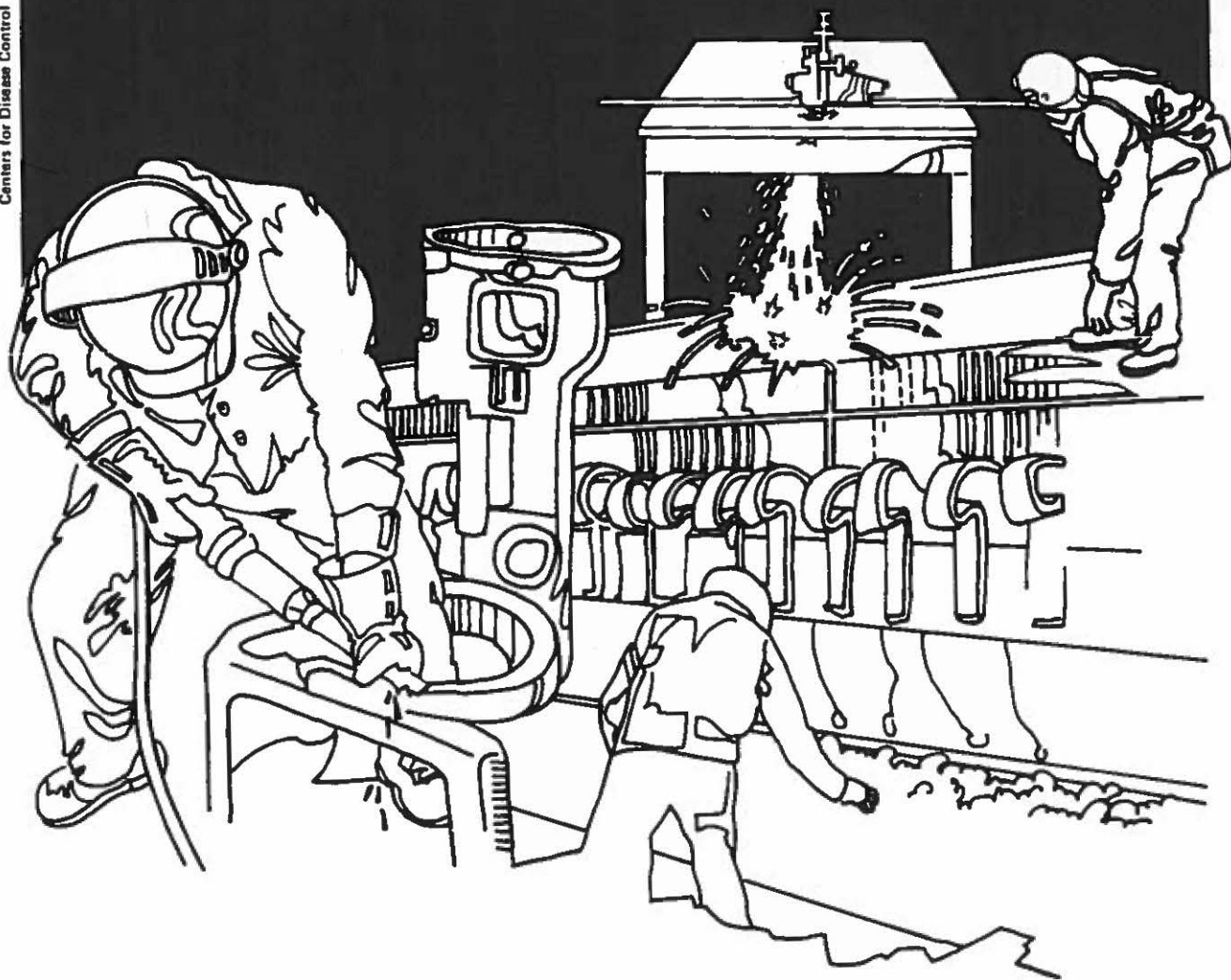


NIOSH



Health Hazard Evaluation Report

HETA 79-59-696

HEWLETT-PACKARD CO.
LOVELAND, COLORADO

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
CENTER FOR DISEASE CONTROL
NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH
CINCINNATI, OHIO 45226

HEALTH HAZARD EVALUATION DETERMINATION REPORT

HE 79-59-696

HEWLETT-PACKARD COMPANY
LOVELAND, COLORADO

JUNE 1980

I. SUMMARY

In March 1979 the National Institute for Occupational Safety and Health (NIOSH) received a request to evaluate occupational exposures to spray paint solvents, catalyst, and water-based organic coatings at Hewlett-Packard Company, Loveland, Colorado. The environmental investigation consisted of breathing zone air samples taken on all painters for specific solvents present in the paints being used. A limited physical examination and a medical questionnaire directed towards the respiratory and dermatological systems were completed on all workers. Pre-shift and post-shift pulmonary function tests were completed on all active spray painters.

At the time of the survey, there was no evidence of respiratory or dermatological problems relating to the spray painters' occupational environment. Although six abnormal spiograms were identified, no significant changes were noted between the pre-shift and post-shift values. Therefore, the abnormalities probably reflect other variables such as cigarette smoking and/or pre-existing asthma. All breathing zone and general room air samples that were taken for toluene, benzene, ethyl benzene, xylene, methyl ethyl ketone (MEK), and cellosolve acetate were well below evaluation criteria and most of the time below laboratory detection limits.

