Prevalenc of disease among vinyl chloride and polyvinyl chloride workers

by

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The emergence in January 1974, of vinyl chloride as a new occupational carcinogen, producing hemangiosarcoma of the liver focused new and much enhanced attention on the toxicologic effects of this chlorinated hydrocarbon.

A considerable number of studies had been published on the rather puzzling and unique syndrome of "acroosteolysis" (Table 1) mainly describing the bone lesions in the distal phalanges of the fingers, but also the associated scleroderma-like skin lesions and the striking vascular (arterial and arteriolar) changes, pathologically the main lesion, leading to secondary bone and skin changes (Tables 2 and 3).

Much less attention had been given to the hepatotoxic effects of vinyl chloride, although convincing experimental evidence had been produced (Torkelson et al., 1961) and scattered reports of ill-defined liver changes were included in some of the studies of vinyl chloride exposed workers (Table 4).

There was evidence that liver damage occurred in workers exposed to higher levels of vinyl chloride. In some studies the liver changes (hepatomegaly, tenderness on palpation, abnormal liver tests) were found to be reversible after cessation of exposure. On the other hand, some observations mentioned the progressive nature of the liver changes, when toxic exposure continued to what was stated to be "chronic hepatitis".

Recent work from Germany (Bernstein et al., 1973) reporting a detailed study of 20 PVC workers, brought a very important contribution to our present knowledge on the liver changes induced by this toxic exposure. The authors were alerted to the problem by the occurrence of upper gastrointestinal bleeding in several workers of a PVC producing plant. Esophageal varices, splenomegaly, hepatomegaly, fibrosis of the liver capsule (by laparoscopy), delay in the secretion of bromsulphalein, thrombocytopenia were the most prominent findings. The pathologic changes encompassed collagenous transformation of the liver sinusoids, focal activation of Kupffer cells, increased collagen deposition in the portal spaces, portal fibrosis, septal and intralobular fibrosis.

There are a few mentions in the literature on lung changes in PVC workers. One case of pneumonconiosis in a thirty year old worker who had inhaled PVC dust was reported. The lung biopsy showed granulomatous lesions (foreign body type) to be present. Fibrotic changes and altered pulmonary function tests were reported in 96 workers exposed to polyvinyl chloride dust; the changes were more pronounced in persons with long exposure (Vertkin et al. 1970).

In the available literature on vinyl chloride and polyvinyl chloride adverse health effects there were no accurate data on the prevalence of disease in exposed workers, the relative prelavence of the various pathologic changes, their possible association and time sequence.

We have undertaken clinical studies of a number of groups of vinyl chloride exposed workers in order to assess the prevalence of vinyl chloride induced changes and to define the natural history of this occupational disease. We are reporting the results of a survey (March 1974) of 267 workers currently employed in a VC polymerization plant, including virtually the entire current production workforce. 87 former workers were examined as well. In both groups, exposure for some had started in 1945, when the plant opened. Altogether 354 men were examined.

Table 5 shows the duration of exposure for the group and Table 6 the age distribution of the men. More than half were under 40 years, 20% were 51 or older.

METHODS

The medical examination included detailed occupational history, past medical history, complete physical examination, with special attention to th skin, hands and feet, arteries accessible to palpation, liver and spleen.

An Allen test for the assessment of the peripheral circulation in th areas supplied by the radial and ulnar arteries was performed. Each examined worker also had the following:

X-rays of the chest, hands and feet; pulmonary function tests; complete blood cell counts including platelets; blood chemistries including bilirubin, SGOT, SGPT, LDH, alkaline phosphatase, total protein, electrophoresis of serum proteins, fibrinogen split products; alpha fetoprotein, antinuclear antibodies, antimitrchondrial antibodies, antismooth muscle antibodies Australia antigen B by radioimmunoassay and liver antibodies. Urinalysis, including microscopic examination of the sediment was done. All examined workers were also tested for carcinogenic embryonic antigen. In a limited number, studies for chromosome breaks in circulating lymphocytes were undertaken.

