RICE UNIVERSITY
GEORGE R. BROWN SCHOOL OF ENGINEERING
HOUSTON, TEXAS 77001

March 26, 1976

DEPARTMENT OF CHEMICAL ENGINEERING

Mr. W. P. Anderson
Tenneco Chemicals
Park 80 Plaza West-One
Saddle Brook, N. J. 07662

Dear Mr. Anderson:

At your request I have reviewed the document "Quantitative Risk Assessment For Community Exposure To Vinyl Chloride", by Robt. E. McGaughy of the U. S. Environmental Protection Agency. I have also considered the atmospheric dispersion modeling and air sampling conducted by your staff to assess the vinyl chloride exposure at your Deer Park site. The following comments pertain to the general risk calculations of McGaughy, my review of your modeling results, and my estimate of the particular risk associated with the Deer Park facility.

The essential elements of an exposure evaluation for a given source are the following:

(1) Estimating the strength and geometry of the pollutant emission.

(2) Modeling the atmospheric dispersion, which combines the source data and meteorological conditions of the site to predict average pollutant concentration as a function of location relative to the center of the source.

(3) Combining this concentration function with population distribution data to obtain human exposure.

The estimate of risk to human health follows from (3) and clinical/epidemiological health observations. In the case of vinyl

chloride exposure at the concentration levels predicted to exist near PVC plants, the health data must be extrapolated downward more than three orders of magnitude (50 ppm to 17 ppb). As McGaughy points out, the difference between the more conservative linear model and the more often accepted log-probit model for extrapolation is about a factor of 100 at the 17 ppb level. His figure B-1 shows the linear model to be more conservative (predicts a higher risk) by a factor of about 10,000 at the 1 ppb level.

These elements are next considered as they apply to the specific problem at hand - the risk to human health of an expanded Tenneco facility at Deer Park. Using data acquired by your staff, I have made approximate corrections to apply McGaughy's methodology to this site.

The important points are:

(1) Your estimate of vinyl chloride monomer emission rate (stack emissions plus fugitive losses) is in the range of 0.1 percent of throughput, contrasted with 4 percent assumed by McGaughy for the typical PVC plant. Accounting for your larger production rate, this still represents a lower emission rate by a factor of about 30.

(2) Your dispersion modeling is directly comparable to McGaughy's since the same mathematical models were used. It is probable, in fact, that the same basic computer program package was used in both studies.

(3) The dispersion model used to prediction concentrations from source rate data is intrinsically linear in source strength. Hence, direct comparison can be made between the concentration plots in McGaughy (Appendix A) and the plots generated by your study. Considering the differences in meteorological conditions between McGaughy's average PVC plant case and your site, I find the two results to be quite equivalent. That is, your concentrations at the same distances from the plant center are approximately 30 times lower than McGaughy's, as expected.

(4) Your inability to detect vinyl chloride at locations more than 1/2 mile down-wind of the present plant site are also consistent with the predictions of the dispersion model, considering the lower limit of your analytical procedures (about 10 ppb).

(5) There are factors ignored in the dispersion modeling which would further decrease the predicted concentrations if accounted for. These include homogeneous decay reactions and reactions with surfaces involving vinyl chloride. The latter may be quite important inside dwellings, etc.

(6) Using the population numbers given by McGaughy in his Table A-1-(a) for the large PVC plant in Deer Park and the decreased concentrations predicted by the dispersion model, I calculate an average exposure of about 0.45 ppb to 131,000 people. The overall effect rate can then be calculated using McGaughy's linear model extrapolation constant of 0.071 cases of angiosarcoma per ppb per million population per year. The result for the Deer Park site is .131 x .45 x 0.071 = .004 cases per year, or one case in 250 years.

(7) Considering the difference between the linear model and the log-probit model discusses above, this estimate may be as much as a factor of 10,000 too high.

Considering the uncertainties in the basic health risk data, I do not consider this result to be very reliable. If it is in fact an upper limit, the risk in my opinion is negligible.

Sincerely,

H. A. Deans

H. A. Deans
Professor of
Chemical Engineering

HAD:ld

by A. Kuzmack and R. McGaughy

The authors have considered a number of very important problems concerned with the assessment of the risk of cancer, especially liver angiosarcoma, and have taken as quantitative an approach as possible in the analysis of the available data. It can be fairly stated that the authors have taken a reasonable approach, their conclusions follow from the assumptions made and, insofar as possible, they have used actual data that bear on the calculation of risk.