On the basis of environmental and medical data, a health hazard in the spray paint booth area did not exist. Recommendations on work practices and biological monitoring procedures necessary to prevent health hazards are included on page 7.

II. INTRODUCTION

NIOSH received a request from plant management of Hewlett-Packard Company at Loveland, Colorado, to determine if there was a health hazard from spray paint solvents, catalyst, and water-based organic

coatings used during painting small computer consoles.¹ Environmental surveys were conducted on June 20 and September 13, 1979. A medical evaluation was conducted on September 12-13, 1979. Medical and environmental data was discussed with workers and plant management.

III. BACKGROUND

The Loveland Division of Hewlett-Packard Company is responsible for the production of electrical parts as well as several types of assembled electrical equipment. At the time of this survey, the facility production schedule consisted of 3 eight-hour shifts. This evaluation was concerned with spray painters that use two waterfall-type spray booth hoods. Environmental and medical evaluations were directed at determining if these workers were overexposed to various ingredients present in paint.

IV. METHODS AND MATERIALS

A. Environmental

Toluene, ethyl benzene, xylene, benzene, cellosolve acetate, and MEK breathing zone and general room air samples were collected on organic vapor charcoal sampling tubes using vacuum pumps operated at 0.05 liters per minute and analyzed according to NIOSH Method P&CAM No. 127.

B. Medical

As the major suspected health effects were related to the respiratory and dermatological systems, all the active painters received the following:

1. An interviewer-administered questionnaire which included sections on occupational history, respiratory history, past medical history, and dermatological history.
2. A limited physical examination with special emphasis on the skin and cardiopulmonary systems.
3. Multistix urinalysis for protein, glucose, and blood was performed on a routine freshly voided random urine sample.
4. Pre-shift and post-shift spirometry was performed using an Ohio 842 dry rolling seal spirometer. The spirometry was performed with the worker in the sitting position with properly applied nose clips. At each examination, at least three or more expiratory curves were obtained until there was

¹Section 20(a)(6) of the Occupational Safety and Health Act of 1970, 19 U.S.C. 669(a)(6), authorizes the Secretary of Health, Education, and Welfare, following a written request by any employer or authorized representative to employees, to determine whether any substance normally found in the place of employment has potentially toxic effects in such concentrations as used or found.

less than 5 percent variation in the forced vital capacity (FVC) measurement. All curves were hand calculated and the best curve (largest sum of FVC and forced expiratory volume in one second [FEV₁]) was used for interpretation. Intermountain Thoracic Society (ITS) standards were used for predicted values. (Reference 1) The ITS graduated scale of interpretations for obstruction and restriction was used (see Table 1). (Reference 1) In comparing pre-shift and post-shift values a 20 percent decrease in the FEV₁ was considered to be significant. (Reference 2) In addition, previous spirometric values were obtained from the company's medical department for comparison to the pre-shift baseline values.

Descriptive statistics in the form of frequency distributions and/or mean, standard deviation (SD), and range were reported for the variables outlined in Tables 4 through 6. Matched paired t test analysis was used to compare postversus pre-shift and pre-shift versus post-shift spirometric variables (FVC, FEV₁, FEF₂₅₋₇₅). (Reference 3)

V. EVALUATION CRITERIA

A. Environmental

Three sources of criteria used to assess the workroom concentrations of the following chemicals are: (1) recommended Threshold Limit Values (TLVs) and their supporting documentation as set forth by the American Conference of Governmental Industrial Hygienists (ACGIH), 1979; (2) NIOSH criteria for recommended standards; (3) Occupational Safety and Health Administration (OSHA) Standards (29 CFR 1910), January 1978.

	Permissible Exposure Limits 8-Hour Time-Weighted Exposure Basis (mg/M ³)	
Ethyl Benzene.....	435.0 (NIOSH)	435.0 (OSHA)
Benzene.....	32.0 (NIOSH)	32.0 (OSHA)
Methyl Ethyl Ketone.....	590.0 (TLV)	590.0 (OSHA)
Xylene.....	435.0 (NIOSH)	435.0 (OSHA)
Toluene.....	375.0 (NIOSH)	375.0 (OSHA)
Cellosolve Acetate.....	270.0* (TLV)	540.0 (OSHA)
Toluene-2,4-diisocyanate (TDI).**	0.04 (NIOSH)	0.14 (OSHA)
Polyaziridine (ethylenimines).**	---	---

mg/M³ = milligrams of substance per cubic meter of air

* = 1979 Notice of Intended Change

** = These chemicals were not being used at the time of this survey. Their toxicology is included since past exposure may be responsible for present pulmonary function abnormalities.

Occupational health standards are established at levels designed to protect individuals occupationally exposed to toxic substances on an 8-hour per day, 40-hour per week basis over a normal working lifetime.

B. Toxicological

Xylene -- Xylene vapor is an irritant of the eyes, mucous membranes, and skin. Gastrointestinal disturbances such as anorexia, nausea, vomiting, and abdominal pain can also occur. Narcosis may occur at high concentrations.