RESULTS

Unfortunately, appropriate VC measurements had not been made in th past. We have findings which suggest, however (Table 7), that VC levels may have been considerable at times, sufficient to produce symptoms of acute overexposure; for example, 14 of the 354 workers had had loss of consciousness at one time or another.

In any case, our data indicate that measured levels would be of limited utility, unless integrated over time, since both total exposure and duration from onset of exposure seem to be of importance in relation to likelihood of abnormality. The influence of these variables may b demonstrated in a number of areas.

This was so insofar as abnormal periph ral circulation was concerned (Table 8).

Numbness and tingling of the fingers, along with increased sensitivity to cold were early symptoms. They were often associated with pain and cyanotic discoloration sometimes also involving the toes. Clasical Raynaud's syndrome, characterized by cold-induced, marked, sudden and sharply demarcated pallor of the fingers followed by cyanotic discoloration in the same area, was found only in 5,6% of the cases. In general, the effect of vinyl chloride exposure on the peripheral vessels of hands and feet appears to produce a wider range of symptoms than those characteristic of Raynaud's syndrome.

As noted, the prevalence of these changes rose with duration of exposure. Typical Raynaud's syndrome was found in almost 10% of cases with more than 20 years of exposure. The prevalence of Raynaud's syndrome was significantly higher in workers with more than 10 years of exposure, than in those with less than 10 years exposure. Symptoms of abnormal peripheral circulation (Table 9) were more frequent in workers currently exposed than in those with past exposure.

An abnormal Allen test indicating delayed arterial circulation in the areas supplied by the ulnar and/or radial arteries, was found in 94 (26.6%) of the examined workers (Table 10).

The percentage of persons with abnormal Allen tests increased with their length of exposure. The prevalence of an abnormal Allen test in workers with more than 5 years exposure was significantly higher than in those with less than 5 years exposure.

Pseudo-clubbing of the fingers was also found with increasing frequency as the duration of employment increased; the overall prevalence was 8,7%; in the group with more than 20 years of exposure it was 17,3% (Table 11). However the difference in the prevalence of pseudo-clubbing between workers with less and more than 5 years of exposure did not quite reach the level of statistical significance. ($\chi^2 = 3,602$) Pseudo-clubbing was almost always associated with signs and/or symptoms of abnormal peripheral circulation, and was a more frequent finding that typical Raynaud's syndrome. In some cases, especially in persons with past exposure, there was a history of Raynaud's syndrome which had gradually faded, while pseudo-clubbing had persisted or even progressed.

Skin changes (Table 12) involving the fingers, hands, forearms and sometimes face, were found in 23 examined workers. The fingers and hands appeared to be slightly edamatous, the skin was thickened, tense and stiff, with decreased elasticity and sparse folding. Well circumscribed plaque r band areas of scleroderma-like skin changes, as described by others, were not found. In 9 of the 23 cases in which skin changes were found, Raynaud's syndrome was also present.

Pain in the small joints of the fingers and hands (Table 13) was a complaint in 28 (8%) of cases and was found mainly in workers with current exposure, even of short duration (Table 14). There was no special trend regarding pain in other joints, except for the wrists, where most of the cases were found in workers with more than 5 years of exposure. There were no symptoms suggesting involvement of the sacroiliac joints, and no radiologic evaluation was undertaken.

bone changes in the distal phalanges of fingers and toes will be separately reported in detail. While slight changes, such as marginal cortical defects and slight tuft resorbtion were not seldomly seen, transverse defects and fractures were found in only 4 cases.

We were interested in the possibility that hypertension (Table 15) might be of importance. We did not find this to be so. The overall prevalence of hypertension (as defined by WHO criteria) was 12%. There was no correlation with peripheral vascular impairment in the hands and feet. Abnormal urinary findings were not prominent either (Table 16).