In any problem of this type, there are bound to be numerous unknowns, data from small samples that are variable in outcome and problems in explaining interspecies differences. Average or median values have been used and weighting factors applied (that themselves are subject to variability) to obtain answers. One cannot fault the approach since it was reasonable and as quantitative as possible. However, in choosing figures from which to estimate risks, the authors have nearly always taken the figure that would lead to a higher estimate of risk of liver angiosarcoma (or cancer). They have evaluated the various possible errors in their estimates as well as possible, however their figures do tend to lead to upper limits of the number of cases to be expected. One example of this is the choice of a linear dose-response curve rather than a curve linear in log dose for calculating the proportion of animals that will develop liver angiosarcoma following exposure to different VC concentrations. Assuming the log dose relationship yields estimates of the number of cases of liver angiosarcoma .01 - .1 times that obtained from the linear dose-response curve.

This study by the authors points up the need further studies to determine the risk of exposure to VC more precisely. For example, it is certainly important to carry out surveys of liver cancer and other liver related diseases in individuals living near VC or PVC plants to compare their incidence with that in some control areas not close to VC or PVC plants. Secondly, some animal studies should be carried out to estimate the dose-response relationship to VC concentration. For example, the lowest non-zero dose of VC in Maltoni's study was 50 ppm and this dose led to a 2% incidence of liver angiosarcomas. This dose is nearly 3000 times larger than the 17 ppb average concentration exposure for people living within 5 miles of PVC or VC plants. One can only speculate on what the incidence of liver angiosarcoma (and other tumors) is for VC concentrations in the neighborhood of 10 - 100 ppb.

The key results in the authors' study are that: an estimated 4.6 million people are living within 5 miles of PVC or VC plants; the average exposure to VC for these individuals is 17 ppb and the estimated number of cancers to be observed in these individuals is 5.5 cases of liver angiosarcoma per year and 11 cases of cancer. Assuming a log probit model reduces the estimated numbers of cases by some factor between 10 and 100. It is evident that if one accepts the log probit model, VC exposure does not present a major risk of cancer, whereas the linear dose response curve indicates a major risk. There is no available data for choosing between these models, so the authors give the results for both models and take the prudent approach in assuming that there is no threshold dose of VC, below which no cases of liver angiosarcoma would be expected to occur.

of VC or PVC plants, the authors essentially used the results of a study by the APHA of census tracts based upon 1970 census population figures. There are known flaws with the estimate (since no adjustment is made for mobility or increase in the population since 1970, or adjustment for age, sex, or direction from plant), however the estimate is unlikely to be in serious error.

Concerning the estimate of the exposure of 17 ppb for the 4.6 million people the estimated ambient concentrations were based upon studies by EPA and Teknekron These studies were reasonably consistent with one another and so this result cannot be criticized severely. Further, adjustments were made for the number of plants, the size of the plants, and meteorological conditions. The weighting factors used for meteorological conditions were 0.56 for low, 1.00 for average, 1.55 for high, and 2.55 for very high. The basis for choosing these figures is not given in this report; reference is made to the Teknekron results.

It is of interest that when the figures for weighted population exposed are given by distance from plant (Table A-2), the major populations exposed are those 1-5 miles from PVC plants. If one were to do a study of incidence of liver angiosarcoma and other tumors in the large cities (about 100,000 or more), then a relatively small number of cities have large populations near PVC plants. From Table A-1 these are: Carson City, Louisville, Burlington, Passaic, S. Kearny, Hicksville, Fitchburg,Springfield, Fedrickton, Williamsville, Oklahoma City, Deer Park, and South Charleston. Incidence of disease studies might be undertaken in those areas.

The major possibility for error in this report (and this is recognized by the authors) is when one attempts to predict the number of cancer (and liver angiosarcomas) based on given levels of exposure to VC. The authors use animal data to predict the results in humans, and then use data from humans insofar as possible to confirm the animal data. Results from the animal and human data agree reasonably well so this gives more confidence in the methodology utilized.