Toluene -- Toluene is slightly irritating to the eyes and mucous membranes. It is toxic by ingestion, inhalation, and skin absorption. Acute poisoning from toluene vapors is rare. Inhalation of 200 parts per million for an 8-hour period will cause impairment of coordination and reaction time. Toluene also produces narcosis. There have been reports of chronic poisoning described as anemia and leucopenia. Biopsy showed bone marrow hypoplasia. (Reference 4)

Methyl Ethyl Ketone (MEK) -- MEK is very irritating to the eyes, nose, and upper respiratory system. At levels below the TLV, it may produce narcosis. Levels found during this study were far below the evaluation criteria.

Benzene -- Benzene is highly toxic either by inhalation or skin absorption. Benzene is metabolized in the body to a phenolic compound which may alter the DNA molecule in bone marrow with injury to blood forming tissue. It produces liver necrosis and is also a central nervous system (CNS) depressant. Benzene is a carcinogen producing leukemia in certain individuals. Benzene is known to cause aplastic anemia, macrocytosis, leucopenia, thrombocytopenia, and hemolysis. (Reference 5)

Ethyl Benzene -- Ethyl benzene is toxic by all three routes of entry (ingestion, inhalation, skin absorption). It is an irritant and CNS depressant. Ethyl benzene may produce pulmonary edema and hepatitis. First symptoms of overexposure include upper respiratory tract irritation, chest constriction, dizziness, and narcosis. There have been no permanent effects reported. (Reference 6)

Cellosolve Acetate -- Cellosolve acetate used in fast drying paints or lacquers may produce irritation of respiratory tract. It also produces narcosis and defatting dermatitis. (Reference 7)

Toluene-2,4-diisocyanate (TDI) -- Present knowledge regarding diisocyanate toxicity has largely been achieved as a result of investigations into the effects of its most widely used derivative, toluene diisocyanate (TDI). Liquid at room temperature, this highly volatile substance possesses a vapor pressure of 0.04mm at 20°C. Its pungent, acrid odor is detectable at concentrations of 0.005ppm. (Reference 8) At levels of 0.5ppm, exposed workers complain of itchy eyes, nasal congestion, and a dry, sore throat. (Reference 8) At higher concentrations, breathlessness, chest tightness, wheezing, and cough may occur. (References 9,10) High exposures may lead to pulmonary edema. (Reference 3) As a rule, skin involvement is usually absent; (Reference 8) however, with prolonged contact,

redness, swelling, and blistering have been noted. (Reference 11) Less common symptoms resulting from TDI exposure include nausea, vomiting, abdominal pain, and occasionally a throbbing headache. (References 8,11) In addition to the irritating effects of TDI, approximately 5 percent of exposed workers develop signs of respiratory sensitization. (Reference 12) The range of onset of sensitization from initial exposure varies from 6 months to 23 years with a mean of about 3 years. (Reference 13) Once sensitization has occurred, exposure to concentrations of TDI as low as 0.0018ppm can provoke asthma-like symptoms including shortness of breath, wheezing, dyspnea, and cough. (Reference 10) These reactions may occur within minutes or hours of exposure, and occasionally a dual reaction is noted with both an immediate and a late (delayed) response. The nature of this sensitization is still unclear; however, pharmacological rather than allergic mechanisms may be involved. (Reference 2)

Polyaziridine -- Aziridines or ethylenimines are a group of organic compounds characterized by a three-membered ring, consisting of two carbon and one nitrogen atom. Liquid at room temperature, ethylenimine is a colorless fluid with an ammoniacal odor and vapor pressure of 160mm at 20°C. (Reference 14) This highly reactive monomer is thermodynamically less stable than the polymeric form. Slow polymerization occurs in an aqueous media; however, typically the process is catalyzed by strong protonic acids. (Reference 15) The polymerization products, known as polyethylenimines or polyaziridines, have found numerous industrial applications. In finishing paints, they are used for improved adhesion and solvent resistance. (Reference 15) When compared with the monomeric form, polyaziridines are considered to be much less toxic. (References 14,15) They have been tested orally to combat hyperacidity and bind bile acids in the intestinal tract; no unusual toxicity was noted. (Reference 15) Despite the apparent innocuous nature of polyaziridines, it is important to consider the toxic effects of possible degradation products. Evans et al, have reported that the pyrolysis of 100gm of polyethylenimine at 500°C resulted in the production of 2.0mg. of ethylenimine as analyzed by high pressure liquid chromatography. (Reference 16) Ethylenimine is included in the federal standard for carcinogens and it is recommended that all contact with it be avoided. (Reference 11) Concentrations of 25ppm for an 8-hour period have been shown fatal to rats and guinea pigs by a combination of lung injury and renal tubular necrosis. (Reference 14) Ethylenimine is a skin sensitizer and a necrotizing agent. (Reference 17) Exposure can result in severe irritation to the conjunctiva and cornea as well as to the mucous membranes of the upper respiratory tract. Besides the production of ethylenimine, polyaziridines heated in air at 90°C will completely disappear into volatile products within 90 days by a combination of autooxidation and piperazine formation. (Reference 15) Piperazines have been used medicinally in the treatment of a number of disorders including ascariasis and enterobiasis. Systemic absorption is variable and the drug is excreted essentially unchanged. Toxic effects known to occur from excessive dosage include nausea, vomiting, diarrhea, and abdominal pain as well as occasional neurotoxicity. (Reference 18) In addition, skin and respiratory sensitization have been

reported in individuals with repeated exposures. Once sensitized, dermal contact can result in erythema and vesiculation. (Reference 19) Pepys et al, have demonstrated that occupational exposure to piperazine dust can provide late asthmatic reactions with symptoms consisting of rhinorrhea, lacrimation, cough, wheezing and shortness of breath noted three to four hours after inhalation. (Reference 20)