The liver was found to be slightly or moderately enlarged in 15% of the cases (1 to 2 finger breadth below the right costal margin; 11 to 13cm vertical span (Table 17); this finding was more frequent in workers with longer duration of exposure. Only in one case was the liver found to be markedly enlarged. The inferior pole of the spleen was palpable in 12 (3.4%) of cases: Again, only one person had marked splenomegaly. There was a highly significant difference in the prevalence of enlarged liver in workers with more than 5 years exposure as compared with those with shorter exposure. The same comparison for splenomegaly did not show a statistically significant difference. According to the past medical history, a diagnosis of cirrhosis of the liver had been mad previously in 5 cases. Esophageal varices had been diagnosed in one patient who had undergone surgery, including portocaval shunt, 12 years before the present examination, with good clinical result.

Abnormal liver function tests (Table 18) were found in small percentages (6% for bilirubin and SGOT, 9% for SGPT), except for alkaline phosphatase which was elevated in 59 (16.6%) of cases).

Duration of exposure correlated with an increase in the prevalence of elevated alkaline phosphatase, especially in workers with more than 5 years of exposure.

There was a higher percentage of abnormal liver tests in workers with current exposure than in those with past exposure (Table 19). Alkaline phosphatase was an exception, since similar proportions of abnormal tests were found in persons with current or prior exposure.

The best correlation between clinical findings of hepatomegaly and/or splenomegaly and abnormal liver function tests was found to be that with elevated alkaline phosphatase (Table 20); 41% of those with such clinical abnormalities had increased alkaline phosphatase.

Hematologic abnormalities were scant in our study. Thrombocytopenia, a frequent finding in the German studies, was present in only one of the examined workers (Table 21).

The possible contributory effect of significant ethanol intake was carefully considered (Table 22): no trend as related to duration of exposure could be found for ethanol intake.

In one-third of the cases with clinical liver and/or spleen changes significant ethanol intake could have been a factor (Table 23), and in 25% of the cases with increased alkaline phosphatase was there a history of significant intake (Table 24). Further, the prevalence of an enlarged

liver and/or spleen in persons with and without significant ethanol intake was compared (Table 25). While there was no statistically significant difference in workers with less than 10 years of exposure, in those with more than 10 years exposure, there was a higher prevalence of enlarged liver and/or spleen in persons with a significant ethanol intake. A multiple factor additive interaction may be considered.

Liver function tests were also analyzed in relation to ethanol intake (Table 26). There was no significantly higher prevalence of elevated alkaline phosphatase, bilirubin or LDH in the group with significant ethanol intake. However, high SGOT or SGPT values were significantly more frequent in this group. It is of interest that elevated alkaline phosphatase, the most frequent biochemical abnormality found, did not correlate with ethanol intake, while there was a significant correlation with duration of vinyl chloride exposure.

Many of the examined workers (Table 27) gave a past medical history of "gastritis," ulcer (gastric and duodenal), upper gastrointestinal bleeding (most often attributed to ulcer) and gallbladder disease. However, it is difficult to know whether such symptoms were more common than might be expected in a similar group without VC exposure.

Out of the 114 workers with such past medical history, there were 28 (25%) who were found to have enlarge liver and/or spleen (Table 28); the percentage was 38% for those with past upper gastrointestinal bleeding and 38.4% for those with a history of gallbladder disease. In the other 242 workers without such a past medical history, 29 (12%) were found to have an enlarged liver and/or spleen.

Chest x-rays of 142 examined workers were available at the time that this report was prepared. In some of the cases linear reticular and less often nodular changes were found in the middle and lower lung fields (Table 29). No relationship was observed with smoking or chronic bronchitis (Tables 30 and 31). The significance of these changes is difficult to evaluate at this point since most of the workers had been exposed to particulate polyvinyl chloride resin as well as to vinyl chloride. Three of those with abnormal chest x-ray findings had had previous dust exposure (in coal mines for periods ranging from 4 to 9 years). There was no relationship between chest x-ray changes and liver changes, but signs and symptoms of peripheral vascular impairment (Raynaud's syndrome, cyanosis, pseudoclubbing, positive Allen test) (Table 32) were found in more than half (53%) of cases with abnormal chest x-ray findings. The prevalence of Raynaud's syndrome was significantly higher in the group with abnormal chest x-rays.