When considering the data from humans, the authors refer to reports by Tabershaw-Gaffey, Nicholson, Heath and Falk, and Wagoner. For each of these studies, data are given concerning the number of individuals studied, the number of cases of liver angiosarcoma, and the approximate length of exposure. However, in calculating the incidence rate, defined as a number of cases of liver angiosarcoma per person per year of exposure, the estimate of .00475 per person year was made only from the Tabershaw-Gaffey report (Page D-4). It is suggested that the data from the other studies might also be used to obtain this type of estimate since it appears that the various studies would lead to estimates that were not very different from one another. One could make reasonable assumptions regarding the average exposure for the workers in each reference. The authors indicate a preference for calculating incidence rates using the distribution of exposure duration and this is available only from the Tabershaw-Gaffey study; this preference may be justified, however when one has a series of several studies that would appear to lead to relatively consistent estimates of incidence rates, it suggests that these studies be used.

On page 7, the authors indicate that when all the uncertainties are considered, their judgment is that the number of cases of liver angiosarcoma produced per year of exposure in people residing near VC plants is somewhere between < 1 and 10 cases, with these cases not to be diagnosed until 15 - 20 years from now. The estimated number of cases of primary cancer at other sites is also between < 1 and 10 cases. It is not clear why the upper limit is not 20 cases in the latter case, since at other places in the manuscript the authors refer to an expectation of twice as many cases of cancer at other sites as cases of liver angiosarcoma. Also, an estimated 1 - 300 cases of serious liver damge would be predicted (though the number could possibly be less). Based on careful review of the methodology used in the report, there are no good reasons to seriously question these figures.

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TABLE I

SUMMARY OF RESULTS FROM INITIAL SCREENING[4]

Case	Precondensation of Stripper Gas	VCM Recovery Method	Emission Control	Net Revenue $/year	Total Capital, $	Total Net[3] Present Value, M$	Economic Rank
1	No	Carbon Bed	Incinerator	- 19,250	3,605,000	- 2,387	13
2	No	Carbon Bed	Boiler	+ 8,260	3,270,000	- 2,073	10
3	No	Carbon Bed	None	+ 9,290	3,215,000	- 2,034	9
4	No	Carbon Bed[1]	Incinerator	- 35,600	3,965,000	- 2,676	14
5	Yes	Carbon Bed	Incinerator	+ 21,180	3,500,000	- 2,226	11
6	Yes	Carbon Bed	Boiler	+ 48,710	3,220,000	- 1,896	7
7	Yes	Carbon Bed	None	+ 49,730	3,160,000	- 1,854	6
8	Yes	Carbon Bed[1]	Incinerator	+ 22,608	3,620,000	- 2,247	12
9	No	None	Incinerator	- 122,830	2,125,000	- 1,805	5
10	No	None	Inc./Acid Rec.[2]	- 29,790	2,960,000	- 2,009	8
12	Yes	None	Incinerator	- 51,660	1,625,000	- 1,229	3
13	Yes	None	Inc./Acid Recov.[2]	+ 22,040	2,115,000	- 1,281	4
15	Yes	Solvent Absorption	Boiler	+ 145,120	1,010,000	- 131	1
16	Yes	Solvent Absorption	Incinerator	+ 119,530	1,400,000	- 473	2

1. In cases 4 and 8 the carbon bed recovers VCM only from the vent condenser stream; gas from the dispersion strippers goes directly to the incinerator.

2. In cases 10 and 13 the HCl from the combustion gases is recovered as 20% hydrochloric acid and a credit is taken for it.

3. Calculated using a 25% discount rate, 10-year life and no salvage value.

4. Cases 11 and 14, omitted here, involved recovery of 36% hydrochloric acid from incinerator combustion gases. The cost estimates from Trane Thermal made it obvious that the cases were not worth comple:

TABLE II

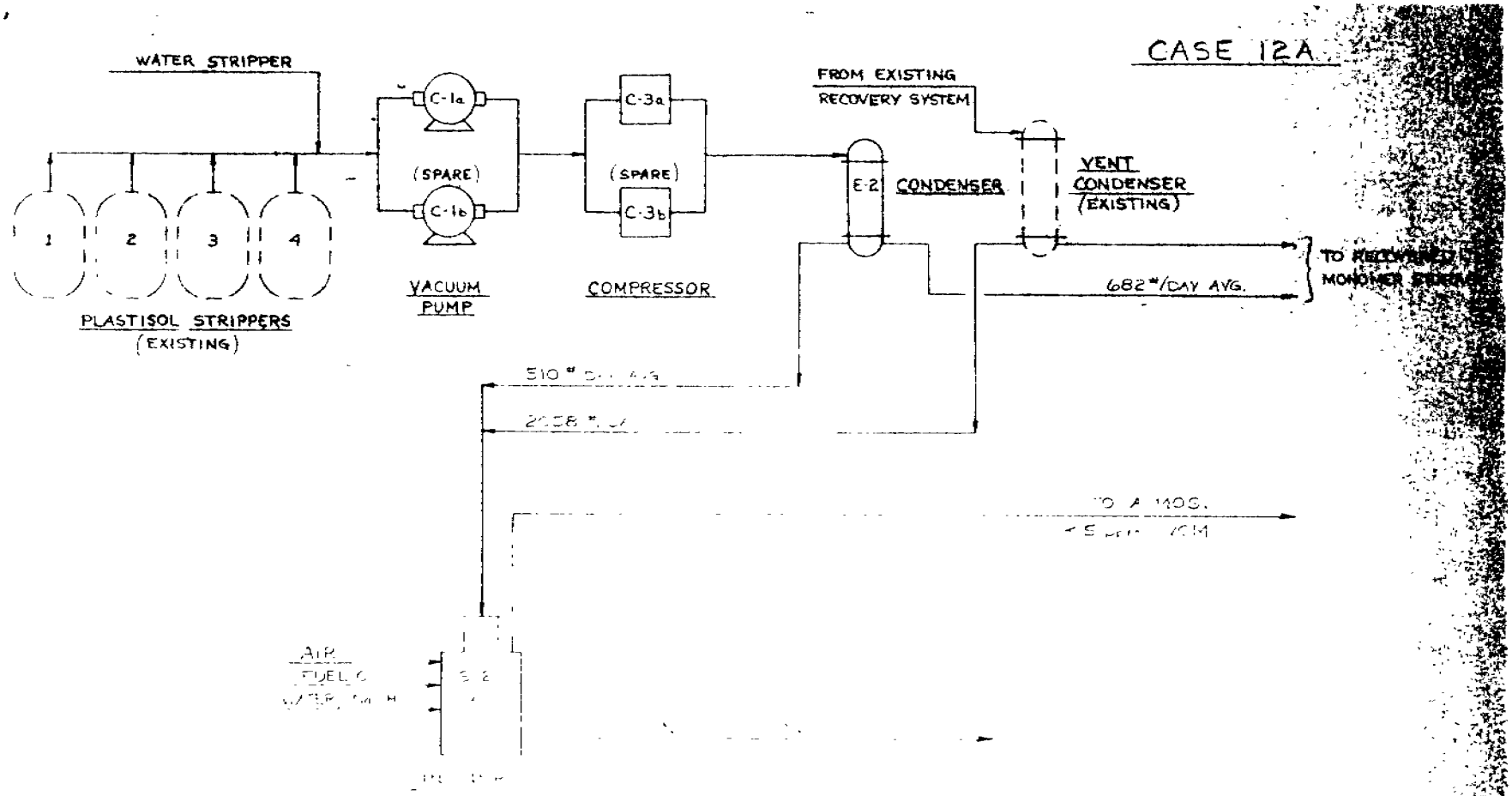
COMPARISON OF ALTERNATIVE APPROACHES

Case	Operating Cost $/Yr.	Operating Revenue (Rec'd VCM) $/Yr.	Net Operating Income $/Yr.	CAPITAL: Process Equip. $	CAPITAL: Methyl Chloride System $	CAPITAL: Utilities $	CAPITAL: Total $	UTILITIES - DEMAND: KVA (Approx)	Steam #/Hr.	Cooling Water GMP	Brine Tons	Chilled Water Tons	Total Net Present Value	Economic Rank
"Pessimistic" Carbon Bed Alone														
7A	378,920	170,970	-207,950	2,321,000	100,000	545,000	2,966,000	435	4,200	920	7	76	-2,649 M	5
Incineration Alone														
12A	198,020	35,880	-162,140	1,475,000	-	150,000	1,625,000	61	-	-	7	18	-1,624 M	3
Solvent Abs./Incineration														
16A	195,790	169,600	- 26,190	1,800,000	-	150,000	1,950,000	50	600	50	10	18	-1,347 M	1
"Optimistic" Carbon Bed/Incineration														
17	295,010	170,970	-124,040	2,868,000	-	295,000	3,163,000	196	1,050	240	7	52	-2,477 M	4
"Optimistic" Carbon Bed Alone														
18	212,260	170,970	- 41,290	1,848,000	-	295,000	2,143,000	190	1,050	240	7	52	-1,525 M	2