VI. RESULTS

A. Environmental

All painters were monitored for each chemical present in the paints they were using. General room samples were also obtained. All breathing zone and general room samples were below evaluation criteria used here and often below laboratory detection limits. Ventilation measurements taken showed an average of 250 linear feet per minute air velocity in the breathing zone of painters. This is very adequate ventilation, which is additionally verified by the extremely low results obtained on the breathing zone air samples. Painters were well educated on the use of the paint booths and toxicity of the paints they were using. Excellent hygiene was also practiced by all painters.

B. Medical

Although the water-based paint with TDI and polyaziridine was no longer in use at the time of the hazard evaluation, the survey was conducted to determine if the complaints attributed to this catalyst had abated. The demographic characteristics of the cohort are shown on Table 4. When analyzed in terms of cigarette smoking history, little difference can be found among the current smokers versus the exsmokers versus the nonsmokers. However, the two individuals suspected of having problems secondary to the catalyst exposure had significantly shorter lengths of employment as spray painters (3 months and 6 months). The historical data obtained from the medical questionnaires is shown in Table 5. Although there appears to be an increased prevalence of allergy and dermatological problems, an association between the symptoms and exposure to TDI and polyaziridine could be found only in the two workers who had been moved to another work area as the result of a rash occurring during the period when the paint containing TDI and polyaziridine was being used. The physical examinations revealed a number of minor abnormalities, none of which could be associated with the workers' present occupational exposures (see Tables 2 and 3). All the urinalyses were normal.

The spirometric data is summarized in Table 6. Matched paired t tests comparing previous values with pre-shift values revealed a significant difference (see Table 6) in the FVCs and FEV₁s. The pre-shift values are consistently larger than the previous values which we feel can be attributed to a difference in spirometric technique and technicians. The column labeled "Previous Measurement" was prepared by Hewlett-Packard nurses and the testing was done prior to any exposure. The previous values were obtained by extrapolation from a flow volume loop whereas the pre-shift values were calculated directly from an expiratory flow

curve. Only simultaneous testing of an individual by both techniques would prove whether or not the difference in values could be attributed to technique. When pre-shift values are compared to post-shift values, there is no statistically significant difference (see Table 6). In addition, no individual manifested a 20 percent change in his FEV₁ during his work shift. Six workers were noted to have mild airways obstruction based on a decreased FEF₂₅₋₇₅/FVC ratio (see Figures 1, 2, and 3). All of these individuals were either cigarette smokers or had historical evidence of asthma. The degree or type of abnormality is apparently not affected by the spray painters' present occupational exposures.

VII. DISCUSSION AND CONCLUSIONS

A health hazard did not exist at this work place at the time of this evaluation. This conclusion is based on the medical and environmental data. At the time of the survey, there was no evidence of respiratory or dermatological problems relating to the spray painters' occupational environment.

Although six abnormal spirograms were identified, no significant changes were noted between the pre-shift and post-shift values. Therefore, the abnormalities probably reflect other variables such as cigarette smoking and/or pre-existing asthma.

III. RECOMMENDATIONS

1. Smoking, eating, and drinking must be prohibited in the work area.
2. The water in the waterfall paint booths should be cleaned regularly.
3. If the paint with TDI and polyaziridine is reinstituted in the spray painters' environment, close evaluation of pre-shift and post-shift spirometry will be necessary. Patch testing for dermatological problems would be useful in identifying the sensitized individuals and in instituting proper control measures. Worker education programs on respiratory usage and protective clothing would also help prevent disease.
4. The affected workers should avoid subsequent exposure to the catalyst or water-based paints containing the catalyst.
5. Further basic research on the degradation of catalyst TDI and polyaziridine would be useful. If the amount of piperazine or ethylenimine monomer is found to be significant, respiratory tract protection measures should be evaluated to be certain that the workers are appropriately protected.

