No. 53 CRESOL

Section VI. HEALTH HAZARD INFORMATION | TLV: 5 ppm (see section II)

Cresol is corrosive to all body tissues. It is absorbed through the skin to produce systemic effects (sometimes delayed). Damaged tissue will show white coloration then redness and then brown or black. Within 30 minutes, skin absorption can produce headache, dizziness, rapid respiration, and weak pulse with unconsciousness or even death resulting from extended contact. Severe injury or death can result from excessive inhalation exposure or ingestion. CONSULT PHYSICIAN PROMPTLY

First Aid:

Eye Contact: Quickly flush the eyes with plenty of running water, including under the eye lids, for 15 minutes; then get prompt medical attention.

Skin Contact: Remove cresol from skin quickly be washing exposed areas well with plenty of water plus soap until cresol odor is gone. (If contact is gross remove contaminated clothing and shoes under the safety shower). Further washing of skin with isopropyl alcohol of 20% glycerine in water, followed by water rinse, may be desirable. Obtain doctor's care promptly for all except the most minor contact. Inhalation: Remove to fresh air. Have qualified person restore breathing; give oxygen if needed. Get(*see Sec. VII) *physician's care promptly. Ingestion: induce vomiting. Repeat with water until vomitus is clear. Get physician's care.

Section VII. SPILL, LEAK & DISPOSAL PROCEDURES

Notify environmental personnel. Refer to plant SPCC plan. Provide exhaust ventilation. Eliminate ignition sources. Exclude from area all except properly equipped clean-up personnel (see Sect. VIII). A water spray can be used to flush cresol away from sensitive area. (Flush to retention area, not to drain!) Contain and collect material in approved container for disposal. Small spills can be picked up with absorbent solid or paper for disposal.

DISPOSAL: Burn scrap material (or its solution in a flammable solvent) using an approved incinerator, or label properly for disposal through a licensed waste disposal company. Follow Federal, State and local regulations.

Section VIII. SPECIAL PROTECTION INFORMATION

When practical, use cresol in completely enclosed systems to prevent exposure. Provide explosion-proof general ventilation and local exhaust ventilation to meet TLV requirements. Use NIOSH approved respirators where the TLV is or may be exceeded. The selection of the appropriate respiratory protection (organic vapor respirator, self-contained breathing apparatus, supplied air respirator, etc.) will depend upon the actual or potential concentration present. Use rubber gloves, rubber apron, rubber boot, acid-proof suit, etc., as circumstances of used require to prevent body contact. Use chemical safety goggles and in addition a face shield where splashing is possible. Provide deluge-type safety shower. An eye wash fountain shall be located in the work area and be immediately accessible for emergency use.

Section IX. SPECIAL PRECAUTIONS & COMMENTS

Store in a cool, clean, well-ventilated place away from oxidizing agents and sources of ignition. Protect containers from physical damage. Avoid use of aluminum, copper, and brass alloys in contact with cresol in storage and process equipment. Cresol is similar in its toxicity and reactivity to phenol. Workers who handle cresol should be well trained in its hazards and well supervised.

D.O.T. Requirements

NA 2076 COMBUSTIBLE LIQUID (if flash point under 200°F)

PLAINTIFF'S EXHIBIT AL-1109

Section X. REFERENCES

ASTM No.: D 3538 Other:

Chemical Abstracts No.: CAS No. 001-391-773

CMA Chemical Safety Data Sheet No.:

NFPA Guides No.:

NSC Data Sheets:

Supplier Product Literature:

No. 47 Phenol

Section VI. HEALTH HAZARD INFORMATION | TLV 5 ppm or 19 mg/m^3 (TWA)

Phenol is a dangerously toxic material! Poisoning can occur by skin absorption, vapor inhalation or ingestion. Headache, dizziness, nausea, weakness, difficulty in breathing, possible unconsciousness and even death can occur. Oral human LDLo is 0.14 g/kg. In addition to poisonous effects phenol is corrosive to tissue. Initial skin contact produces a white, softened area; later severe skin burning with local anesthesia occurs. The phenol is absorbed through the skin, into the blood, producing systemic toxic effects.

FIRST AID: (Provide help without delay!)

Skin Contact: Wash affected area immediately with alcoholic soap and water, then with alcohol or olive oil. For skin contact over large areas, get medical attention. Observe for systemic toxic effects.

Eye Contact: Promptly wash 15 minutes with running water. Get medical help.

Inhalation: Remove victim to fresh air; obtain medical attention.

Ingestion: Give victim 3 glasses water or milk. Obtain medical attention immediately for gastric lavage and treatment! (NIOSH recommends inducing vomiting.)

Section VII. SPILL, LEAK & DISPOSAL PROCEDURES

Notify Environmental personnel; refer to plant SPCC Plan. Cordon off area; remove heated surfaces and ignition sources; provide adequate ventilation. Wear NIOSH approved self-contained breathing respirators where a large spill surface area allows high vapor concentrations. Prevent skin contact with protective clothing, gloves, etc. Absorb liquid spills on paper, vermiculite or other absorbent and place in a metal container. Scoop solid spills into a metal container.

Dispose of by burning in a high temperature incinerator, or via a licensed waste disposal company. Do not flush down drains!

(Water can be used to flush phenol away from sensitive areas to a holding area.)

Section VIII. SPECIAL PROTECTION INFORMATION

Provide efficient exhaust ventilation to meet TLV requirements. Hoods should maintain 100 lfm face velocity. When phenol is heated vapor inhalation can become a serious hazard where the TLV concentration is or may be exceeded, use NIOSH approved respiratory protection. The selection of the appropriate respiratory protection (chemical cartridge respirator, supplied air respirator, self-contained breathing apparatus, etc.) will depend on the actual or potential concentration present. Prevent skin contact by wearing Buna-N or vinyl gloves, aprons and boots. Use safety glasses in normal use or face shields where splashing may occur. Eye wash and safety showers shall be located in the work area and be immediately accessible for emergency use. Use an alcohol soap to remove phenol from skin. Remove contaminated clothing immediately (under the shower!) and launder before reuse.

Section IX. SPECIAL PRECAUTIONS & COMMENTS

Prevent skin contact! Do not breathe vapors! Remove contaminated clothing immediately! Areas of use or handling should not allow smoking or eating. Store in a cool, dry well-ventilated area, away from heated surfaces, open flames and ignition sources. Separated outdoor storage preferred.

Do not allow employees who have diseases of the central nervous system, liver, kidneys or lungs work in areas of phenol exposure. Preplacement and periodic medical exams should be provided to those working with phenol.

Do not allow untrained workers to handle phenol!

D.O.T. Requirements

POISON B

Section X. REFERENCES

ASTM No.: D2439; D2286 Other: CHRIS (1974)

Chemical Abstracts No.: 000-108-952

CMA Chemical Safety Data Sheet No.: SD-4

NFPA Guides No.:

NSC Data Sheets: 405A

Supplier Product Literature:

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[illegible] = Chemosis and [illegible] Opacity
3 degree = Strong Opacity

Tablet I. Overview of pts treated with Polyethylene 40 Eye irritations from Phenol or similar Chemicals

age	degree of burn	treatment	results
	caused by ~~[illegible]~~	Irigation ~~Irrigation~~	
26	1. Degree ~~Grade~~ "M-oil" both eyes	2 Units # 1:2	The redness of the conjunctiva diminished. Cornia stayed clear
56	Phenol; 1. Degree Left eye	2 Units # 1:2	The redness of the conj. diminished - Cornia stayed.
63	Phenol, 3. Degree Cornia Opaque cloudy	3 Units (strike # 1:1 and ~~physiol~~ saline sol.	During the 1st 20 minutes the redness receded the Corn cleared up
71	"M-oil", 1st degree both eyes	2 Units # 1:2	The redness of the conjunc remained longer but cleared after 2 days. The Cornia Clea
40	Phenol, 3rd degree Cornia partially opaque	3 Units # 1:1 also irrigation with ~~physiol~~ Saline Sol.	Treated by Gov. Hosp quick recovery
25	Phenol, 1-2nd degree both eyes	2 Units 1:2	The redness of the conj. receded quickly Cornia Cl.
21	Phenol, 1st degree right eye	2 Units 1:2	Healed without complicatio.
42	Phenol, 1-2nd degree right eye	2 Units 1:2	" " "
28	Phenol 1st degree both eyes	2 Units 1:2	Conjunctive was still irritated for 2 days. Healed without complication
59	Phenol 1-2 degrees right eye	2 Units 1:2	early diminishing of the redness of the Conj. Cornia stayed clear
29	Phenol 1-2nd degree right eye	2 Units 1:2	Conj. remained red for 4 days - healed without complications.

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COMPARISON OF DECONTAMINATION PROCEDURES FOR ACUTE DERMAL PHENOL EXPOSURES IN SWINE

T. G. Pullin, M. N. Pinkerton, R. V. Johnston, D.V.M., D. J. Kilian, M.D.

Lister introduced the use of phenol in 1867 when he treated compound fractures and reported surgical cures of what had usually been fatal injuries. The drug found immediate popularity with physicians and patients. It was a mild local anesthetic and, when irrigated through a wound, decreased pain.[1] It remained the favorite disinfectant among doctors and surgeons until about 1900. Since that time preparations containing phenol have been used topically as antipruritics and as antiseptic dressings for the treatment of burns, wounds, and indolent ulcers.[2] Since the First World War, phenol has become of tremendous industrial importance as the basic material for the production of a great number of products, such as plastics and explosives.[3] There were 2.3 billion pounds of phenol produced in 1974.[4] Phenolic resins account for about half of the total phenol market, and a major outlet for phenolic resins is as an adhesive in plywood. Phenolic molding compounds account for about 25 to 30% of the total phenolics market. The other two large-volume outlets for phenol are bisphenol-A and caprolactam. Each accounts for about 15% of the phenol market. The two major outlets for bisphenol-A are epoxies and polycarbonates, while better than 90% of all caprolactam goes into nylon.

Phenol has a marked corrosive effect on tissue. It is especially dangerous and insidious because it exerts a local anesthetic action which may prevent perception of painful stimuli until

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serious burns have occurred. Phenol is readily absorbed through the intact skin and mucous membranes of animals and man. It exerts a marked action on the central nervous system (CNS) with the prominent effects in man of CNS depression. The circulation is also markedly depressed by phenol, and death in man due to phenol poisoning is usually due to respiratory or cardiac failure.[5]

Since phenol is manufactured in large quantities for the preparation of great variety of substances, its handling gives rise to some hazard from regularly or frequently repeated exposures, as well as to serious danger from extensive accidental contact.[6] Thus, the manufacture and handling of large quantities of phenol make it important that a rationale be established for procedures for the removal of this material from the skin of persons endangered by its contact.[7]

It is accepted that the best rationale is first aid that removes phenol from the skin rapidly and completely. However, there continues to be considerable controversy over which solvent is best suited for this purpose. Alcohol, water and soap, glycerol or oils are some of the solvents that have been suggested.

Alcohol was the time-honored solvent for removing phenol accidently spilled upon the skin. However, when studies showed that alcohol is absorbed through the skin in sufficient quantities to produce poisoning, the possibility of employing other solvents as a substitute was investigated.[6] Considerable confusion and controversy have subsequently arisen as to which solvent is the

most effective. Water is thought by many to be the most satisfactory agent for general use because it is commonly available in such quantities as to provide efficient means for flooding the whole surface of the body. However, in a recent investigation by Conning and Hayes[8], glycol, polyethylene glycol or a polyethylene glycol/methylated spirit mixture (PEG/IMS) was recommended as the solvent of choice. This prompted the British Chemical Industry Safety Council of the Chemical Industries Association to recommend these solvents for decontaminating skin exposed to phenols. A later publication[9] confirmed Conning and Hayes results when they concluded that a PEG 300/IMS mixture used either as a spray or for swabbing was superior to water in preventing skin damage and systemic effects.