TABLE III

DETAILED OPERATING COSTS FOR ALTERNATE APPROACHES

Case	Steam Use #/Day	Steam Cost $/Yr.	Power Use KW Hr./Day	Power Cost $/Yr.	Fuel Oil Use GPD	Fuel Oil Cost $/Yr.	Caustic $/Yr.	Chilled Water $/Yr.	Brine $/Yr.	Cooling Water $/Yr.	Nitrogen $/Yr.	Carbon/ Solvent Make-Up $/Yr.	Maintenance Taxes, Ins. $/Yr.	Total Operating Cost $/Yr.
7A	79,470	95,960	2,380	22,990	0	0	0	12,690	5,880	42,840	2,280	5,500	190,780	378,920
12A	0	0	1,091	10,540	55	7,700	59,200	9,070	5,880	-	-	-	105,630	198,020
16A	12,010	14,500	1,170	11,300	110	15,400	370	9,070	8,400	8,750	-	4,500	123,500	195,790
17	13,900	16,790	2,201	21,260	110	15,400	10	11,220	5,880	11,110	5,610	3,300	204,430	295,010
18	13,900	16,790	2,093	20,220	0	0	0	11,220	5,880	11,110	5,610	3,300	138,130	212,260



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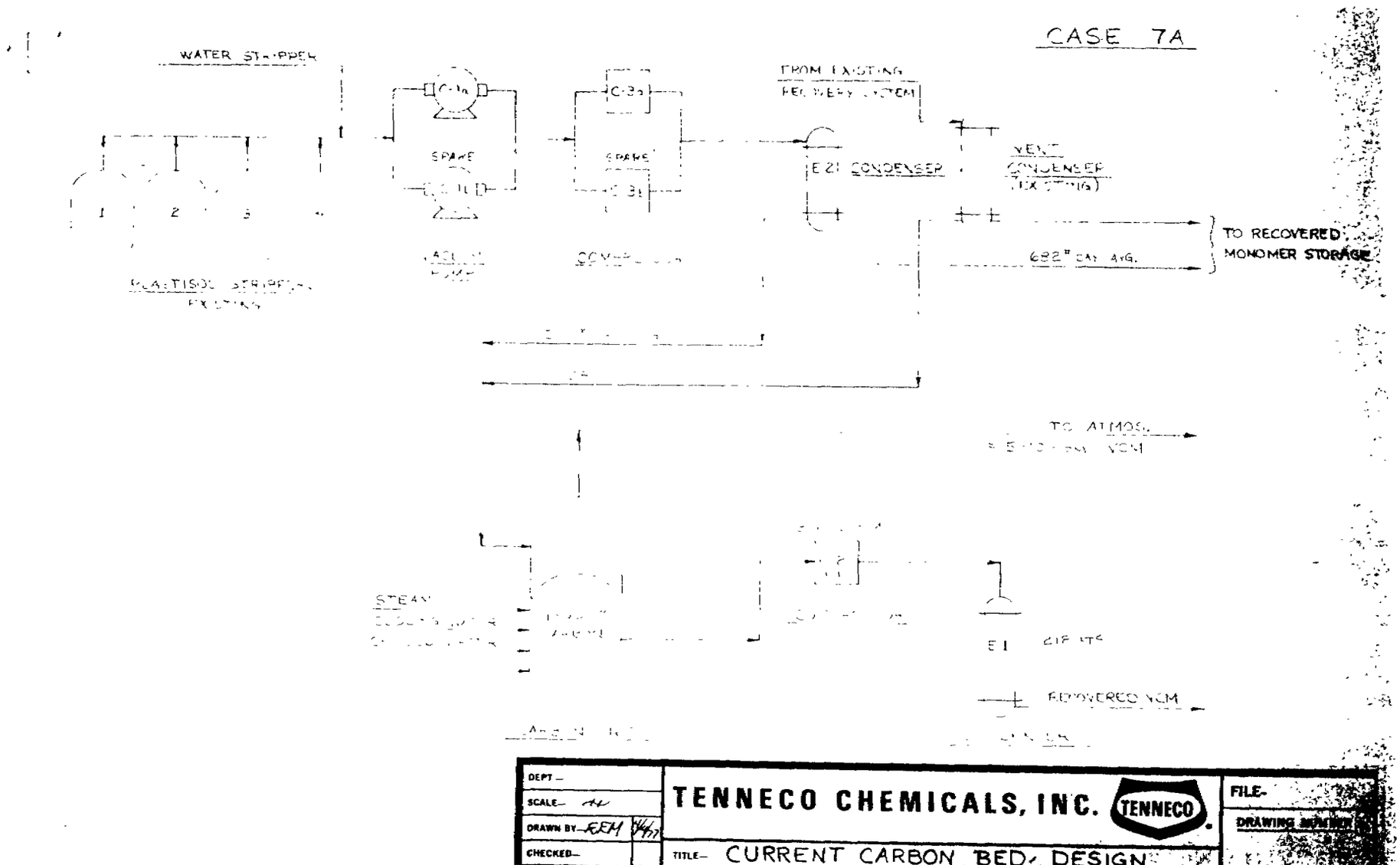