Discussion and Conclusions

Our findings confirm the broad outlines of the clinical syndromes reported by other investigators.

In the examined group relatively high VC exposure had occurred in the past, and symptoms of acute prenarcotic effects were reported by more than half of the examined workers.

Clinical signs and symptoms of abnormal peripheral circulation in the fingers and toes were quite frequent, with cyanosis present in 13 percent, excessive sensitivity to cold in 18 percent and numbness and tingling of the fingers in 24 percent of cases. A typical Raynaud's syndrome was present in 5.7% of cases. An abnormal Allen test, indicating delayed arterial circulation in the areas supplied by the ulnar and/or radial arteries was found in 94 (26.6%) of the examined workers. The prevalence of all these abnormalities increased with length of VC exposure.

Hepatomegaly was a finding in 15% of the examined workers, while splenomegaly was found in 3.4%. Cirrhosis of the liver had been previously diagnosed in 5 workers, and in one esophageal varices with upper gastrointestinal bleeding had led to surgery including portocaval shunt.

Alkaline phosphatase was the test which was most frequently found to be abnormal and showed the best correlation with clinical findings.

Hepatomegaly and elevated alkaline phosphatase were significantly more frequent in workers with longer VC exposure.

Radiologic pulmonary changes, linear reticular and nodular opacities in the lower and middle lung fields were found in a number of cases; the prevalence of these changes was higher with longer duration of exposure, and there was a significant association with peripheral circulation abnormalities. The evaluation of these pulmonary changes is still in progress.

Our results indicate that liver involvement and probably lung involvement in PVC workers are significant and should be given full attention in the medical surveillance of workers with past exposure.

Future exposure to vinyl chloride should be controled in order to prevent development of all adverse effects, including the carcinogenic effect.

Table 1

Acroosteolysis

1957	Filatova and Gronsberg	"Angioneurosis of spastic character"
1963	Suciu et al	- Raynaud's syndrome - (hands and feet) 6% of 168 exposed workers - Scleroderma-like skin changes - (hands, feet, face, neck, thorax) 3.6% of examined
1966	Cordier	- Raynaud's syndrome - Scleroderma-like skin changes - Lytic lesions of terminal phalanges in hands and feet
1967	Harris and Adams	- The same changes and - pseudoclubbing of fingers - Involvement of sacro-iliac joints and patella
1967	Wilson et al	-
1971	Dinman et al	
1972	Markowitz et al	Skin biopsy and pathology data
1972	Jühe et al	Arteriography Occlusion of interosseous arteries
1973	Misgeld et al	Arteriography → spastic arteries visualized only after Priscol

Table 2

Acroosteolysis

Bone Changes

- Marginal and/or cortical defects in distal phalanges
- Loss of cortex in tufts of the distal phalanges
- "Half-moon" defects or cuts
- Transverse defects or fractures
- Complete resorbtion of tufts and part of shafts
- Involvement of the larger bones
  Cystic lesions and increased radiolucency
  (ulna, radius, humerus, os calcis, patella)
- Erosive and sclerotic changes in sarco-like joints

Table 3

Acroosteolysis

Skin biopsy pathology

- Thickening of dermis
- Nonfibrillary homogenization of collagen and thickened collagen bundles
- Disorganization of elastic fibers, which are split and broken
- Inflammatory infiltrates, predominantly perivascular (lymphocytes, few histiocytes)
- Thickening of the media of dermal arterioles
- Uneven endothelial thickening
- Marked swelling of dermal nerve fibers vacualization of axones
- Intersitital edema
- Dilatation of lymphatics