The rat was used as the animal model in both of these studies. Extrapolation of animal data to humans presents a very difficult problem. Most scientists face this problem today by choosing an animal species that most closely resembles man in respect to the particular problem. Since this problem involves absorption of phenol through the skin, an animal with skin similiar to the human is indicated. Such similarities to man as sparse hair coat, a thick epidermis that has a well-differentiated undersculpture, a dermis that has a well-differentiated papillary body and a large content of elastic tissue indicates swine as a better animal model.

In previous studies, mortality and/or skin damage was used as

the primary deciding factor(s) in the conclusions and recommendation of the studies concerning the most effective solvent for removing phenol from the skin. A better, more sensitive indicator of the most effective solvent would be the measurement of the amount of phenol absorbed into the blood.

Thus, this study was undertaken in swine, a better animal model, to determine by measurement of phenol absorbed into the blood whether water or a PEG/IMS mixture is the most effective solvent for decontamination of skin exposed to phenol.

MATERIALS AND METHODS

General. Mixed-breed swine, weighing 15-47 kg. were used in this study. They were fed approximately 5 pounds Purina Pig Chow/pig/day and water ad libitum. They were housed in a climate conditioned animal room measuring 18 by 13 feet.

Phenol AR (Mallinckrodt) with a melting point of 40.5°C was used as a liquid by maintaining it at 45°C in a water bath. The phenol was applied as a liquid film layer over the application site of the swine by use of a roll-on applicator bottle, made from an empty deodorant bottle.

The polyethylene glycol/methylated spirits (PEG/IMS) mixture was used as supplied from an industrial phenol plant. This mixture is currently being used at this plant and consists of a 6:1 ratio by volume of polyethylene glycol 300 to methylated spirit (95% ethanol, 5% methanol).

Prior to the application of phenol to the skin, the animal

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was weighed and restrained, and the hair was clipped from the application site. Blood samples were obtained for analysis prior to and periodically following exposure by means of jugular vein puncture.

After exposure and decontamination, the animals were caged individually for 5-7 days in a metabolism cage. Urine samples were collected for analysis during this period. Necropsy was done on the animals 2-4 weeks post exposure.

Analytical Techniques. Determination of "free" phenol levels in swine plasma was accomplished by gas chromatographic analysis.[10] This analytical procedure was developed in this laboratory since a suitable method could not be located in the published literature. Approximately 2 ml of the heparinized blood sample was centrifuged at 1500 rpm for 5-7 minutes. The plasma was separated and used for analysis without any further sample preparation. The Varian Aerograph Gas Chromatograph, model 2100-21 was used for the analysis with the following parameters:

Mode: Flame ionization detector with electronic peak integration.

Injector Temperature: 150°C

Detector Temperature: 250°C

Column Temperature: 225°C

Carrier Gas: Purified helium, 40 ml/min., 65 psi

Range: 10^{-11} amp/mv

Injection Sample: 5 µl

G. C. Column: 6 ft. x 2 mm I.D. glass, 15% Carbowax 20M-TPA on Chromasorb G, 80/100 mesh, non-acid washed (NAW)

Standards: External phenol-water

Total phenol in the urine, including the conjugates, was also determined by gas chromatographic analysis. Acid hydrolysis is the first step in the preparation of the sample for this analys Four ml of perchloric acid is mixed with 10 ml of urine, and this mixture is heated to 95°C for two hours. After this period the mixture is allowed to cool to room temperature, and then approximately 5-10 g of sodium chloride is added. After mixing, two ml of diisopropyl ether is added and the volume is made up to 25 ml with distilled water. This solution is then mixed for one minute and allowed to stand until the aqueous and ether layers separate. The ether layer containing the extracted phenol is then decanted and saved for analysis. The Hewlett-Packard 5830A gas chromatograph was used for this analysis with the following parameters:

Mode: Flame ionization detector

Injector Temperature: 200°C

Detector Temperature: 225°C

Column Temperature: 150°C

Carrier Gas: Helium 40 ml/min., 50 psi

Range: 10^{-9} amp/mv

Injection Sample: 2 µl

G. C. Column: 6 foot x 1/8 inch stainless steel, 2% Ethylene

Glycol Adipate on Chromasorb G-AW-DMCS

Standards: External phenol-water

Hematology, Clinical Chemistry and Cytogenetics. Hematological, clinical chemistry and cytogenetic analyses were conducted on blood samples that were drawn pre-exposure and 24 hours, 48 hours, 7 days and 14 days post-exposure. The hematological determinations included complete blood count (CBC), while clinical chemistry analyses consisted of blood urea nitrogen (BUN), serum glutamic pyruvic transaminase (SGPT), serum glutamic oxalacetic transaminase (SGOT) and creatinine determinations. Cytogenetic analysis was performed on the peripheral lymphocytes. The methodology used for culture of the lymphocytes and preparation and scoring of the slides is described in a separate publication.[11]

Dermal Toxicity. Since there was no information regarding the dermal toxicity of phenol to swine, a series of experiments was performed to determine a proper dosage for the decontamination studies. The amount of phenol, the surface area of the application site and the duration of exposure before decontamination were investigated. It was desirable to have the dosage and duration that would realistically simulate an industrial exposure and produce a plasma phenol level curve as determined by the gas chromatograph.

Decontamination Studies. After the initial series of experiments on dermal toxicity was completed, the amount of phenol, surface area involvement and duration of exposure were

defined for the decontamination studies. These were maintained constant throughout the studies so that the only variable was the type of decontamination.

Phenol was applied topically at a dosage of 0.05% of the body weight over 35-40% of the total body surface area. It was left in contact with the skin for either one or 2.5 minute(s). This was followed by showering with water for 15 minutes with or without scrubbing, or swabbing with a PEG 300/IMS mixture for 15 minutes. The water shower was accomplished by use of the same type of safety shower apparatus that is used in an industrial chemical plant for personnel decontamination in the event of chemical exposure. Swabbing was achieved by use of a single white 8 x 8 in. washcloth soaked to excess in the PEG 300/IMS fluid, and rubbing the contaminated area in all directions with slight hand pressure. Approximately three gallons of the PEG 300/IMS were us during the decontamination period for each animal.

Control animals were exposed to phenol in exactly the same manner as the experimental animals with the exception that no decontamination procedure followed the exposure to the control animal.

Four female pigs were used in each of the water decontamina and PEG 300/IMS decontamination groups and three in the control group.

Data from each group was compared and statistically analyze

under the direction of Dr. E. B. Whorton, Jr., Associate Professor and Director, Division of Biometry, Department of Preventive Medicine and Community Health, University of Texas Medical Branch, Galveston, Texas.

RESULTS

Dermal Toxicity

It was determined through a series of trial exposures that a topical application of undiluted phenol to swine consisting of 0.05% of the body weight over 35-40% of the total body surface for a duration of one minute produced a plasma phenol level curv that was well within the limits of detectability of the gas chro matograph. This dosage was lethal to two of three swine that were not decontaminated.

Several other facts became evident from this initial series of experiments. Phenol was absorbed rapidly and was slowly elim inated from the blood. The data from these initial trial exposu showed (Table 1) that increasing the quantity of phenol did not correspondingly increase the amount of plasma phenol as substantially as did increasing the surface area. - True!

In the determination of the duration of exposure to use in the decontamination studies, an interesting fact became evident seen in figure 1. There was no significant difference (F=1.5; p between the free plasma phenol levels of a one minute exposure c pared to a 2.5 minute exposure.

An ancillary study developed out of this initial set of experiments from the desire to learn if washing with soap and water was more efficient than water alone. As seen in figure 2, soap and water did not differ significantly ($F < 1$; $p > .25$) in the amount of free plasma phenol levels.

Decontamination Studies

Control - No Decontamination. In all of the control animals there was a rapid onset, within 3-5 minutes post exposure, of twitching and tremors of the muscles. This occurred first in the muscles of the face and eyelids, then spread over the entire body with uncoordinated movements of the legs becoming very prominent. The pupils became dilated within 3-5 minutes after exposure. There was some salivation and nasal discharge, and dyspnea was observed approximately 5 minutes after exposure. One of the three control animals began to improve 6-8 hours post-exposure and was markedly improved at 8 hours post-exposure. However, the other two animals expired within 95 minutes after exposure. The signs in these two animals became progressively worse until they lapsed into a general state of lethargy 5-7 minutes before death. At this point the tremors diminished followed shortly by convulsions, coma, fully dilated eyes, cyanosis and death (Table 2). Post-mortem examination of these two pigs showed no gross pathological changes.

Following exposure to the phenol, a white pellicle was formed within 3 minutes over the entire area of the exposed skin. This was followed by severe edema at 8 minutes and discoloration of the

area at 20-30 minutes. The skin was penetrated deeply, and there was extensive necrosis.

The mean peak concentration of 52.6 ppm free plasma phenol occurred in the surviving animal at 1.75 hours post exposure (figure 3). Phenol was still detectable in the blood at 8.75 hours post exposure, but the analysis on the next sample drawn at approximately 23 hours post exposure showed no free plasma phenol level. The highest value of free plasma phenol, 56.4 ppm, recorded in this study occurred in a control animal that expired.

Total urinary phenol concentration reached a peak of 19,958.8 ppm 6.75 hours after exposure in the control animals. As figure 4 shows, the peak was followed by a rapid elimination until pre-exposure levels were approached at approximately 53 hours after exposure.

Water Decontamination. There was no mortality in the animals decontaminated with the water shower (Table 2). The signs of acute phenol poisoning, though not as severe, were the same in these animals as in the control pig that survived. Also, these swine began to improve 2-3 hours post-exposure and were considerably more lively 24 hours after exposure.

The white pellicle was present in scattered patches over the phenol exposed area in these swine, and was not as extensive as in the control animals. These patches were no longer discernable after one hour, and at this point the entire exposed area was erythematous. Edema was first observed at 8-12 minutes in these

animals, but it did not become severe until approximately one hour after exposure. There was no discoloration of the skin and necrosis was slight to moderate.

Phenol was absorbed rapidly in spite of the water decontamination. The peak concentration of 24.2 ppm free plasma phenol occurred at 15 minutes post exposure. However, as the curve in figure 3 shows, water decontamination significantly reduced the amount and retention time of absorbed phenol when compared to the controls.

The effect of water decontamination upon total urinary phenol concentration closely follows that of the free plasma phenol levels This type of decontamination, as noted in figure 4, reduced the highest amount of total urinary phenol concentration to 7485.9 ppm. This is a substantial reduction when compared to the controls. However, the time required for elimination, 53.75 hours was approximately the same as that of the controls.

PEG 300/IMS Decontamination. There were no significant observable differences between the clinical responses of the swine decontaminated with PEG 300/IMS and those described of the pigs decontaminated with water. As noted in Table 2, there also was no mortality in these swine. (what if one pig had died, they'd have really condemned PEG3a

Figure 3 shows that the plasma phenol absorption curve of the animals decontaminated with PEG 300/IMS is almost identical to the curve of the water decontamination. The peak concentration of 21.70 ppm plasma phenol occurred at the same time, 15 minutes

post-exposure, but was slightly lower than that of the water treatment. The retention time, 2.75 hours, was slightly shorter than the 4.25 hours of the water treatment, but overall there was no significant difference ($F < 1$; $p > .25$) between the water and PEG 300/IMS decontamination curves. However, as found for the water treatment, PEG 300/IMS significantly decreased both the amount and retention time of absorbed phenol when compared to the control.