IX. REFERENCES

1. Kanner RE, Morris AH, eds: Clinical pulmonary function testing: A manual of uniform laboratory procedures for the intermountain area. Salt Lake City, Utah: Intermountain Thoracic Society, 1975.
2. Butcher BT, Karr RM, O'Niel CE, et al: Inhalation challenge and pharmacological studies of TDI sensitive workers. J Allergy Clin Immunol 64:146 1979

3. Colton T: Statistics in medicine. Boston: Little, Brown and Company, 1974.
4. Sax, N. Irving. Dangerous Properties of Industrial Materials, Fourth Edition, Van Nostrand Reinhold Company, 1975, p. 1174.
5. Plunkett, ER. Handbook of Industrial Toxicology, Chemical Publishing Company, New York, 1976, pp. 50-52.
6. Ibid., p. 164-165.
7. Proctor, NH, Hughes, JP. Chemical Hazards of the Workplace, J.B. Lippincott Company, 1978, p. 14.
8. Brugsch HG, Elkins HB: TDI toxicity. New Engl J Med 268:353, 1963.
9. Hama GM: Symptoms in workers exposed to isocyanates. Archives of Industrial Hygiene 16:232, 1957.
10. Parkes WR: Occupational lung disorders. London: Butterworth and Company, 1974, pp. 450-457.
11. Key MM, Henschel AF, Butler J, Ligo RN, Tabershaw IR: Occupational diseases: A guide to their recognition. Washington, D.C.: National Institute for Occupational Safety and Health, 1977. (DHEW (NIOSH) Publication No. 77-181.)
12. Karr RM, Davies RJ, Butcher BT, et al: Occupational asthma. J Allergy Clin Immunol 61:54, 1978.
13. Chester EH, Schwartz HJ: Study session on occupational asthma. J Allergy Clin Immunol 64, 1979.
14. Carpenter CD, Smyth HF, Shaffer CB: The acute toxicity of ethylenimine to small animals. Journal of Industrial Hygiene and Toxicology 30:2, 1948.
15. Dermer OC, Ham GE: Ethylenimine and other aziridines. New York: Academic Press, 1969.
16. Evans DJ, Mayfield RJ, Russell IM: Rapid estimation of trace amounts of ethylenimine by high pressure liquid chromatography. J Chromatogr 115: 391, 1975.
17. Dernehl CV: Clinical experiences with exposures to ethylene amines. Industrial Medicine and Surgery 20:541, 1951.
18. Litton Industries: Physicians' desk reference. 32nd ed. Ordell, New Jersey: Medical Economics Company, 1978.
19. McCullagh SF: Allergenicity of piperazine: A study in environmental aetiology. Br J Ind Med 25:319, 1968.
20. Pepys J, Pickering AC, Loudon HWG: Asthma due to inhaled chemical agents--piperazine hydrochloride. Clin Allergy 2:189, 1972.

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XI. DISTRIBUTION AND AVAILABILITY

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Copies of this report have been sent to:

1. Hewlett-Packard Company.
4. U.S. Department of Labor/OSHA - Region VIII.
5. NIOSH - Region VIII.
6. Colorado Department of Health.
7. State Designated Agency.

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

Table 1
Criteria for Spirometric Interpretation

Obstructive
Lung Disease
Criteria

Categories	FEV ₁ /FVC	FEF ₂₅₋₇₅ %/FVC	FEV ₃ /FVC
Normal	>0.69	and >0.65	--
"Severe"	<0.45	--	--
"Moderate"	0.45-0.60	--	--
"Mild"	0.61-0.69	or <0.65	--
"Suggested but probably normal spirometry"	--	--	<0.93

Restrictive
Lung Disease
Criteria

Categories	FVC/FVC pred
Normal	<u>≥</u> 0.81
"Severe"	<u>≥</u> 0.50
"Moderate"	0.51-0.65
"Mild"	0.66-0.80

Table 2

Breathing Zone and General Room Air Concentrations of
Benzene, Toluene, Xylene, Methyl Ethyl Ketone (MEK), and Cellosolve Acetate

Hewlett-Packard Company
Loveland, Colorado

June 20, 1979

Sample Number	Job Classification	Sampling Time	mg/M ³				
			Benzene	Toluene	Xylene	MEK	Cellosolve Acetate
1	Spray Painter	8:50 AM - 11:20 AM	*	*	*	*	*
2	Spray Painter	8:57 AM - 11:20 AM	*	*	*	*	*
3	Spray Painter	9:00 AM - 11:20 AM	*	*	*	*	*
4	General Room	9:12 AM - 2:12 PM	*	*	*	*	*
5	Spray Painter	12:10 PM - 2:10 PM	*	*	*	*	*
6	Spray Painter	12:00 N - 2:08 PM	*	*	*	*	*
7	Spray Painter	11:58 AM - 2:00 PM	*	*	*	*	*
EVALUATION CRITERIA			32	375	435	590	270
LABORATORY LIMIT OF DETECTION mg/sample			0.01	0.01	0.02	0.01	0.03

* = below laboratory limit of detection

Table 3

Breathing Zone and General Room Air Concentrations of
Toluene, Ethyl Benzene, and XyleneHewlett-Packard Company
Loveland, Colorado

September 13, 1979

Sample Number	Job Classification	Sampling Time	mg/M ³		
			Toluene	Ethyl Benzene	Xylene
1	Painter	8:12 AM - 1:49 PM	1	*	*
2	Painter	8:12 AM - 10:30 AM	2	*	*
3	Painter	8:12 AM - 10:30 AM	1	*	1
4	Painter	8:15 AM - 1:49 PM	3	1	4
5	General Room	8:10 AM - 1:48 PM	1	*	*
6	General Room	8:10 AM - 1:48 PM	1	*	*
EVALUATION CRITERIA			375	435	435
LABORATORY LIMIT OF DETECTION mg/sample			0.01	0.01	0.02

* = below laboratory limit of detection

Table 4.