Table 4

Evidence of liver damage in PVC exposed workers

1949	"Hepatitis-like liver changes"	Tribuch et al. (Russia)
1963	"Chronic epithelial hepatitis" in 15% of cases; hepatomegaly, increased bilirubin and prothrombin time, abnormal Takata-Ara test.	Pushin (Russia)
1963	Hepatomegaly in 30% of 168 examined workers, splenomegaly (6%). Liver biopsy in 2 cases: "chronic hepatitis."	Suciu et al (Romania)
1967	Hepatomegaly; persistent raised bilirubin.	Harris (England)
1967	Increased BSP retention, raised icterus index——related to degree of exposure.	
1972	Pain in RUQ and abnormal liver tests (in 2 out of 7 patients with scleroderma-like skin changes).	Jühe et al (Germany)
1973	RUQ discomfort; abnormal liver changes.	Misgeld et al (Germany)

Table 5

Duration of vinyl chloride exposure among 354 workers in Niagara Falls

Duration of exposure (years)	Currently employed	Formerly employed	Total
≤ 2	36	25	61
2.1–5	62	13	74
5.1–10	47	15	62
10.1–20	77	27	104
20+	45	7	52
	267	87	354

Table 6

Age distribution of 354 vinyl chloride exposed workers (Niagara Falls)

Duration of exposure (years)	Age groups (years)					
	21-30	31-40	41-50	51-60	60+	Total
≤ 2	39	9	8	4	1	61
2.1-5	45	17	9	1	3	75
5.1-10	17	18	24	3	0	62
10.1-20	0	50	30	21	3	104
20+	0	4	14	27	7	52
Total	101	98	85	56	14	354

Table 7

Symptoms of acute overexposure among 354 vinyl chloride workers

Duration of Exposure (years)	Number of examined workers	Symptoms				Total	%
		Headache	Light-headedness	Dizziness	Loss of Consciousness		
≤ 2	61	12	21	9	3	28	46
2.1-5	75	13	30	30	3	43	57
5.1-10	62	8	25	20	0	33	53
10.1-20	104	17	40	44	7	56	53
20+	52	7	26	32	1	36	69
Total	354	57 (16%)	142 (40%)	135 (38%)	14 (4%)	196	55%

Table 8

Clinical signs and symptoms of abnormal peripheral circulation among 354 vinyl chloride exposed workers

Duration of Exposure (years)	Total Number	Numbness Tingling		Excessive Sensitivity to cold		Pain		Cyanosis		Involvement of toes		Rayna synd
		No.	%	No.	%	No.	%	No.	%	No.	%	No.
≤ 2	61	2	(3%)	2	(3%)			2	(3%)			
2.1-5	75	16	(21%)	11	(15%)	4	(5%)	4	(5%)	1	(1%)	2(2
5.1-10	62	11	(18%)	11	(18%)	3	(5%)	10	(16%)	5	(8%)	4(6
10.1-20	104	36	(35%)	26	(25%)	11	(11%)	19	(18%)	11	(11%)	9(8
20.1	52	20	(38%)	13	(19%)	10	(19%)	11	(21%)	8	(15%)	5(9
Total	354	85	(24%)	63	(18%)	28	(8%)	46	(13%)	25	(7%)	20(5

*Prevalence in workers with more than 10 years exposure significantly higher than in those with less than 10 years exposure.

$\chi^2 = 5.783$

$p < 0.02$

Table 9

Raynaud's Syndrome among 354 vinyl chloride workers

Exposure	Number	Raynaud's Number	%
Active	267	17	6.4
Prior	87	3	3.4
	354	20	5.6

Table 10

Abnormal Allen Test among
354 vinyl chloride workers

Duration of Exposure (years)	Abnormal Test Results: Current Exposure	Prior Exposure	Total*
≤ 2	7/36	2/25	9/61 (14.7%)
2.1-5	11/62	7/13	18/75 (24.0%)
5.1-10	16/47	7/15	23/62 (37.1%)
10.1-20	22/77	5/27	27/104 (26.0%)
20+	12/45	5/7	17/52 (32.7%)
	68/267 (25.5%)	26/87 (29.9%)	94/354 (26.6%)

Radial - 10
Ulnar - 38
Both - 46

†Prevalence of abnormal Allen Test in workers with more than 5 years exposure significantly higher than in those with less than 5 years exposure.