The PEG 300/IMS total urinary phenol curve is very much like the water total urinary phenol curve with identical elimination times of 53.75 hours (figure 4). When compared to the controls, PEG 300/IMS decontamination, like water, substantially decreased the amount of the highest total urinary phenol concentration. In the case of the PEG 300/IMS, the peak was 5546.9 ppm. All values for PEG-3 & IMS are lower

Necropsy. There were no significant gross pathological changes other than the skin lesions in any of the animals at necropsy.

Hematology and Clinical Chemistry. There were no phenol-related differences between the hematological and clinical chemistry determinations accomplished in the water and PEG 300/IMS decontaminated swine and those accomplished in the control swine.

Cytogenetics. Chromosomal aberration data is presented in Table 3. There were 11 abnormal cells found out of the 1125 cells (0.9%) scored in this study. Only one aberrant cell was discovered in the 125 metaphase spreads (0.8%) of the control group. In comparison, ten abnormal cells were found in the

PEG/ vs H_2O
Peak concentration 21.7 ± 24.2 ppm
Retention time 2.75 ± 4.25 hours
Peak urinary Conc. 5550 ± 7485 ppm
No statistical significance?

1000 spreads (1.00%) of the decontaminated groups.

No aberrations were discovered in the control group after the phenol exposure. There was only one aberration (chromatid) found in the water decontaminated swine after exposure, and this was not significantly different from the control. One chromosome and six chromatid aberrations were discovered in the PEG 300/IMS decontaminated group after the phenol exposure, but this was not significant when compared to the control.

DISCUSSION

Dermal Toxicity

Phenol was very caustic to the skin of the pigs, producing extensive local necrosis in a short time. Apparently it acted on the tissue by denaturing and coagulating the proteins as evidenced by the white pellicle of precipated protein. PEG 300/IMS was not observed to have an antidotal effect on the phenol burns in the swine. There was no significant difference between the severity of the skin necrosis and amount of exfoliation produced when either PEG 300/IMS or water decontamination was utilized. → This is only a human observation!

Phenol was also absorbed rapidly in all swine regardless of when decontamination occurred. The signs of systemic phenol intoxication in the swine were similar to those described in other animals by earlier investigaters[6]. First there was a stimulation of the central nervous system (CNS) as manifested in muscle tremors. In those animals that died, this stimulation was manifested further

in convulsions. This was soon followed by depression of the CNS and death resulted from respiratory failure.

Although the number of animals used in the initial trial exposure was small, the data, within the limits of our experiments, indicated that the quantity of phenol is not the only major factor influencing absorption of phenol. That is, the extent of the absorption of phenol by a unit area of skin is little influenced by the quantity of phenol applied, but is largely dependent upon the magnitude of the skin area exposed. A logical explanation of this result, which agrees with the conclusions of Deichmann et al[7] and Conning and Hayes[8], is that injury to the skin in the area of contact in the form of a coagulative pellicle results in retardation of the rate of absorption of phenol. This is further confirmed by the fact that a 2.5 minute exposure did not significantly increase the amount of free plasma phenol level over a one minute exposure.

Urinary phenol excretion was rapid in the swine with maximum elimination occurring from approximately 6 to 48 hours post exposure. Complete elimination of urinary phenol took approximately two days in all animals, and this is somewhat similar to the results of Piotrowski[12]. He found that elimination of urinary phenol from human volunteers exposed to phenol vapor through the skin occurred within one day.

It has been stated that phenol is capable of breaking chromosomes[13], but this was not evident in this study. The acute dermal

phenol exposures with or without decontamination produced no significant increase in chromosome aberrations above the background level. However, it should be emphasized that this study involved acute dermal exposures. Cytogenetic analysis of chronic phenol exposures should be performed before it is concluded that phenol is incapable of inflicting chromosomal damage.

Decontamination Studies

According to Conning and Hayes[8], the coagulated protein layer produced by phenol on the skin appears to interfere with the ability of other liquids to penetrate the skin. The result is that merely pouring fluid on the affected skin is ineffective. They recommended a swabbing procedure, thereby removing the superficial layers of necrotic keratin, with swabs soaked in a polyethylene glycol/methylated spirit mixture. They further stated that a substantial therapeutic effect is achieved.

The results of this study contradict these conclusions. There was no significant difference between the free plasma phenol curve of water decontamination and that of the PEG 300/IMS. This result points out that flooding with water was as effective as swabbing with PEG 300/IMS and that PEG 300/IMS has no more therapeutic value than water. The ineffectiveness of the mechanical action of swabbing is further borne out by the fact that swabbing with soap under a water shower did not significantly reduce phenol absorption when compared to showering with water with no swabbing (figure 2).

Both water and PEG 300/IMS are effective treatments for phenol

burns and systemic phenol intoxication. In comparison to the exposed controls, toxic signs, burn severity and recovery time were substantially reduced when either of these solvents were used. Both reduced mortality by diminishing phenol absorption as indicated by the plasma phenol curves.

Thus it follows that flooding with water or swabbing with PEG 300/IMS over the contaminated skin area, thereby accomplishing prompt removal of the phenol, is a very effective emergency treatment. However, of the two solvents studied, only water is commonly available in such quantities as to provide efficient means for flooding the whole surface of the body. For this reason, and this reason only, water is the most satisfactory decontaminant for acute dermal phenol exposures.

SUMMARY

Phenol is an important industrial chemical that is capable of producing fatalities in humans due to systemic absorption through the skin. This risk dictates that the most effective decontamination procedure be established for the rapid removal of this material from the skin. Recent reports in the literature caused controversy by concluding that a polyethylene glycol/methylated spirits (PEG/IMS) mixture was superior to water as a decontaminant for acute dermal phenol exposures. This study was undertaken to determine which of these materials is the most effective first aid measure. Molten

phenol AR was applied topically to swine at a dosage of 0.05% of the body weight over 35-40% of the total body surface area. Duration of the exposure was one minute followed by either showering with water for 15 minutes or scrubbing with PEG 300/IMS (6:1 by volume) for 15 minutes. Controls consisted of phenol exposed swine with no decontamination. Blood was drawn prior to and periodically after exposure for gas chromatographic analysis of "free" phenol plasma levels. Phenol was absorbed rapidly with peak concentrations occurring at 15-30 minutes regardless of the decontamination procedure. Decontamination substantially reduced the amount and retention time of absorbed phenol when compared to exposed controls. However, there was no statistically significant difference in the amount and retention time of absorbed phenol after the water and PEG 300/IMS decontamination procedures. These data plus the universal availability of water indicates that it is the decontaminant of choice.

ACKNOWLEDGEMENT

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ACCREDITATION

The Biomedical and Comparative Toxicology Research Laboratory is fully accredited by American Association For Accreditation Of Laboratory Animal Care.

[illegible] Phenol Marasmus. J. Occ. Med. 14:55-56, 1972.

2. Freeman, M. V., J. H. Drai[illegible] and Elsie Alvarez. Cutaneous Absorption of Phenol. J. Lab. Clin. Med. 38:262-266, 1951.

3. Deichmann, William B. Local and Systemic Effects Following Skin Contact With Phenol-A Review of the Literature. J. of Ind. Hygiene & Tox. 3:146-154, 1949.

4. Anderson, Earl V. Pickup in phenol awaits housing upturn. Chemical & Engineering News. 53, no. 30:8-9, 1975.

5. Goodman, Louis S. and Alfred Gilman. The Pharmacological Basis of Therapeutics. Fourth Edition. The MacMillan Co., New York. pp.1035-1037.

6. Deichmann, Wm. B. and S. Witherup. PHENOL STUDIES VI The Acute and Comparative Toxicity of phenol and o-m-and p-Cresols for Experimental Animals. J. Pharmacol. Exp. Ther. 80:233-240, 1944.

7. Deichmann, Wm. B., Sulvan Witherup and Marjorie Dierker. PHENOL STUDIES XII. The Percutaneous And Alimentary Absorption Of Phenol By Rabbits With Recommendations For The Removal Of Phenol From The Alimentary Tract Of Skin Of Persons Suffering Exposure. J. Pharmacol. Exp. Ther. 105:265-272, 1972.

8. Conning, D. M. and M. J. Hayes. The Dermal Toxicity of Phenol: an investigation of the most effective first-aid measures. Brit. J. Industr. Med. 27:155-159, 1970.

9. Brown, Vernon K. H., Valerie L. Box, Barry J. Simpson. Decontamination Procedures for Skin Exposed to Phenolic Substances. Arch. Environ. Health 30:1-6, 1975.

10. Pinkerton, Michael N. A Simple and Precise Technique for the Determination of Phenol Concentrations in Swine Blood Plasma. The Dow Chemical Company Report in Progress, 1976.

11. Mensik, D. C., R. Langfitt. Cytogenetic Effects in Swine Following Acute Dermal Phenol Exposure. The Dow Chemical Company in Progress, 1976.

12. Piotrawski, Jerry K. Evaluation of exposure to phenol: absorption of phenol vapor in the lungs and through the skin and excretion of phenol in urine. Brit. J. Industr. Med. 28:172-178, 1971.

13. Goldstein, Avram, Lewis Aronow and Sumner M. Kalman. Principles of Drug Action The Basis of Pharmacology. First Edition. Harper & Row, New York. pp.647-653.

TABLE 2

EFFECTS OF DECONTAMINATION ON SWINE FOLLOWING ONE MINUTE DERMAL EXPOSURE* TO UNDILUTED PHENOL

Type Decontamination	Responses Observed After Decontamination									
	Tremors and Twitching	Dilated Pupils	Salivation and Nasal Discharge	Dyspnea	Convulsions	Coma	Cyanosis	No. Deaths/ No. Animals	Dermal Necrosis	Recovery Time
None	++	++	++	++	++	+	+	2/3	Severe	8 Hrs.
Water Shower-15 Minutes	+	+	+	+	-	-	-	0/4	Slight to Moderate	2-3 Hrs.
PEG 300/IMS Swab 15 Minutes	+	+	+	+	-	-	-	0/4	Slight to Moderate	2-3 Hrs.

*Dermal Exposure - Amount Phenol Applied = 0.05% Body Weight of Pig.

Approximate Surface Area Exposed = 35-40% Of Total Body Surface Area.

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Type Decontamination	Time - Post Exposure	No. Cells Scored	Multiple Aberrations/Cells Scored (1)	Miscellaneous Aberrations/Cells Scored (2)	Chromatid Aberrations/Cells Scored (3)	Chromosome Aberrations/Cells Scored (4)	No. Abnormal Cells/Cells Acored (5)
NONE	Pre-exposure	25	0	0	0.04	0	0.04
(Control)	24 hrs.	25	0	0	0	0	0
	48 hrs.	25	0	0	0	0	0
	7 days	25	0	0	0	0	0
	14 days	25	0	0	0	0	0
WATER	Pre-exposure	100	0	0	0.01	0	0.01
	24 hrs.	100	0	0	0	0	0
	48 hrs.	100	0	0	0	0	0
	7 days	100	0	0	0	0	0
	14 days	100	0	0	0.01	0	0.01
PEG 300/IMS	Pre-exposure	100	0	0	0.01	0	0.01
	24 hrs.	100	0	0	0.02	0	0.02
	48 hrs.	100	0	0	0.01	0	0.01
	7 days	100	0	0	0.01	0	0.01
	14 days	100	0	0	0.02	0.01	0.03

(1) Multiple Aberrations -- A heavily damaged cell containing aberrations too numerous to count.