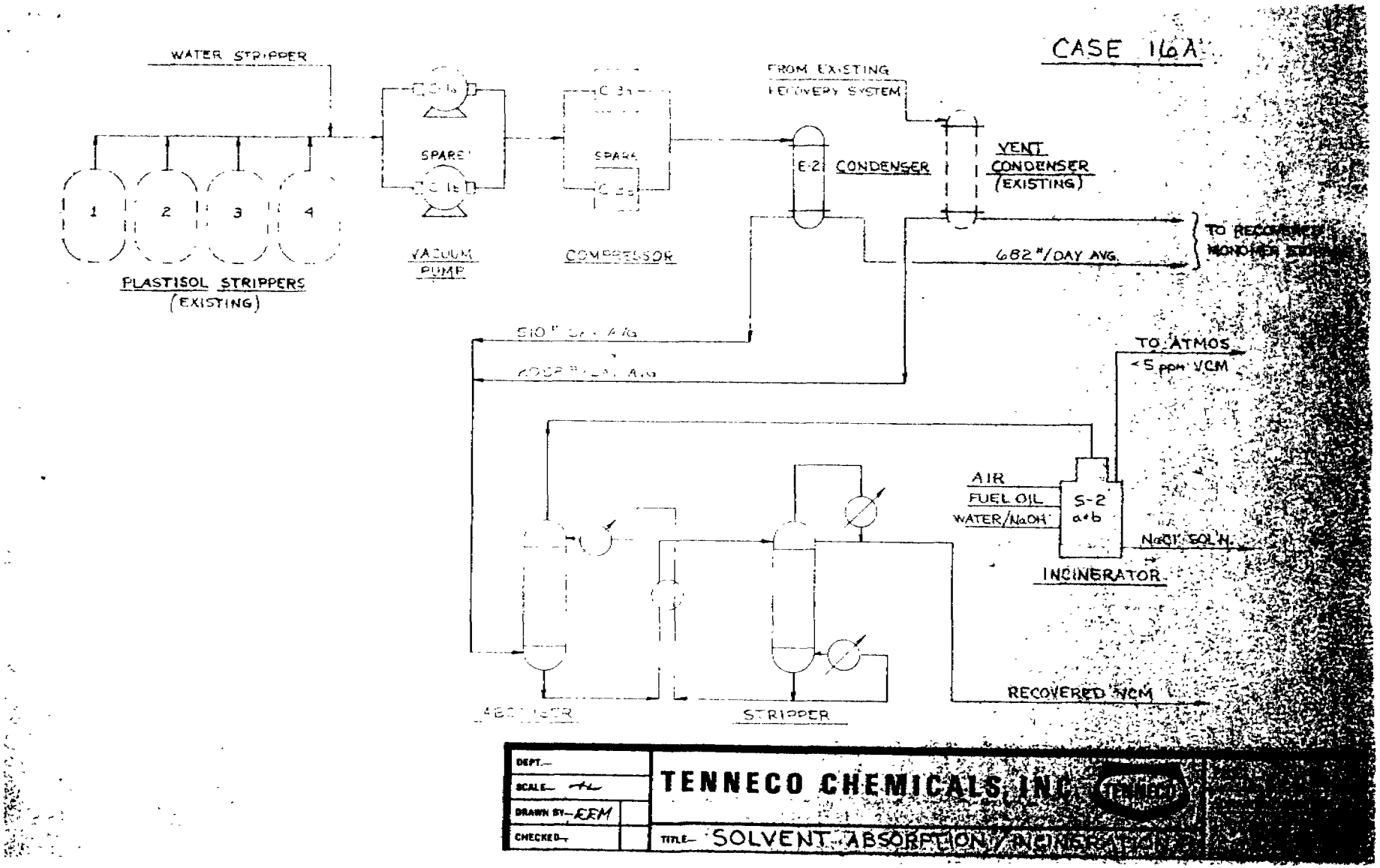