Demographic Characteristics of Cohort

	Current Smokers (n=6)	Exsmokers (n=5)	Nonsmokers (n=6)	Total Group (n=17)
Age				
Mean	29.67	34.40	32.50	32.06
SD	8.57	13.94	7.64	9.68
Range	22-41	20-53	24-43	20-53
Length of Employment (in months)				
Mean	10.00	36.50	27.83	24.09
SD	7.13	48.23	48.41	38.18
Range	3-22	1.5-120	2-126	1.5-126
Years of Formal Education				
Mean	12.00	12.60	11.83	12.12
SD	1.10	0.89	1.60	1.22
Range	10-13	12-14	9-14	9-14

Table 5
Summary of Medical Histories

Medical History	Current Smokers (n=6)	Exsmokers (n=5)	Nonsmokers (n=6)	Total Group (n=17)
Respiratory symptoms				
Chronic bronchitis	1	0	1	2
Dyspnea on exertion	3	1	1	5
History of asthma	0	2	0	2
Dermatological symptoms				
History of skin disease				
a. past	1	2	2	5
b. recent (within 1 year)	2	0	2	4
c. family	0	0	1	1
History of allergies	2	3	3	8
History of hospitalizations	3	3	1	7
History of nasal polyps	0	2	0	2
History of sinusitis	2	1	2	5
History of conjunctivitis	0	0	0	0
History of heart disease	0	1	1	2
History of weight loss	0	2	1	3
History of jaundice	0	0	1	1
History of hepatitis	0	0	1	1
History of nephritis	0	0	0	0
History of endocrine disease	0	0	1	1
History of neurological disease	2	1	0	3
History of arthritis	0	1	1	2

Table 6
Selected Spirometric Tests Compared by Previous Measurement and Shift
(FVC and FEV₁ in liters, FEF₂₅₋₇₅ in liters per second)

ID Number	Test	Previous Measurement	Preshift Measurement	Change from Previous to Preshift Measurement	Postshift Measurement	Change from Preshift to Postshift Measurement	Predicted Measurement
* 1	FVC	4.45	4.77	0.32	4.60	-0.17	3.97
	FEV ₁	2.67	3.01	0.34	2.87	-0.14	3.16
	FEF ₂₅₋₇₅	1.30	1.29	-0.01	1.12	-0.17	3.96
2	FVC	4.74	5.57	0.83	5.69	0.12	4.80
	FEV ₁	4.08	4.46	0.38	4.75	0.29	4.13
	FEF ₂₅₋₇₅	5.23	4.22	-1.01	4.99	0.77	5.23
* 3	FVC	4.77	4.65	-0.12	4.80	0.15	4.47
	FEV ₁	3.35	3.24	-0.11	3.40	0.16	3.67
	FEF ₂₅₋₇₅	1.38	1.99	0.61	2.10	0.11	4.54
4	FVC	6.74	7.30	0.56	7.10	-0.20	4.85
	FEV ₁	4.07	5.10	1.03	5.05	-0.05	4.17
	FEF ₂₅₋₇₅	3.94	3.61	-0.33	3.59	-0.02	5.25
5	FVC	4.81	5.20	0.39	5.70	0.50	4.55
	FEV ₁	3.65	4.02	0.37	4.14	0.12	3.65
	FEF ₂₅₋₇₅	3.11	3.89	0.78	3.96	0.07	4.39
* 6	FVC	5.14	5.90	0.76	5.90	0.00	4.80
	FEV ₁	4.21	4.40	0.19	4.52	0.12	4.13
	FEF ₂₅₋₇₅	4.54	3.60	-0.94	3.51	-0.09	5.23
7	FVC	5.02	5.10	0.08	5.00	-0.10	4.88
	FEV ₁	3.34	4.34	1.00	4.17	-0.17	4.27
	FEF ₂₅₋₇₅	3.55	4.90	1.35	4.24	-0.66	5.47

Table 6 (con't)