(2) Miscellaneous Aberrations -- A cell containing pulverizations, endoreduplications, etc.

(3) Chromatid Aberrations -- Includes breaks with fragment, breaks without fragments, intrachanges, and interchanges.

(4) Chromosome Aberrations -- Includes breaks with fragments, breaks without fragments, dicentrics, rings, translocations, and inversions.

(5) Abnormal Cells -- Scored as one if a cell contained any of the abnormalities listed in notes (1), (2), (3) or (4).

*Dermal Exposure -- Amount Phenol applied = 0.05% body weight of pig.
Approximate surface area exposed = 35-40% of total body surface area.
Duration of exposure before decontamination = one minute.

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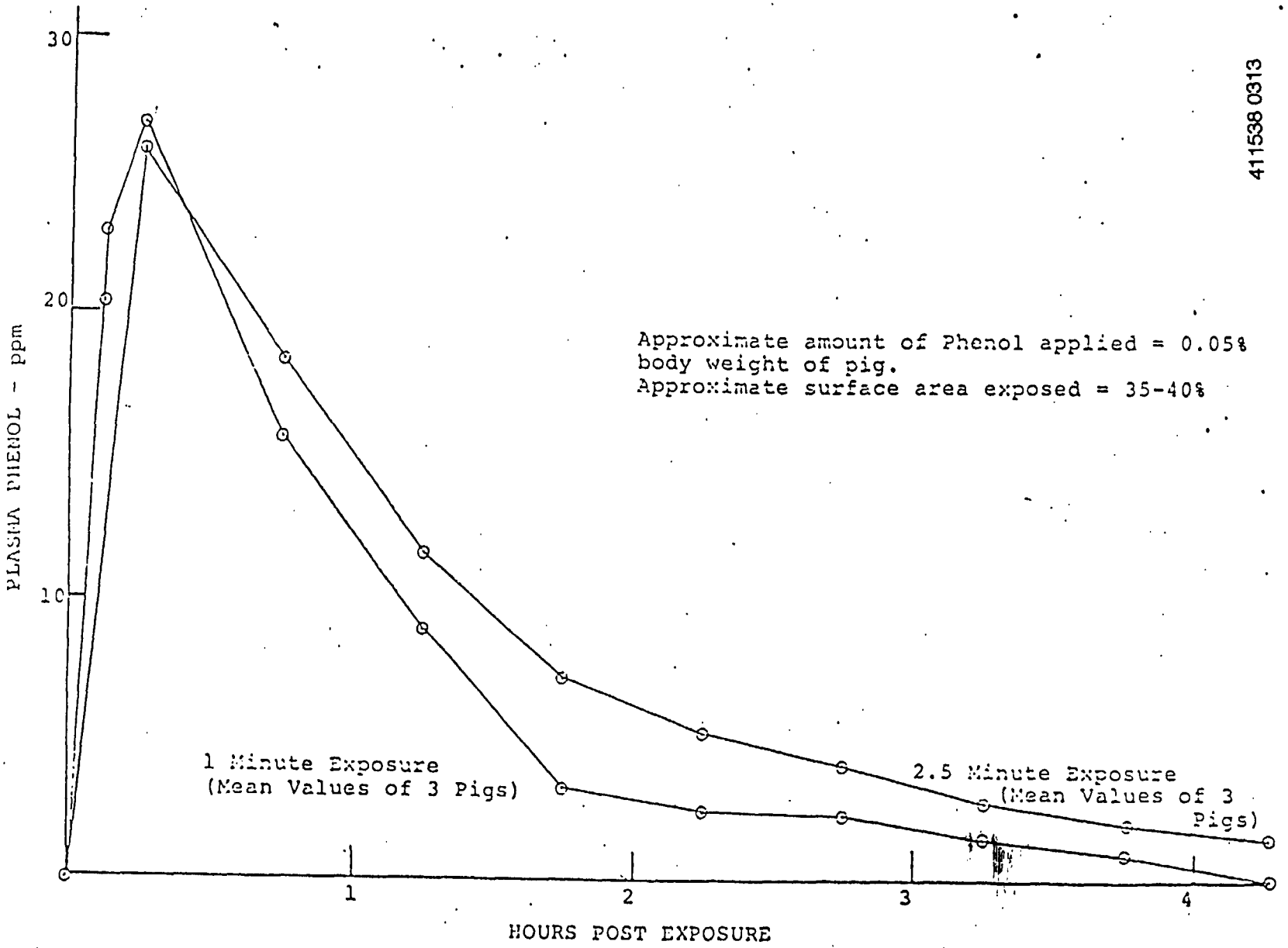

Figure 1 Free plasma phenol levels in swine following varying exposure times and 15 minute showering with water.

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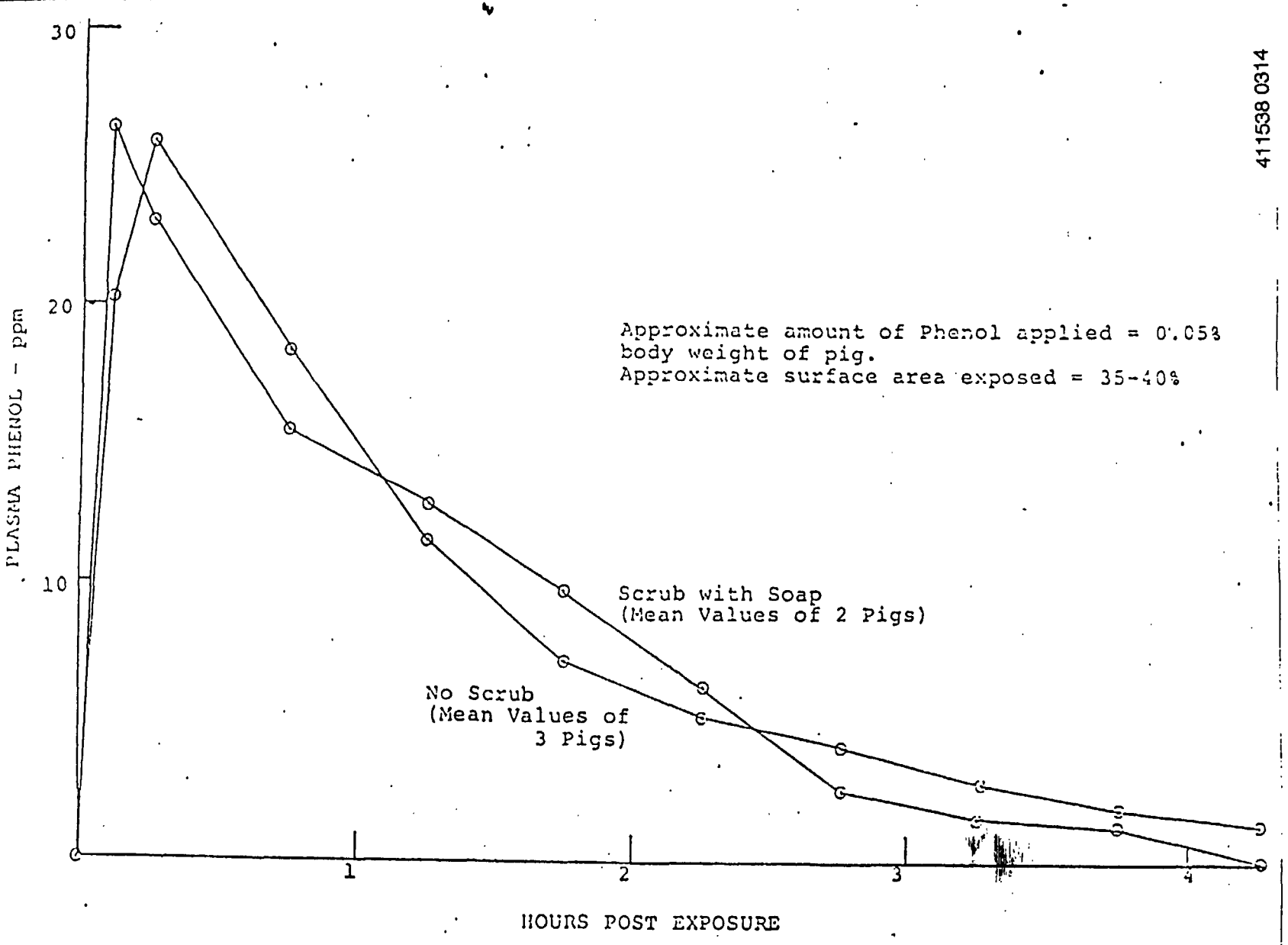

Figure 2 Free plasma phenol levels in swine following 2.5 minute exposure and 15 minute showering with water.

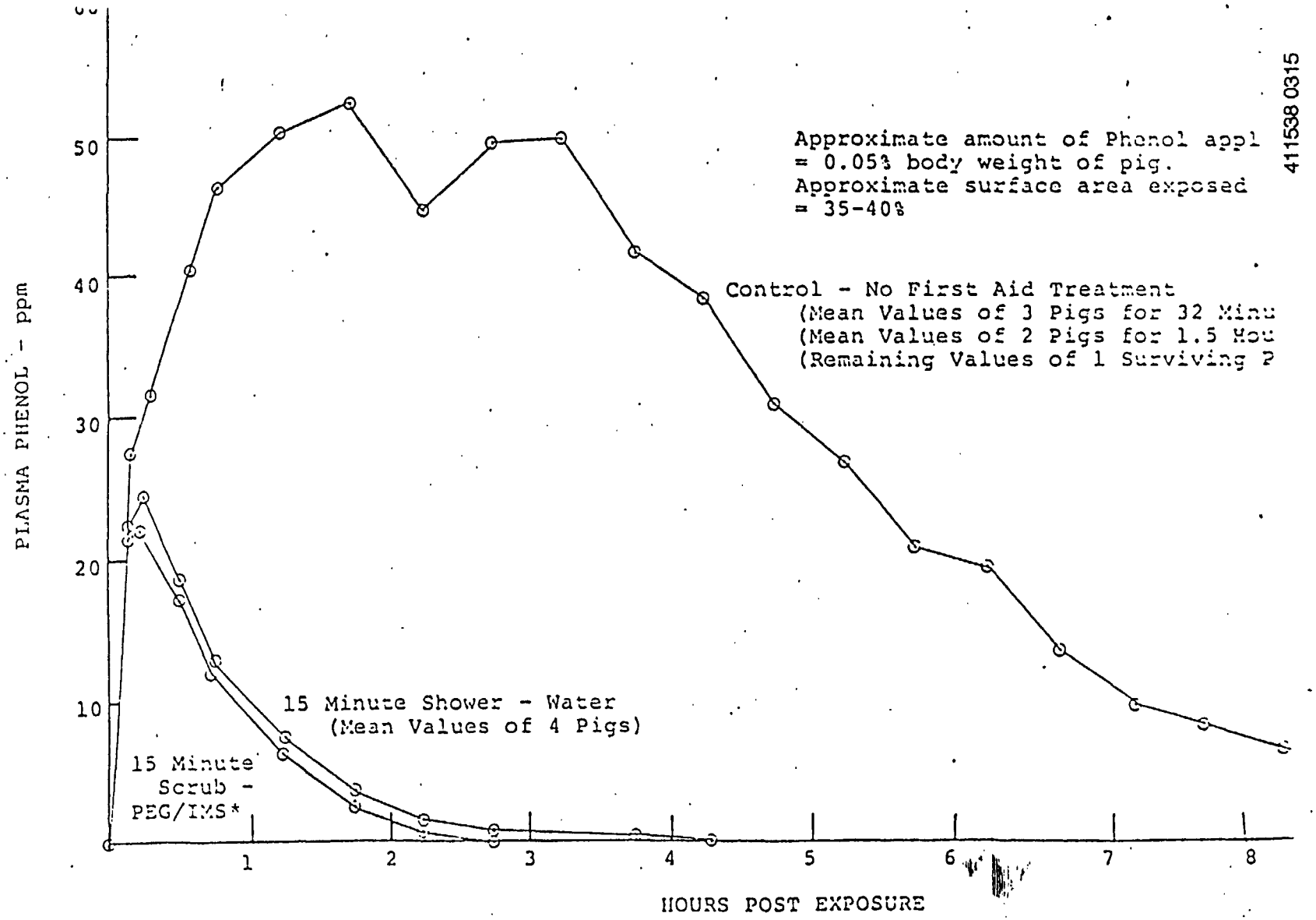

Figure 3 Free plasma phenol levels in swine following 1 minute exposure.