$\chi^2 = 5.084$
$.002 < p < 0.05$

Table 11

Psuedo-clubbing of fingers among 354 vinyl chloride workers

Duration of exposure (years)	Pseudo-clubbing of fingers: Current exposure	Past exposure	Total	
≤ 2	2/36	2/25	4/61	(6.5%)
2.1-5	3/62	0/13	3/75	(4.0%)
5.1-10	3/47	3/15	6/62	(9.7%)
10.1-20	7/77	2/27	9/104	(8.6%)
20+	7/45	2/7	9/52	(17.3%)
			31/354	(8.75%)

In 25 cases - signs/symptoms of peripheral vascular disease.

In 8 cases - typical Raynaud's syndrome.

Table 12

Skin changes in 354
vinyl chloride exposed workers

Duration of exposure (years)	Total number examined	Skin changes	
		Number	%
≤ 2	61	4	6.5
2.1-5	75	2	2.6
5.1-10	62	3	4.8
10.1-20	104	8	7.7
20+	52	6	11.5
Total	354	23	6.4

Raynaud's syndrome was present in 9 cases.

Tabl 13

Pain in joints of fingers and hands

Duration of exposure (years)	Total number examined	Fingers, Hands		Wrists	
		No.	%	No.	%
≤ 2	61	5	8.1	1	1.6
2.1-5	75	5	6.7		
5.1-10	62	2	3.2	1	1.6
10.1-20	104	8	7.7	3	2.9
20+	52	8	15.4	1	2.0
Total	354	28	8.0	6	1.7

Note: Difference in prevalence not significant for workers with less than 10 years or more than 10 years of exposure.

Table 14

Pain in the joints of fingers and hands

	Total number examined	Pain in finger and hand joints	
		No.	%
Current exposure	267	24	9.0
Past exposure	87	4	4.6
Total	354	28	8.0

Table 15

Hypertension among 354 vinyl chloride workers

Duration of exposure (years)	Exposure	Hypertension: Systolic	Diastolic	Systolic and Diastolic	Total
≤ 2	36 Current	0	0	2	2
	25 Prior	0	1	2	3
2.1-5	62 Current	1	0	2	3
	13 Prior	1	0	0	1
5.1-10	47 Current	1	0	2	3
	15 Prior	0	1	1	2
10.1-20	77 Current	3	2	4	9
	27 Prior	3	1	2	6
20+	45 Current	3	5	4	12
	7 Prior	0	0	2	2
Total	354	12	10	21	43 (12%)

Note: Workers with hypertension had no greater prevalence of peripheral vascular changes in hands and feet.

Elevated BUN present in only three instances.

Table 16

Abnormal urine findings among 354 vinyl chloride workers

Duration of exposure (years)	Current exposure	Protein 1+	RBC	Casts
≤ 2	36 Current	1	0	2
	25 Prior	2	0	0
2.1-5	62 Current	2	0	2
	13 Prior	1	0	0
5.1-10	47 Current	1	1	1
	15 Prior	0	1	0
10.1-20	77 Current	1	6	0
	27 Prior	1	1	1
20+	45 Current	1	3	0
	7 Prior	0	0	0
	354	10	12	6

Table 17

Hepatosplenomegaly among
354 vinyl chloride workers

Duration of exposure (years)	Total number examined	Enlarged liver*		Enlarged spleen**	
		No.	%	No.	%
≤ 2	61	4	6.5	1	1.6
2.1-5	75	5	6.7	2	2.7
5.1-10	62	7	11.3	2	3.2
10.1-20	104	20	19.0	3	2.9
20+	52	17	32.7	4	7.7
Total	354	53	15.0	12	3.4

*Difference in prevalence of enlarged liver in workers exposed for less than 5 years as compared to those exposed for more than 5 years. $\chi^2 = 12.107$, $p < 0.001$

**For enlarged spleen $\chi^2 = 0.945$, n.s.

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Table 18

Abnormal LFT among 354 vinyl chloride workers

Duration of exposure (years)	Total number examined	Bilirubin >1.1	SGOT >50	SGPT >36	LDH >225	Alk. Phos.* >86	
≤ 2	61	2	3	5	0	9	12.6%
2.1-5	75	5	4	7	0	7	9.3%
5.1-10	62	7	3	5	4	11	17.7%
10.1-20	104	2	6	8	3	19	18.2%
20+	52	5	4	6	1	13	25.0%
Total	354	21(6%)	20(6%)	31(9%)	8(2.3%)	59	(16.6%)

*Prevalence of elevated alkaline phosphatase significantly higher in workers with more than 5 years exposure than in those with less than 5 years exposure.