ID Number	Test	Previous Measurement	Preshift Measurement	Change from Previous to Preshift Measurement	Postshift Measurement	Change from Preshift to Postshift Measurement	Predicted Measurement
* 8	FVC		5.40		5.30	-0.10	4.90
	FEV ₁		3.86		3.93	0.07	4.20
	FEF ₂₅₋₇₅		2.84		2.73	-0.11	5.27
* 9	FVC	5.75	6.29	0.54	6.11	-0.18	5.49
	FEV ₁	4.66	5.36	0.70	5.15	-0.21	4.71
	FEF ₂₅₋₇₅	5.78	6.17	0.39	6.11	-0.06	5.73
10	FVC	5.46	5.70	0.24	5.90	0.20	4.29
	FEV ₁	4.25	4.55	0.30	4.71	0.16	3.53
	FEF ₂₅₋₇₅	4.22	4.60	0.38	4.76	0.16	4.46
*11	FVC	4.80	5.21	0.41	5.40	0.19	4.85
	FEV ₁	4.08	4.35	0.27	4.42	0.07	4.17
	FEF ₂₅₋₇₅	5.06	4.65	-0.41	4.74	0.09	5.25
*12	FVC		4.65		4.77	0.12	5.08
	FEV ₁		4.34		4.45	0.11	4.33
	FEF ₂₅₋₇₅		6.28		6.46	0.18	5.36
*13	FVC	5.29	5.85	0.56	5.65	-0.20	5.35
	FEV ₁	4.77	5.10	0.33	4.99	-0.11	4.61
	FEF ₂₅₋₇₅	6.05	6.09	0.04	6.14	0.05	5.67
14	FVC	4.80	4.60	-0.20	4.69	0.09	4.90
	FEV ₁	3.30	3.44	0.14	3.40	-0.04	3.97
	FEF ₂₅₋₇₅	2.29	2.84	0.55	2.61	-0.23	4.73
15	FVC	5.19	5.17	-0.02	5.40	0.23	4.77
	FEV ₁	4.25	4.27	0.02	4.43	0.16	3.96
	FEF ₂₅₋₇₅	4.23	4.97	0.74	4.82	-0.15	4.85

Table 6 (con't)

ID Number	Test	Previous Measurement	Preshift Measurement	Change from Previous to Preshift Measurement	Postshift Measurement	Change from Preshift to Postshift Measurement	Predicted Measurement
16	FVC FEV ₁ FEF ₂₅₋₇₅						
*17	FVC FEV ₁ FEF ₂₅₋₇₅	4.89 4.42 6.50	5.25 4.97 7.95	0.36 0.55 1.45			4.92 4.30 5.49

Significance by Matched Paired t test

FVC	$p < 0.002$	NS
FEV ₁	$p < 0.001$	NS
FEF ₂₅₋₇₅	NS	NS

Significance by matched paired t test of smokers and nonsmokers

Smokers FVC	$p < 0.009$	NS
FEV ₁	NS	NS
FEF ₂₅₋₇₅	NS	NS

Nonsmokers FVC	NS	NS
FEV ₁	$p < 0.02$	NS
FEF ₂₅₋₇₅	NS	NS

*Indicates smokers (both current and ex).