*Mean Values of 4 Pigs.

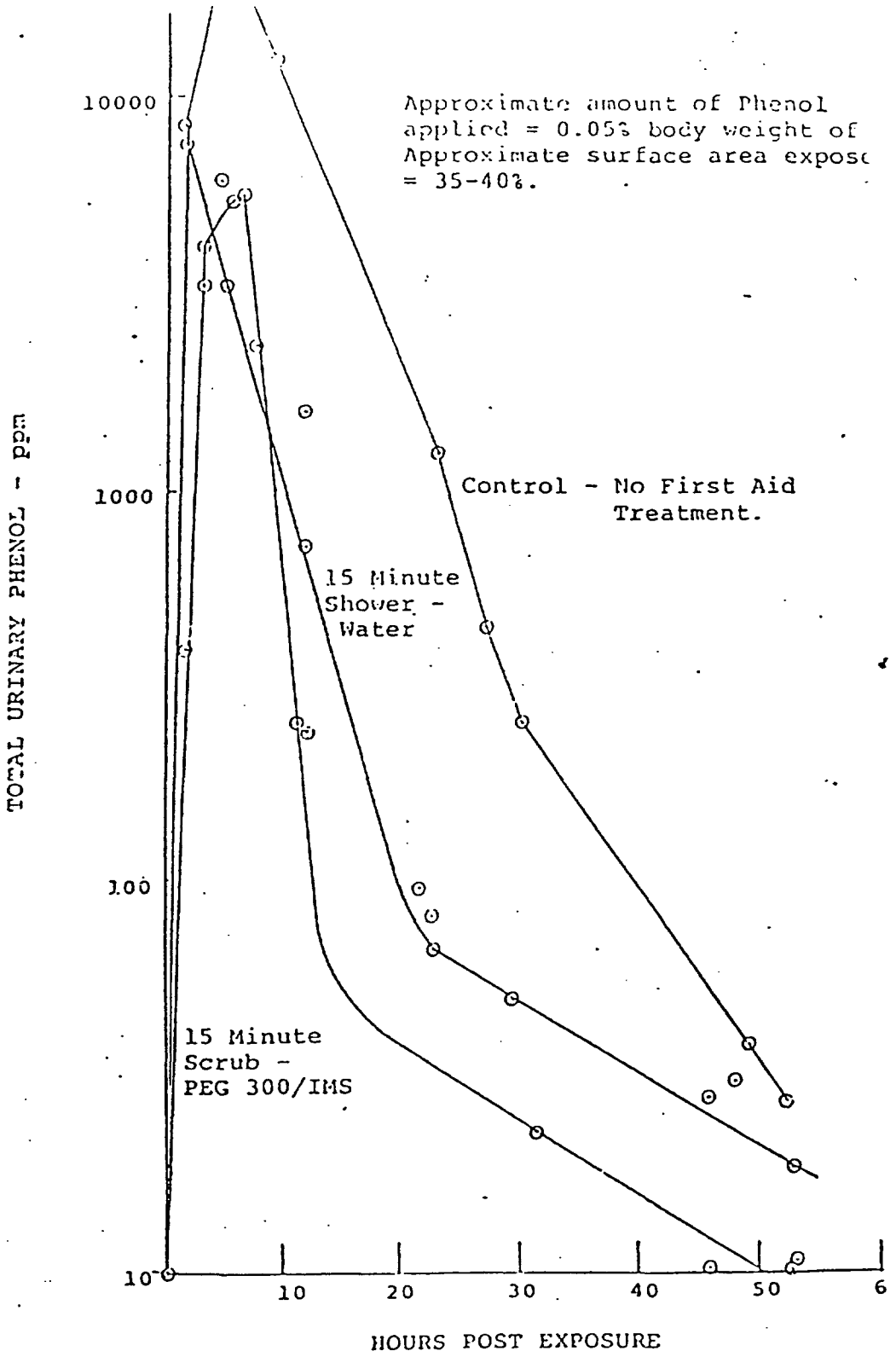

Figure 4 Total urinary phenol levels in swine following 1 minute dermal exposure to undiluted phenol.

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November 15, 1977

File c
Pg Programs!

Judy K. Aeschliman, R. N.
Phelps Dodge Magnet Wire Company
P. O. Box 600
Fort Wayne, Indiana 46801

Dear Judy:

Thank you for your letter of October 25, 1977. We have not conducted any laboratory experiments, nor treated any further phenol burn victims. All I can offer is another reference on phenol burns, this one from a German source in which Polyethylene glycol was found to be better in the treatment of phenol burns of the eyes, and some comments on the Dow Corning communique.. The eye reference is written in German. Lang, K. Zeitschrift Fur Artzliche Fortbild (Jena) 63 (13) 705-708, 1 July, 1969. The author describes his experience at the Leuna Chemical Works. Previously they used 15% ethyl alcohol for irrigation of the eyes for a period of ten years. This was followed by distilled water, and at present, Polyethylene glycol 400. During the period 1965 to 1968, good results were observed with Polyethylene glycol. The use of water and alcohol for first aid frequently led to a clear cornea after the first aid treatment, which became opaque after five days, presumably due to continuing absorption of phenol.

My comments on the Dow Corning communique are as follows: The use of pigs as an animal model in this publication is better than those used by other investigators. It is unfortunate that they only used water and the Polyethylene glycol/industrial methylated spirits solution. They describe industrial methylated spirits as 95% ethanol and 5% methanol. The Polyethylene glycol (PEG) alone, or diluted 50% with water, was not studied. It has been shown that ethyl alcohol can be absorbed through the abraded skin, and our argument is that toxic blood levels of alcohol can occur, especially if methanol is used. Also, alcohol is an excellent solvent for phenol and would act as a carrier for the phenol through the skin, increasing the blood level. There are no blood alcohol levels quoted in this article from Dow. The blood urea nitrogen levels were mentioned in the materials and methods and also blood studies, but no results were published except the chromosome damage. In our series,

Amen!

One patient sustained significant renal damage from the absorbed phenol and another, mild to moderate damage. In addition to the renal damage, our patients also sustained red cell hemolysis and persistent anemia with depressed reticulocyte counts, which suggest bone marrow damage. In view of these serious side effects, I think that anything that will minimize absorption of phenol should be used.

I cannot argue with the recommendation of the initial immediate flushing "with copious quantities of water" and have made the same recommendation in our section on first aid. "Speed is of the essence" and most of the phenol can be removed by this method. However, the concentrated phenol forms a phenol/protein complex in the epidermis and this superficial slough should be rubbed vigorously away. If water-soaked sponges are used, the phenol can be diluted and further absorption through intact, as well as damaged skin may occur. Therefore, it is best to use the PEG at this time. I disagree strongly with Doctor Scharnweber's statement that water should be "continued to be used" when it appears a better method is available to minimize phenol absorption.

The Polyethylene glycols can be diluted 50% in water and still act as a more efficient solvent and decontaminant than water alone. Therefore, great quantities of PEG need not be stored. Most industrial firms using phenol would have safety showers and a deluge system and these would naturally be the first to be used in any accident. Polyethylene glycol should be available in the first aid station and I doubt if this would interfere with the use of copious water irrigation as the Dow people suggest.

I have enclosed a copy of our reprint with the pertinent statements underlined. I believe there is enough clinical and experimental evidence on the efficacy of PEG to warrant its use at the first aid level of care. The cost is not great and the rules for decontamination are not complex.

If you have any further questions, I would be happy to try and answer them.

Yours sincerely,

Russel Pardoe

Russel Pardoe, M. D., Chief
Plastic & Reconstructive Surgery

RP:rp

Glycol Solvent Used in Phenol Burn Therapy

Medical Tribune Report

SAN ANTONIO—The use of pure water should be avoided in the emergency treatment of severe phenol burns, according to Dr. Russel Pardoe, chief of the Division of Plastic and Reconstructive Surgery at Santa Clara Valley Medical Center in San Jose, Calif.

The best solvent to use is polyethylene glycol 400, diluted 50% with water, Dr. Pardoe told the American Burn Association meeting here. Undiluted polyethylene glycol absorbs water, has a heative dilution of about 10°C, and would introduce the risk of further thermal injury. Diluted, however, the polyethylene is "less viscous," and it "is an extremely good solvent for the phenol. It will take out other chemicals that might have been mixed with the phenol, and it doesn't have toxic fumes," Dr. Pardoe said.

Phenol is now being used more commonly than before in household products such as dry cleaning fluids, Dr. Pardoe said. One man that he treated had left cleaning fluid on his trousers for 24 hours, and it burned through the skin to his kneecap.

Dr. Pardoe stressed however, that the best and cheapest form of treatment for any burns is prevention. He said any factory or establishment that has phenol on the premises should be very well prepared.

"It's important to have rescue gear and first aid equipment and good communications," he said.

Protective clothing of proper design is important not only for employees working in aeas where phenol is stored or used, but also for the rescue team as well. Dr. Pardoe cited one industrial accident in which about 15 rescuers sustained burns. The only man who was wearing protective clothing in that accident received the deepest burn because his clothing trapped the phenol solution around his belt and in his boots.

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PHENOL BURNS*

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Roland T. Minami
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*Presented at the eighth annual meeting of the American Burn Association, San Antonio, Texas, April 1-3, 1976.

Requests for reprints should be addressed to:

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PHENOL BURNS

Summary

This case report describes the treatment of 3 men who suffered severe phenol burns while working in a chemical manufacturing plant. Emergency room care is described, and treatment received in the burn unit at the Santa Clara Valley Medical Center is evaluated. The toxic action of phenol, and its effects on the human system, are explained. Based on the experience with these 3 patients, specific precautions are outlined for those who are in contact with phenol, and a method of treating phenol burns is presented, including a discussion of the most effective solvents.

"It appears that all that is requisite is to dress the wound with some material capable of killing the septic germs, provided that any substance can be found reliable for this purpose, yet not too potent as a caustic."

Joseph Lister, 1867.

Phenol (carbolic acid) is an aromatic hydrocarbon derived from coal tar. It was discovered in 1834 and initially used in crude form for the treatment of sewage.