$\chi^2 = 3.84$ p=0.05

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Table 19

Abnormal LFT among 354 vinyl chloride workers currently or previously employed

Liver function test		Current employment (267)	Prior employment (87)	Hepatosplenomegaly, Hepatic tenderness (66)
Bilirubin	>1.1	18 (6.7%)	3 (3.6%)	---
SGOT	>50	17 (6.4%)	3 (3.6%)	6/20 (30.0%)
SGPT	>36	26 (9.7%)	5 (5.7%)	8/31 (26.9%)
LDH	>225	6 (2.4%)	2 (2.3%)	---
Alk. Phos.	>86	43 (16.0%)	16 (18.0%)	27/59 (45.8%)

Table 20

Relation of hepato and/or splenomegaly to elevated alkaline phosphatase among 354 vinyl chloride workers

Duration of exposure (years)	No.	Enlarged liver and/or spleen, or hepatic tenderness	Elevated alkaline phosphatase	
≤ 2	61	7	3	43%
2.1-5	75	7	1	14%
5.1-10	62	10	4	40%
10.1-20	104	20	10	45%
20+	52	20	9	45%
	354	64	27	41%

Table 21

Hematologic changes in 354 vinyl chloride workers

	Total number examined	Thrombocytopenia <150,000	Leucopenia <4,000	Leucopenia 4,000-5,000	Hemoglobin <14 gr.
≥ 2	61		1	8	1
2.1-5	75		3	6	1
5.1-10	62		1	2	
10.1-20	104			6	4+
20+	52	1		6	2++
Total	354	1 (0.3%)	5 (1.4%)	28 (7.6%)	8 (2.2%)

+persons with past history of G.I. bleeding.

Table 22

Alcohol intake
in 354 vinyl chloride exposed workers

Duration of exposure (years)	Total number examined	Alcohol Intake ++ (1-2 quarts/week)	+++ (2-3 quarts/week)	++++ Known alcoholic	Total No.	Total %
2 ≥	61	10	5	0	15	24.6
2.1-5	75	11	4	1	16	21.2
5.1-10	62	13	0	0	13	21
10.1-20	104	9	4	0	13	12.5
20.1->	52	10	1	1	12	23
Total	354	53 15%	14 3.9%	2 0.6%	69	19.5%

with thiouracil and died three months after

Table 23

Significant alcohol intake and liver changes (clinical)

Duration of exposure (years)	Total number examined	Enlarged liver and/or spleen or hepatic tenderness		Cases with significant alcohol intake	
		No.	%	No.	%
≤ 2	61	7	11.5%	2	3.3%
2.1-5	75	7	9.4%	3	4.0%
5.1-10	62	10	16.0%	4	6.5%
10.1-20	104	21	20.0%	4	3.8%
20+	52	20	38.0%	8	15.0%
	354	65	18.2%	21	6.2%

Table 24

Elevated alkaline phosphatase and significant alcohol intake

Duration of exposure (years)	Total examined	Alkaline phosphatase		Significant alcohol intake	
		No.	%	No.	%
≤2	81	9	12.6	1	1.6
2.1-5	75	7	9.3	1	1.3
5.1-10	62	11	17.7	6	9.7
10.1-20	104	19	18.2	2	1.9
20+	52	13	25	4	7.7
Total	354	59	16	14	3.9