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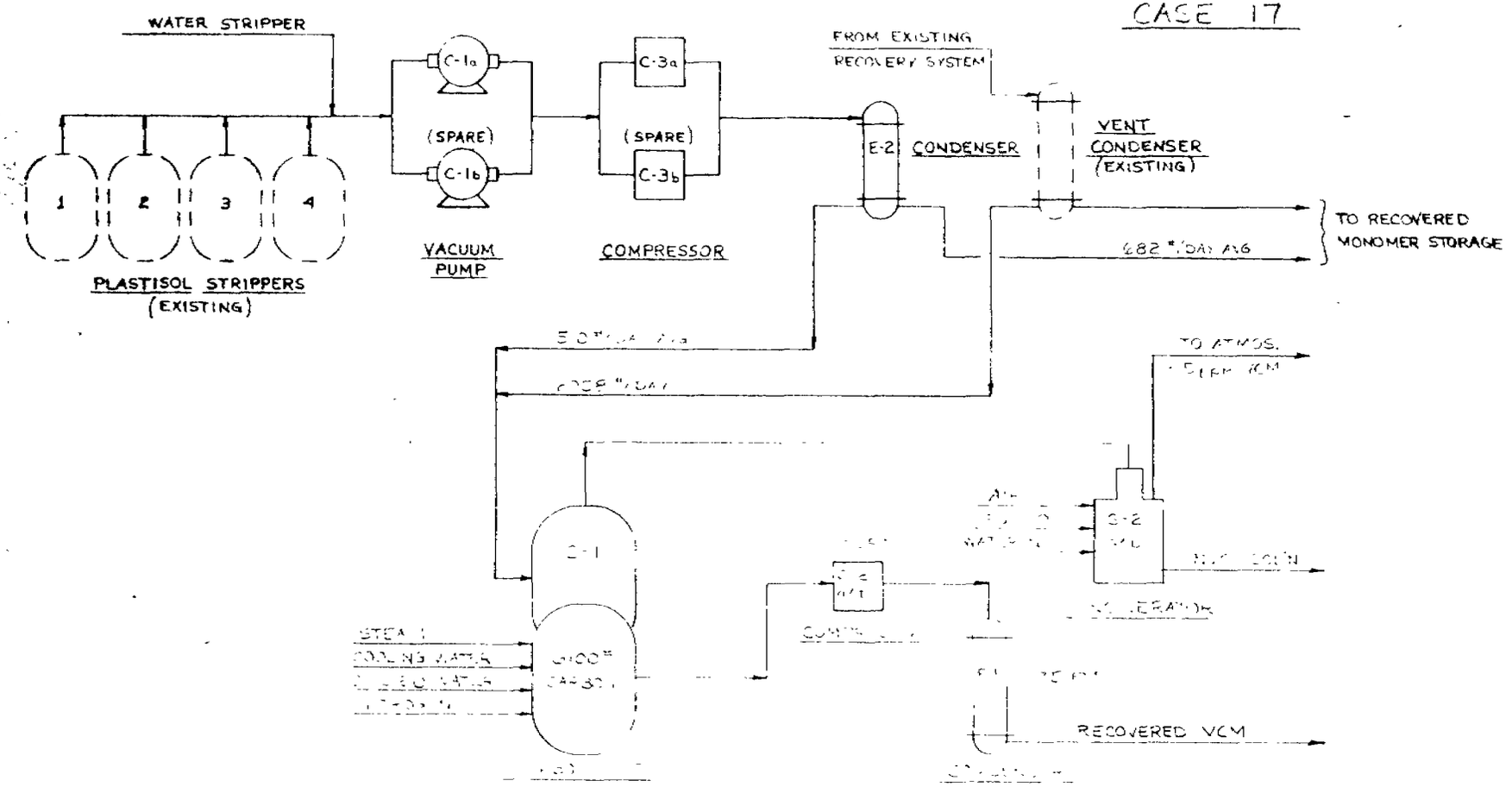