Its antiseptic action on human wounds was first demonstrated by Lemaire in France in 1864 (Lemaire, 1865) and Lister in Scotland in 1867 (Lister, 1867). Lister reported the successful use of phenol in the treatment of compound fractures. Five years later, he recommended a phenol spray, in a concentration of 1 part phenol to 40 parts water, for antisepsis in the operating theater. In 1881, Koch (Koch, 1881) quantitatively evaluated the germicidal action of phenol. The phenol coefficient is still the standard to which the bactericidal action of other

germicides is compared (Rideal and Walker, 1903).

Human injury from phenol contact was recognized over 100 years ago. "Phenol marasmus" was described as an occupational hazard, especially to surgeons and their assistants, resulting from chronic contact and inhalation of phenol. Symptoms of this syndrome included weight loss, headache, vertigo, dark urine, and anorexia, and sometimes death resulted (Merliss, 1972).

The medical literature contains numerous reports of severe injury or death from phenol burns of the skin (Table I). Because of its toxicity, the use of phenol in medicine is now limited. It is used for chemical face peeling, for nerve injections, and as a topical anesthetic for skin and mucous membranes.

Phenol is widely used in industry for disinfectants in cleaning agents, in cosmetics, and as an intermediary in the manufacture of plastics, dyes, fertilizers, and explosives.

CASE REPORT

Three men were working in a chemical plant which manufactures plastic insulating material. They were transferring a solution containing 50 per cent phenol at 200°C from one 1,000 gallon tank into an adjacent tank containing 400 gallons of cold water and small quantities of hydrochloric acid, potassium hydroxide, and parachlorotoluene. The agitator was not turned on until late in the transfer, resulting in a steam explosion which forced open the steel tank cover and sprayed hot liquid and vapor for a distance of approximately thirty feet in all directions.

At the time of the accident, two of the men (E.S. and D.G.) were not wearing protective safety clothing, while the third man (H.V.) was wearing a vinyl jacket and trousers with ankle-length boots.

Two men (H.V. and D.G.) escaped across a catwalk to safety showers. E.S. was trapped behind the vessel by the spray and jumped through a narrow opening between the floor and the wall to the ground twelve feet below. As a result, he

was exposed to the spray for the longest time.

A deluge system was activated 10 to 20 seconds after the accident occurred, releasing 0.3 gallons of water per minute per square foot, a spray heavy enough to limit vision to less than 6 feet. In the safety shower, Virgil H.V. initially removed only his jacket and he consequently sustained the deepest burns of the lower extremities, especially around the ankles (Fig. 1).

The 3 men collapsed and were carried to safety showers outside the building. All their clothing was removed, and they were supported under the showers and scrubbed with soap. They were then transported by ambulance to the emergency room at Stanford University Medical Center, where they arrived approximately 30 minutes after the accident.

Emergency Room Care

On arrival at the emergency room, it was estimated that 2 of the men (H.V. and E.S.) had approximately 60 per cent total body surface area burns, and the third man (D.G.) had approximately 80 per cent body surface area burns.

Patient H.V., who had worn protective clothing, was conscious and tremulous. Examination of the skin revealed erythema and blister formation over his legs, arms and back.

Patient E.S. (Fig. 2), with the longest exposure to the spray, was uncooperative, screaming in pain and convulsing. He was initially sedated with intravenous morphine sulphate, diazepam, and pentobarbital, followed by intubation and assisted respiration.

The third patient, D.G. (Fig. 3), with the largest burn surface area, suffered a respiratory arrest enroute by ambulance and was maintained on a resuscitator until intubation could be performed in the emergency room. He was unresponsive except to deep, painful stimulation. Initial arterial blood gases showed a serum bicarbonate of 12.5 mEq/l, and he was given 200 mEq of sodium bicarbonate intravenously.

All 3 men were resuscitated with the rapid infusion of lactated Ringer's

solution. Because of the inhalation of phenol fumes, and the resultant risk of chemical pneumonitis, methylprednisolone sodium succinate (Solu-Medrol$^{(R)}$), 30 mg/kg body weight, was given intravenously to each man. Prophylactic penicillin and tetanus toxoid were also administered.

D.G. and E.S. showed evidence of seizure activity and were kept sedated with diazepam and pentobarbital throughout the first 24 hours. These men also sustained burns to the eyes, and fluorescein staining revealed ulceration of the cornea and conjunctiva in D.G. There was similar damage in E.S., with the exception of the central cornea which had been protected by contact lenses. This epithelial loss was superficial and healed in several days without scarring.

Approximately 30 minutes after admission, all 3 patients developed gross hematuria, hemoglobinuria, and oliguria. The diuretics mannitol and furosemide were used to maintain urine output, and sodium bicarbonate was given to alkalinize the urine and to correct the acidosis in D.G. and E.S. Approximately 5 liters of fluid were given to these 2 in the first 8 hours, with 3.5 liters of urine from D.G., and 1 liter from E.S.

The chemical agents responsible for the injury were initially not known; however, irritating fumes were given off from the skin and hair and copious lavage with normal saline was performed. At 2 hours post burn, it was learned that potassium hydroxide was a constituent, and 2 per cent acetic acid soaks were applied and rinsed off. This resulted in a color change in the patients' skin, from a bright, deep red to yellow, as one of the byproducts of the chemical reaction was subsequently learned to be a phenol compound with pH indicator properties. At 3 hours post burn, it was learned that phenol was the main agent responsible for the injury, and propylene glycol, the only glycol product available, was applied topically and repeatedly wiped or washed away.

D.G., with the greatest surface area burn, had a rectal temperature of 34.3°C 2 hours after admission. His temperature remained approximately 35°C throughout

the night, then gradually rose to 37°C by 18 hours after admission. H.V. and E.S. had temperatures of approximately 36°C for 12 hours and these gradually rose. By 24 hours after admission, all 3 men had temperatures of approximately 37.5°C and this level continued throughout their hospital stay. The low temperatures were probably due to exposure and the frequent application of moist soaks. Also, there may have been some direct effect on the temperature regulating center by the phenol absorbed into the bloodstream (Goodman and Gilman, 1975).

D.G. was acidotic for approximately 18 hours after admission (Bennett et al., 195(The arterial blood pH reached a low of 7.25 2½ hours after admission, and the serum bicarbonate, after the initial low of 12.5 mEq/l, remained at 18-19 mEq/l, and the $_pCO_2$ on the respirator approximately 35 mm Hg for 18 hours. A total of 700 mEq of sodium bicarbonate was given intravenously in intermittent doses throughout the first 10 hours (Eggleston and Feierabend, 1975). At the same time, the serum potassium level fell from an initial 4.1 mEq/l to 2.4 mEq/l 6 hours after admission. A total of 150 mEq of potassium chloride was added to the intravenous fluids throughout the first 14 hours, and following this, the serum potassium returned to normal levels (Fig. 4). The urinary electrolytes were sodium 103 mEq/l and potassium 8 mEq/l at 10 hours after admission, and sodium 70 mEq/l and potassium 52 mEq/l at 22 hours. A similar trend to acidosis and low potassium was observed in E.S. After 24 hours, the serum potassium reached normal levels in all patients and no further supplements of intravenous potassium chloride were given. On admission, nasogastric tubes were passed into E.S. and D.G. The volume aspirated by continuous suction was less than 400 ml each in the first 16 hours.

The low serum potassium in the presence of acidosis, hemolysis, tissue destruction and modest potassium replacement remains unexplained. The frequent application of isotonic saline soaks to the large surface area burn may have removed some potassium by diffusion. A similar condition has been observed in our unit when patients with very large surface area burn are immersed in a hydrotherapy

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tank for more than 30 minutes daily and the solution in the tank is hypo-osmolar. A gradual fall in serum sodium ion results from diffusion into the tank water.

Twelve hours after admission, the intubated patients were examined with the fiberoptic bronchoscope, which revealed erythema and patchy ulceration of the trachea down to the carina.

Burn Unit Treatment

Eighteen hours after admission, with their condition relatively stable, the 3 patients were transferred to the burn unit at the Santa Clara Valley Medical Center. There polyethylene glycol 400 was used to swab down the skin. Application of this undiluted solvent caused an unexpected exothermic reaction. Irritating fumes were still being given off from the skin and hair, so the hair was shaved off.

Blood phenol level tests were not available, but urinary phenol levels on a 12-hour collection, starting about 28 hours after injury, were as follows: E.S., 157 mg/100 ml; D.G., 106 mg/100 ml; and H.V., 55.5 mg/100 ml. These levels fell rapidly until, by the fourth day, values were: E.S., 31 mg/100 ml; D.G., 1.4 mg/100 ml.* The ratio of free to conjugated phenol at 34 hours post burn was 2 to 1.

Nasogastric tube feedings were begun after 36 hours. Nasal endotracheal intubation was maintained for E.S. and D.G. for 7 days, and they were gradually weaned from the respirator by intermittent mandatory ventilation. Pulmonary function studies showed a gradual return to normal.

Most of the burned areas were partial thickness; H.V. required debridement and skin grafting of the lower extremities, and E.S. of the right groin. Subsequent treatment consisted of daily hydrotherapy in dilute hypochlorite or iodophor solutions and topical antibacterial dressings with silver sulfadiazine then nitrofurazone (Furacin$^{(R)}$). The local treatment with respect to time is graphically illustrated in Fig. 5.

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* the Hine Laboratory

The initial oliguria was followed by a steady rise in serum creatinine and blood urea nitrogen in E.S., and to a lesser extent in D.G. (Fig. 6 and 7). Creatinine clearance was: E.S., 7 ml/min/1·73 m^2; D.G., 72 ml/min/1·73 m^2; and H.V., 95 ml/min/1·73m^2. These changes in E.S. and D.G. were attributed to a phenol-induced toxic nephropathy.

E.S. was maintained on a low protein diet via nasogastric tube for 3 days, with regular insulin as necessary. There were no problems with hyperkalemia, acidosis or fluid overload.

The hemoglobin levels in all 3 patients fell progressively throughout the first 10 days, and remained low for a month. Slight elevations occurred in H.V. and E.S. following blood transfusion prior to anesthesia for skin grafting (Fig. 8).

The reticulocyte counts in E.S. fell from 2.2 per cent (59 x $10^3/mm^3$) on the eleventh day to 0.5 per cent (19 x $10^3/mm^3$) 1 week later. It did not rise above 2.2 per cent for several months, whereas D.G. reached 5 per cent, and H.V. 3 per cent 2 weeks following the injury. There was also a progressive pancytopenia throughout the first week (Fig. 9), and platelet counts fell about the fourth day (E.S., 134,000/mm^3; D.G., 110,000/mm^3; H.V., 170,000/mm^3), and then rose steadily. Iliac crest bone marrow aspirated from E.S. on day 17 was reported as normocellular.

All 3 patients were discharged in less than 1 month. Follow-up in clinic 3 months later revealed a return to normal of the central nervous system, as well as pulmonary, renal and hemopoietic function, and only patchy areas of hyper and hypopigmentation remain on the skin.

DISCUSSION

Phenol (carbolic acid or hydroxbenzene) is a weak acid, pKa 9.98. It exists in pure form as hygroscopic, colorless, rhombic crystals, with a melting point of 43°C and a boiling point of 182°C. It is very soluble in ethyl alcohol, glycerol,

vegetable oils and in fats, and poorly soluble in hydrocarbon mineral oils (Goodman and Geiger, 1935). Its solubility in water is 9.3 gms per 100 gms at 20°C. It has a low fatal dosage; LD_{50} is estimated to be 8-15 gms in adults.