Table 25

Hepato- and/or splenomegaly
and significant alcohol intake

	Total number examined	Significant alcohol intake			No significant alcohol intake		
		Total number	Enlarged liver and/or spleen		Total number	Enlarged liver and/or spleen	
			No.	%		No.	%
≤ 2	61	15	2	13.4%	46	7	15.0%
2.1-5	75	16	2	12.5%	59	4	6.8%
5.1-10	62	13	4	30.5%	49	5	10.2%
10.1-20	104	13	4	30.5%	91	17	18.7%
20+	52	12	8	75.0%	40	12	30.0%
Total	354	69	20	29.0%	285	45	16.0%

Difference in prevalence of enlarged liver and/or spleen between the group with alcohol intake and those without alcohol intake non-significant for workers with less than 10 years of exposure. $\chi^2 = 1.951$ n.s.

Significant for workers with more than 10 years exposure.

$\chi^2 = 7.243$
$p < 0.01$

Table 26

Alcohol intake and abnormal liver function tests

	Total number examined	Alkaline phosphatase		Bilirubin		SGOT		SGPT		LDH	
		No.	%	No.	%	No.	%	No.	%	No.	%
Significant alcohol intake	69	14	20.3%	1	1.4%	9	13.0%	11	16.0%	2	2.9%
Non-significant alcohol intake	285	45	15.8%	20	7.0%	11	3.9%	20	7.6%	6	2.1%
Total	354	59	16.0%	21	6.0%	20	6.0%	31	9.0%	8	2.3%
		$\chi^2 = 0.810$ n.s.		$\chi^2 = 3.087$ n.s.		$\chi^2 = 8.790$ $p < 0.01$		$\chi^2 = 5.537$ $p < 0.01$		n.s.	

Table 27

Gastrointestinal disease (by history) in 354 vinyl chloride exposed workers

	No.		%	
"Gastritis"	47		13	
Ulcer (gastric or duodenal)	31	52	9	15
Ulcer and upper G.I. bleeding	21		6	
Gall bladder disease	13		3.6	
Total	112			

Table 28

Gastrointestinal disease (by history) and enlarged liver and/or spleen (clinical examination)

	Total number	Enlarged liver	
		No.	%
"Gastritis"	47	8	17.0%
Ulcer (gastric or duodenal)	31	7	22.6%
Ulcer and upper G.I. bleeding	21	8	38.0%
Gall bladder disease	13	5	38.4%

Table 29

Chest x-ray abnormalities* among 142 vinyl chloride workers

Duration of Exposure (years)	Current Exposure	X-Ray Findings: Normal	Abnormal	
≤ 2	13 Current	12	1	5.2%
	6 Prior	6	0	
2.1-5	32 Current	25	7	23.0%
	3 Prior	2	1	
5.1-10	19 Current	18	1	11.0%
	8 Prior	6	2	
10.0-20	32 Current	17	15	48.6%
	5 Prior	2	3	
20+	23 Current	15	8	33.3%
	1 Prior	1	0	
Total	142	104	38	27.0%

*Data incomplete.

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Table 30

Chest x-ray and smoking

Chest x-ray	Total number	Smokers		Ex smokers		Non-smokers	
		No.	%	No.	%	No.	%
Normal	104	53	51	23	22	28	27
Abnormal	38	25	66	6	16	7	18

Prevalence of abnormal chest x-ray in persons with history of smoking not significantly different from that in non-smokers. $\chi^2 = 1.083$ n.s.

Table 31

Chest x-ray and chronic bronchitis

Chest x-ray	Total number	History of chronic bronchitis		No history of chronic bronchitis	
		No.	%	No.	%
Normal	104	20	21	84	79
Abnormal	38	8	19	30	81

Table 32

Abnormal chest x-ray and abnormal peripheral circulation

Chest X-Ray		Raynaud's syndrome		Cyanosis		Pseudo-clubbing		Abnormal Allen test	
		No.	%	No.	%	No.	%	No.	%
Normal	104	5	4.8	9	8.6	8	7.7	20	19
Abnormal	38	7	18.4	6	16.0	7	18.4	13	34
		$\chi^2 = 6.667$ $p < 0.01$						$\chi^2 = 3.501$ n.s.	