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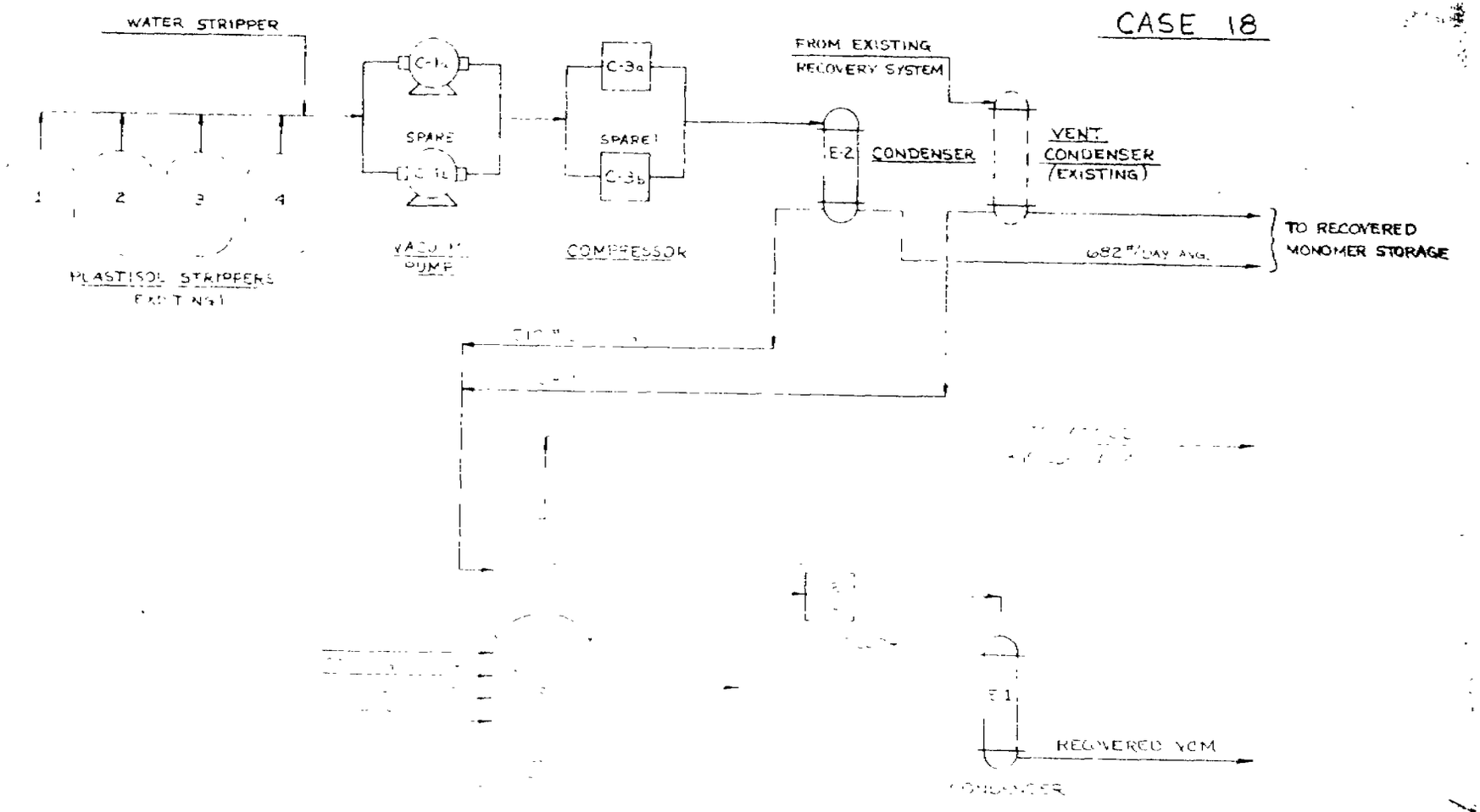

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