There are two main classes of toxic action: chemical and physical. The physical effects may result from adsorption on cell surface layers, solution in lipids, and coagulation of cell protein (Ferguson, 1939; Cooper, 1912). Physical toxic action is a function of the intrinsic toxicity of the substance and its phase distribution equilibrium, that is, the equilibrium between the concentration of the active substance in the circumambient medium or in the body fluid of the organism, and in the concentration at the surface layer, or in the particular cells or structures that are directly affected by it (the biophase). The primary effect is reversible. Irreversible states are due to secondary processes. Allawala et al. proved that relative toxicity could be related to solubility, which provided a measure of the chemical potential (Allawala et al., 1954). Ferguson showed that at equilibrium, the chemical or thermodynamic potential of the drug, not the concentration, was the same in all phases (Ferguson, 1939). With chemical toxicity the effect is determined not by an equilibrium concentration, but by the number of molecules reaching the site of toxic action.

Giles et al. described the form of the adsorption isotherm for monohydric phenol on a polar substrate (Giles et al., 1960). There was moderate intermolecular attraction between the hydrophobic benzene ring residue causing vertical packing in regular array on the adsorbed layer. There was also strong competition for sites on a polar substrate from solvent molecules, especially polar ones such as water or ethanol. The force of attraction is localized in the hydroxyl group, and phenol is adsorbed as a single undissociated unit and not as a micelle.

The bacteriocidal action of phenol is due to bacterial cell membrane damage as well as intracellular absorption and inactivation of enzyme systems, a

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combination of physical and chemical toxicity. Concentrated solutions may cause coagulation of the cell membrane or cytoplasm and delay intracellular absorpt (Hugo, 1967).

Metabolism

Many phenol products occur naturally in plants and animals. Phenol is a normal constituent of human urine (Piotrowski, 1971; Walkley et al., 1961). The body has 3 general mechanisms for the disposal of phenol: conjugation, oxidation, and excretion (Capel et al., 1972).

All the phenols ingested in the diet are conjugated in the intestine to phenyl sulfates and glucuronides before absorption into the portal bloodstream. Conjugation of free phenol also occurs in the liver, kidney and red blood cells.

Powell et al. have shown that in the rat phenol is not concentrated in the liver, and when the liver and gastrointestinal tract are removed, the phenol detoxication products are still formed and excreted in the urine (Powell et al., 1974). Deichmann found phenol concentrated in the liver and central nervous system in rabbits after phenol poisoning (Deichmann, 1944). He also showed that lethal doses decreased the excretion of conjugated phenol from the damaged kidneys and increased the metabolic oxidation.

Oxidation

In experimental animals, 25 to 50 per cent of sublethal doses of phenol are oxidized to carbon dioxide and water, while small quantities are oxidized to catechol and quinol. The catechol and quinol are then conjugated and excreted in the urine, or broken down further to dark-colored substances which are then excreted.

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Excretion

Initially, the free phenol circulates and is excreted in the urine (Fig. 10). The severity of the systemic toxicity is proportional to the concentration of

free phenol (Deichmann and Keplinger, 1963). If the circulating level of free phenol is high, organ damage occurs which impairs conjugation. This results in the death of the animal, or increased oxidation and survival. Traces of free and conjugated phenol are excreted in the feces and traces of free phenol are excreted in the sweat and exhaled air (Deichmann, 1944).

Phenol Toxicity (Fig. 11)

Phenol is readily absorbed through the intact or abraded skin, from subcutaneous tissue, and from the mucous membrane of the gastrointestinal tract (Ruedemann and Deichmann, 1953; Deichmann and Witherup, 1944). Phenol vapor is rapidly absorbed from the lungs into the circulation, the rate of absorption being proportional to the concentration of phenol vapor (Piotrowski, 1971). However, in the skin, the rate of absorption is proportional to the surface area involved and the duration of contact, rather than to the phenol concentration (Deichmann and Witherup, 1944).

The local necrosis is proportional to the concentration. Therefore, with concentrated solutions the intense necrosis in the epidermis and upper dermis forms an avascular barrier which delays further absorption of the drug. Dilution with a solvent such as water or alcohol may increase the amount of phenol absorbed.

Skin

Depigmentation (leucoderma) is a complication of chemical face peeling and contact with phenolic compounds in industry. Patients with occupational leucoderma may develop progressive areas of depigmentation long after contact with the chemical compound has ceased (editorial, Lancet, 1974; Gellin et al., 1970). The mechanism is believed to involve competitive inhibition of the enzyme tyrosinase in the oxidation of tyrosine and dopa to melanin, and selective damage to the melanocytes (McGuire and Hendee, 1971; Kahn, 1970).

Central Nervous System

Phenol has a marked effect on the central nervous system, which is manifest

by initial stimulation resulting in increased reflex activity, tremor, myoclonic jerks, and convulsions. There is a very short time interval between the administration of phenol and the onset of convulsions. Angel and Rogers postulated that the phenolic radical may form hydrogen bonds with the amide group of proteins in the nerve cell membranes, causing an increase in permeability, resulting in hyperexcitability (Angel and Rogers, 1972). Deichmann showed the tremors became apparent in rabbits when the total phenol reached levels ranging from 6.2 to 12.6 mgs per cent in the blood (Deichmann and Witherup, 1944). Stimulation is followed by depression, and death results from respiratory failure. In man, little preliminary central stimulation is observed and the prominent effects are those of central nervous depression (Goodman and Gilman, 1975). Two of the 3 patients in this incident showed C.N.S. stimulation with tremor and convulsions, and one severe respiratory depression requiring intubation and respiratory assistance.

Peripheral Nervous System

Increasing concentrations of phenol solutions initially inactivate and then destroy the nerve fiber endings for pain, touch, and temperature. As a result, phenol burns are hypo or anesthetic, and dilute phenol solutions are used as antipruritics (Deichmann, 1949).

Cardiovascular System

Toxic doses of phenol cause an initial rise in blood pressure, followed by a marked fall, due mainly to central vasomotor depression causing loss of vasoconstrictor tone, but also due to decreased cardiac output from a direct effect on the myocardium (Gleason et al., 1963). Tachycardia occurs early, followed by bradycardia and arrhythmias (Deichmann and Keplinger, 1963; von Oettingen, 1949). Phenol resembles the salicylates in producing a metabolic acidosis, as well as a powerful antipyretic effect from direct action on the hypothalamus, which can cause hypothermia.

Red Blood Cells

Chan et al. showed that lysol, a proprietary preparation containing 50 per cent cresol, with similar toxic effects to phenol (Deichmann, 1944; Deichmann and Keplinger, 1963), produced methemoglobin formation, Heinz bodies and a decrease in red cell glutathione (GSH) levels (Chan et al., 1971). Heinz bodies are rounded globules of denatured globin in the red cell or free in the serum. They are the result of the action of oxidants on the hemoglobin molecule, frequently from those substances with a benzene ring nucleus. Reduced glutathione (GSH) protects hemoglobin from irreversible oxidation. The effects were also greater in red cells deficient in glucose 6 phosphate dehydrogenase (G6PD), indicating an intact hexose monophosphate shunt has a protective effect on the red cell. Phenol may also cause hemolysis by its action on the red cell membrane. Fisher showed that phenol poisoning also damaged erythropoiesis in the marrow (Fisher, 1955). In these cases, E.S. had a low reticulocyte response for two weeks following the injury.

Kidneys

By the time the phenol is excreted in the urine, the other organs have already been damaged (Goodman and Geiger, 1935). Renal damage is caused by:

(1) The excretion of unconjugated phenol which damages the glomeruli and the tubules (Georgiev et al., 1972).

(2) Hypovolemic shock leading to renal ischemia.

(3) Hemolysis resulting from the precipitation of hemoglobin in the glomeruli and the tubules forming obstructive casts.

Liver

Unconjugated phenol causes central lobular necrosis which may be reflected in elevated serum bilirubin levels. This damage limits the ability of the liver to conjugate, and as a result, the body oxidizes more of the free phenol. Powell et al. showed that in rats orally ingested phenols were conjugated in the intestine

before absorption into the bloodstream, and intravenous phenol given to hepatectomised rats was conjugated and excreted in similar quantities to intact controls, indicating the importance of sites other than the liver for conjugation (Powell et al., 1974).

PRECAUTIONS AND TREATMENT

Those who use phenol should be aware of the toxic effects, methods of preventing exposure, and the first aid treatment in the event of an accident.

Prevention

In industry, it is important to educate those who handle phenol during production, as well as the rescue workers, first aid technicians and plant physicians who might handle the toxic materials or treat the injured in the event of an accident.

The design of the plant should include adequate ventilation, safety showers, alternate escape routes, an automatic deluge system in areas of high risk, and effective methods of waste disposal without environmental pollution.

At all times, in designated areas of risk, the workmen should wear protective clothing which cannot be dissolved by phenol. This should include protective headgear with a face shield and flap to cover the back of the neck, protective gloves, and above-ankle boots worn beneath the protective trousers. In the case of H.V., the phenol liquid ran off the trousers into the ankle-length boots, resulting in full thickness burns of the ankle region (Fig. 1).

An adequate communications system should be installed to notify the administrative center of the occurrence of an accident, and rescue workers, rescue gear and first aid equipment should be rapidly available at the site. Following this accident, safety equipment lockers were installed with self-contained breathing gear, protective clothing, explosion-proof flashlights, and communication bullhorns for the rescue workers, as well as stretchers, blankets and first aid materials. These lockers are kept fastened but not locked, and opening the door activates a rotating red

beacon on the roof. The lockers are designed to be transported by a forklift vehicle to the area of the plant where they are needed.

First Aid

The first aim is to prevent further contact with the phenol, and the second is to prevent absorption. Once phenol is absorbed, the toxic effects cannot be prevented. Experiments in animals show that absorption of phenol, not concentrated enough to cause local necrosis, can result in systemic toxicity (Deichmann, 1949).

As soon as possible, the victim should be removed from the site of the accident where phenol liquid and vapor may still be present. All clothing should be removed, and the phenol should be washed from the body surface to prevent local necrosis and systemic absorption. Water should only be used if available at high flow rates, such as a safety shower or an industrial deluge system. If water is not available, or after deluge, the skin should be swabbed with a suitable solvent. Solvents in order of preference are polyethylene glycol, propylene glycol, glycerol, vegetable oil, and soap and water (Deichmann and Witherup, 1944).

The phenol may be trapped in the hair of the scalp and face, and this hair should be removed as soon as possible. Respiratory support should be started with high oxygen flows by mask or resuscitator. Respiratory failure may occur early and positive pressure ventilation by mask may be necessary until the patient can be intubated.

Hospital

The patient should be transported to the nearest hospital emergency room as soon as possible. Intubation and positive pressure ventilation with a volume control respirator may be necessary if central nervous system depression is present. Intravenous fluids should be started at a high infusion rate in order to administer drugs, to prevent shock, and to promote urine output. Hemolysis and the resulting hemoglobinuria may be severe, and diuretics, such as mannitol, may be required (Cirksena et al., 1964).

Sodium bicarbonate should be given to raise the pH of the urine and decrease precipitation of hemoglobin in the renal tubules. It is also indicated for the metabolic acidosis which is a result of the large burn surface area plus the direct effect of the phenol (Eggleston and Feierabend, 1975; Bennet et al., 1950). The temperature should be monitored and external warming may be necessary if hypothermia occurs as a result of the direct effect of phenol on the hypothalamic temperature control center, as well as the cooling effect of the fluid irrigation of the body surface.