Figure 1: FEF₂₅₋₇₅ Determinations in Workers with Abnormal Spirometries

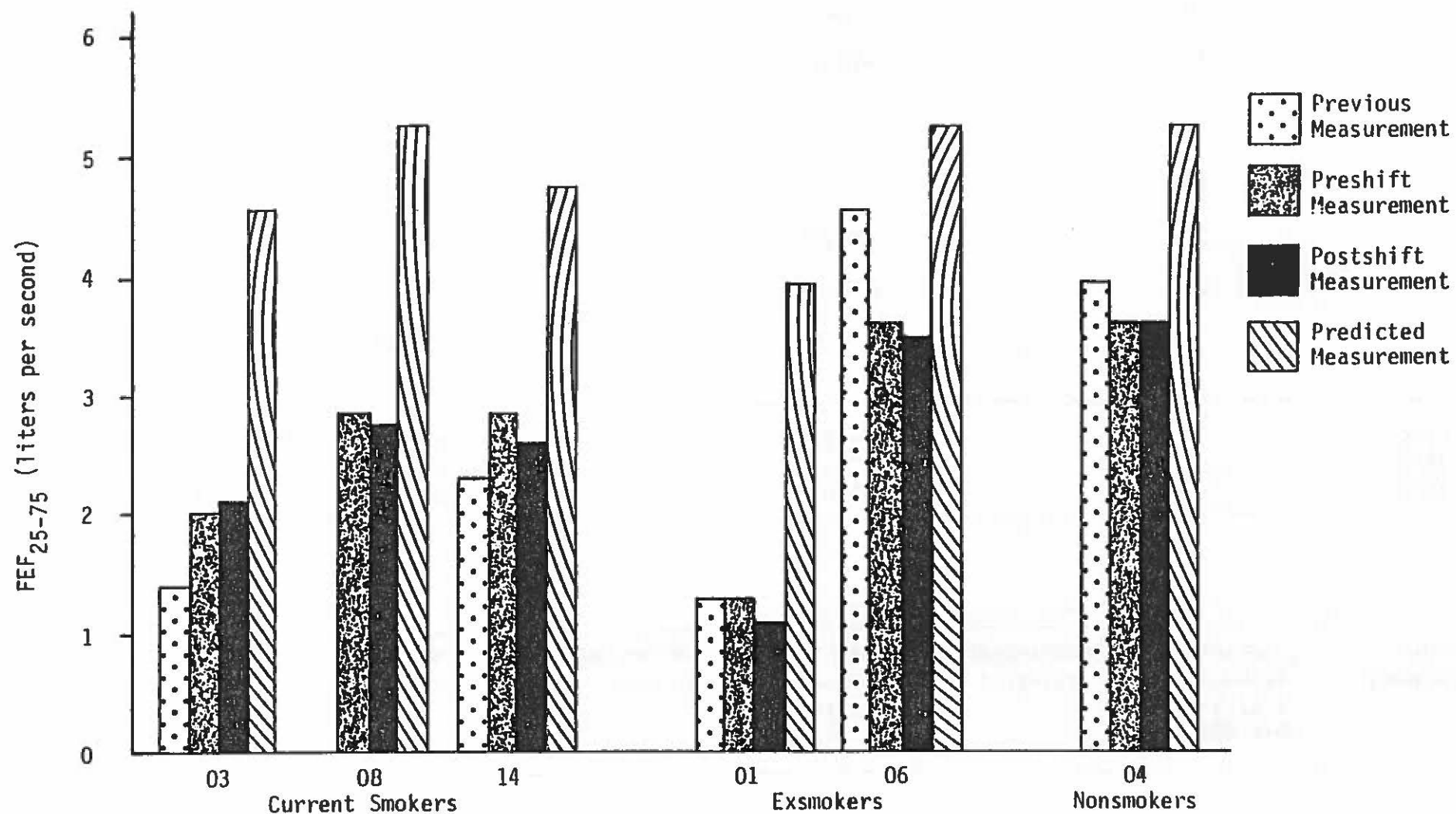


Figure 2: FVC Determinations in Workers with Abnormal Spirometries

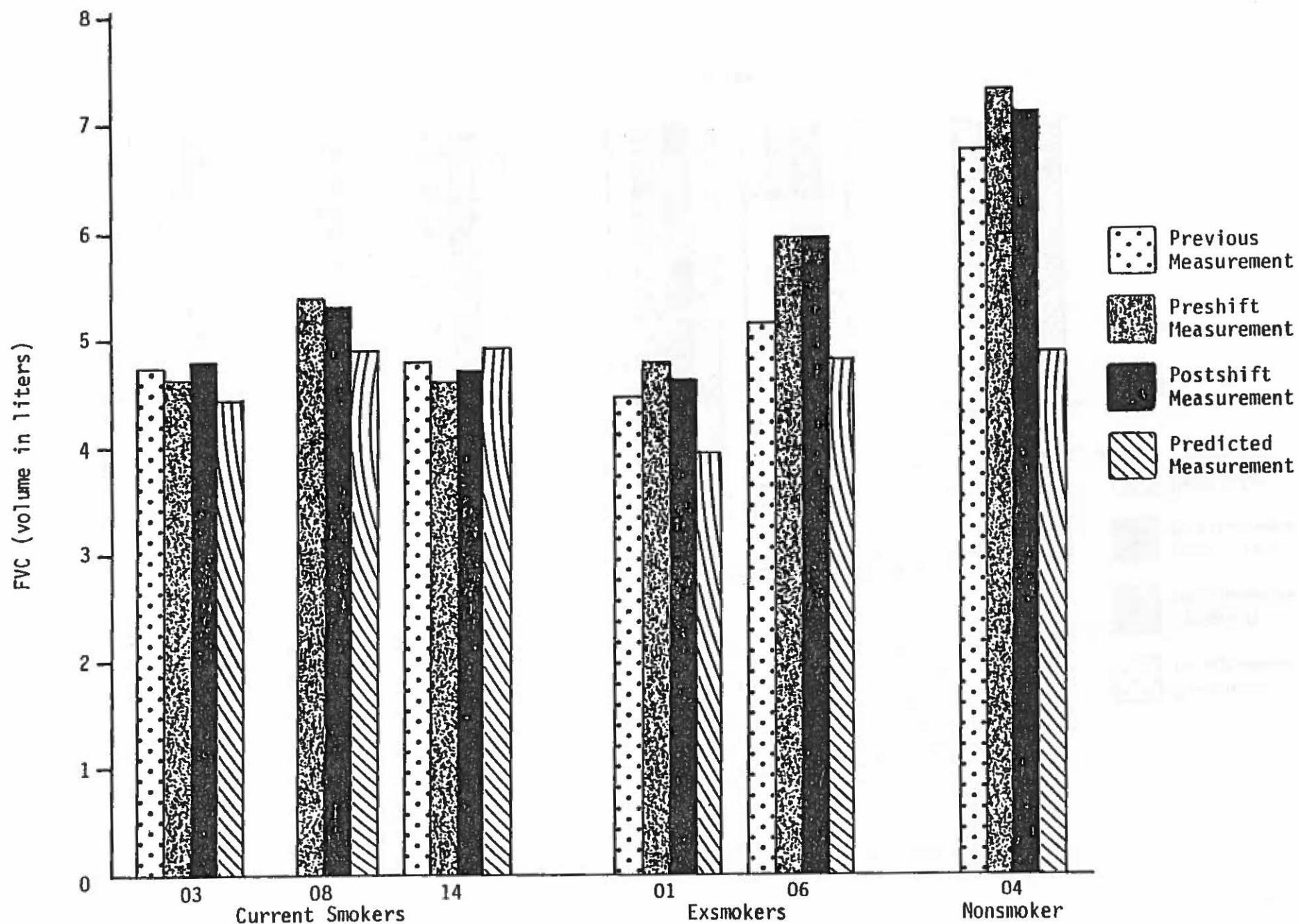


Figure 3: FEV₁ Determinations in Workers with Abnormal Spirometries