Cardiac monitoring should be performed, but EKG tracings are difficult to obtain in the presence of muscle tremors.

Potassium levels in serum and urine should be closely monitored and, if low, potassium should be given intravenously to maintain normal serum levels.

Digitalization may be contraindicated in the presence of arrhythmias and potassium ion changes. Dopamine hydrochloride appears to be the preferred drug for decreased cardiac output, because of its inotropic effect and increased renal blood flow.

If there are signs of muscle irritability from C.N.S. stimulation, intravenous anticonvulsants such as diazepam or barbiturates should be given to prevent seizures, in which case respiratory assistance would then be required.

High urine flows should be maintained to remove the hemoglobin and the free unconjugated phenol from the circulation, as it is the unconjugated phenol which is responsible for organ damage. If acute renal failure occurs, fluid intake and drug dosages will have to be adjusted with appropriate measures to prevent hyperkalemia and acidosis.

While supportive measures are being carried out, continued irrigation and swabbing with solvents should continue. The swabbing should be vigorous enough to remove the epidermal slough with its phenol-protein complex. This treatment should be performed in a well-ventilated area, as the fumes are very irritating to the attendants.

Blood tests should include hematocrit and white cell count, with serum electrolytes, serum creatinine and urea nitrogen, arterial blood gases and samples of blood and urine for phenol levels.

Follow-up treatment includes care of the burn wounds, with monitoring of the depressed blood cells, and transfusion of blood as necessary.

Solvents

Contraindicated solvents. Gentle swabbing with water should not be performed, as this will dilute the phenol and increase the amount absorbed (Brown et al., 1960; Conning and Hayes, 1970). Mineral oil is a poor solvent for phenol, and hexachlorophene is a chlorinated phenol (3, 4, 6 - trichlorophenol) which may itself be absorbed and result in central nervous system toxicity (Kimbrough, 1973). Alcohol is a good solvent for phenol, but may be rapidly absorbed, especially through injured skin, carrying with it the dissolved phenol (Roberts and Anderson, 1975; Deichmann and Witherup, 1944). Also, if there is a large burn surface area, the amount of alcohol absorbed could be toxic. The use of methyl alcohol as recommended by Brown (Brown et al., 1975) would have a higher risk than ethyl alcohol (Scheuplein and Blank, 1973).

Preferred solvents. The polyethylene glycols (PEG) of average molecular weight 300 and 400 (PEG 300 and PEG 400), are viscous, colorless liquids. They readily mix with water, giving off some heat when diluted. These glycols are good solvents for phenols and are nonirritating to skin, mucous membranes, conjunctiva and cornea (Conning and Hayes, 1970).

For both convenience and effective treatment, wash liquids of low viscosity are needed. The flow times of various fluids at room temperature were measured and the relative viscosity with respect to water was recorded (Table 2). A PEG water mixture 1:1 is about twice as viscous as water alone. Glycerol, by comparison, is about 160 times more viscous than water.

On contact with human skin, polyethylene glycols, 300 and 400, produce a

heat sensation caused by the moisture of the skin diluting the glycol. This phenomenon is usually unexpected, and may lead to additional thermal injury to the skin. The magnitude of heat evolution is dependent on the diluent content of the polyethylene glycol. In a series of experiments in which equal volumes of PEG 300 and 400 containing varying amounts of ethanol and of water were mixed with equal volumes of water, the resulting temperature rise above ambient was measured. The data in Table 3 and Fig. 12 indicates that a 50 per cent solution with PEG 400 in water generates negligible heat on further dilution, and no heat sensation could be noticed when washing human skin with it. In contrast, if 70 or 95 per cent ethanol is used as the diluent, substantially more heat is generated on further dilution with water.

The solubility of phenol in PEG 300 or 400 decreases with increasing water content. A semi-quantitative value for this relationship was obtained by determining the volume of solvent required to dissolve a given weight of phenol. The data in Table 4 suggests that an aqueous solution of 50 per cent PEG 300 or 400 has ample phenol solvent power to serve as an effective wash liquid. No heat reaction was noticed when phenol was mixed with this PEG solution, and both visual observations and odor detection confirmed its effectiveness. This fluid has low viscosity, spreads readily on the skin, and can be removed easily by subsequent washing with water. Ethanolic PEG solutions have greater phenol solvent power, but are not recommended as a wash liquid since they are flammable, possess an intoxicating odor, and are readily absorbed and therefore could cause problems with extensive washings.

Acknowledgement

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Table 1

FATAL CASES OF PHENOL POISONING

1. Abrahams, 1900	7-day-old male infant touched in right groin by nursemaid with phenol on thumb and index finger. Died in 10 hours.
2. Turtle and Dolan, 1922	30-year-old male broke bottle of crude carbolic acid in pocket and burned left buttock, thigh, and genitals. Died one week later with anuria.
3. Miller, 1942	18-year-old male with ringworm had housekeeper apply mixture of phenol and camphor to shoulders and trunk; mixture diluted with water because of burning sensation. Died in 15 minutes.
4. Cronin and Brauer, 1949	10-year-old male with 25 to 30 per cent partial thickness kerosene burns dressed with foille (R), containing approximately 2 per cent phenol in corn oil. Died in coma after 3 days.
5. Griffiths, 1973	23-year-old male, without protective clothing, dismantled industrial filter and was sprayed with phenol across thighs. Died in 10 minutes.

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Table 2

FLUIDITY OF WASH LIQUIDS*

Liquid	Relative Viscosity
Water	1
Glycerol	158
PEG-300 (J.T. Baker)	12.5
PEG-400 (GAF 400)	16.0
PEG-400:H_2O(1:1)	1.9
PEG-400:EtOH(2:1)	3.5
PEG-400:EtOH(1:1)	1.8
PEG-300:EtOH(2:1)	2.5

* Laboratory analysis by Klaus Dahl, Ph.D.

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Table 3

HEAT OF DILUTION DATA FOR POLYETHYLENE GLYCOL BASED WASH LIQUIDS*

Diluent Used for Component A	Composition of Component A				Composition of Component B	Temperature Rise Resulting from Mixing of Equal Volumes† of A & B °C	
	PEG	EtOH	H_2O	Σ % diluent	H_2O	$\Delta T_{PEG-300}$	$\Delta T_{PEG-400}$
Water	100	-	0	0	100		10
	90	-	10	10	100		6.4
	80	-	20	20	100		5
	70	-	30	30	100		2.9
	50	-	50	50	100		0.6
70% Ethanol	86	10	4	14	100	8.6	9.8
	71	20	9	29	100	7.1	7.9
	57	30	13	43	100	6.3	7.0
	29	50	21	71	100	4.7	4.1
95% Ethanol	89.5	10	0.5	10.5	100	10.6	11.2
	79	20	1	21	100	9.2	10
	68	30	2	32	100	8.5	9.5
	47	50	3	53	100	7.3	7.4

† 10 ml

* Laboratory analysis by Klaus Dahl, Ph.D.

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Table 4

SOLUBILITY DATA FOR PHENOL IN POLYETHYLENE GLYCOL BASED WASH LIQUIDS**

Solvent Composition, %			Volume of Solvent (ml) Required to Dissolve 0.5 g Phenol at 25°C*	
PEG	EtOH	H_2O	PEG-300 System	PEG-400 System
43	5	52	1.5	1.6
36	10	54	2.0	2.4
29	15	56	2.2	2.2
14	25	61	2.6	3.0
50	0	50	-	1.5
45	0	55	-	2.0
40	0	60	-	3.0
35	0	65	-	3.0
25	0	75	-	5.1

* Solubility of phenol in PEG-300 is 0.5g/0.35 ml, and for PEG-400 is 0.5g/0.80 ml.

** Laboratory analysis by Klaus Dahl, Ph.D.

LEGENDS

Figure 1: Patient H.V. Full thickness burns, ankle region.

Figure 2: Patient E.S. Partial thickness burns to back.

Figure 3: Patient D.G. Partial thickness burns.

Figure 4: Patient D.G. Acidosis and hypokalemia in first 18 hours.

Figure 5: Local treatment of burns.

Figure 6: Serum creatinine in the three patients.

Figure 7: Blood urea nitrogen in the three patients.

Figure 8: Hemoglobin in the three patients; arrows indicate blood transfusion.

Figure 9: White cell counts in the three patients.

Figure 10: Phenol metabolism.

Figure 11: Phenol toxicity.

Figure 12: Heat of dilution data for PEG-400 based wash liquids.

Figure 1

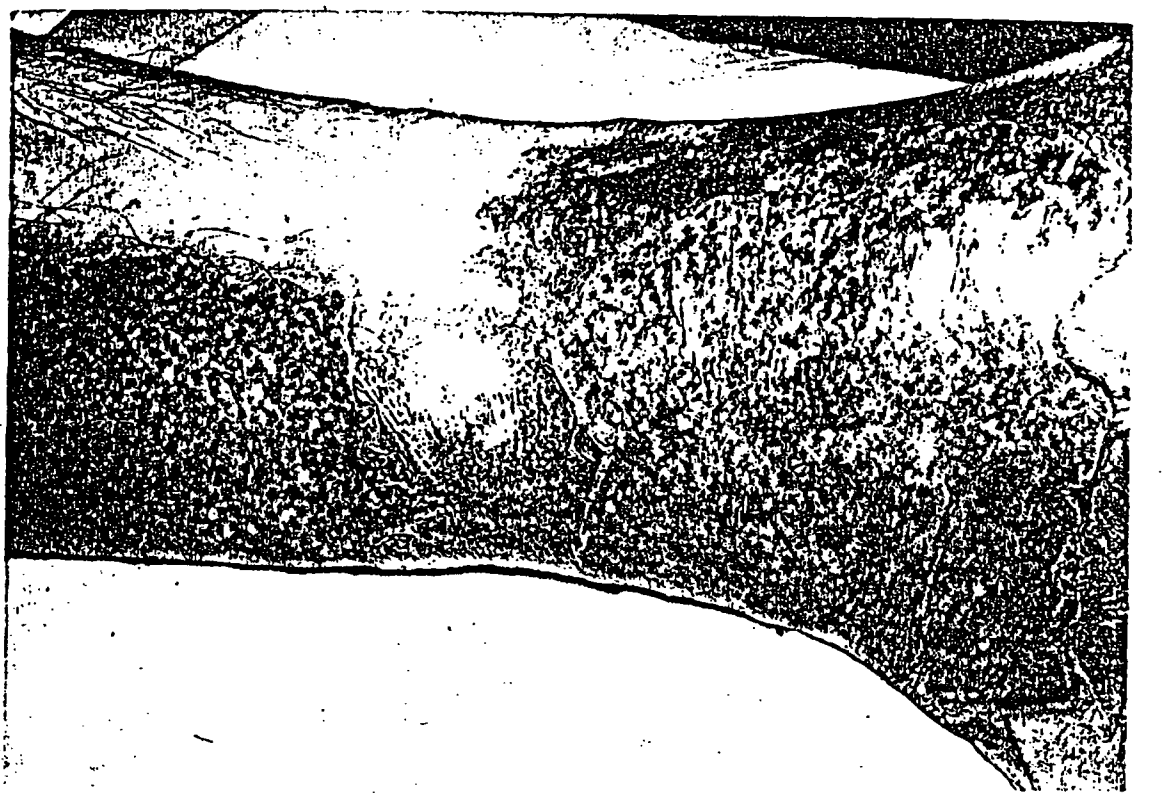

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Figure 2

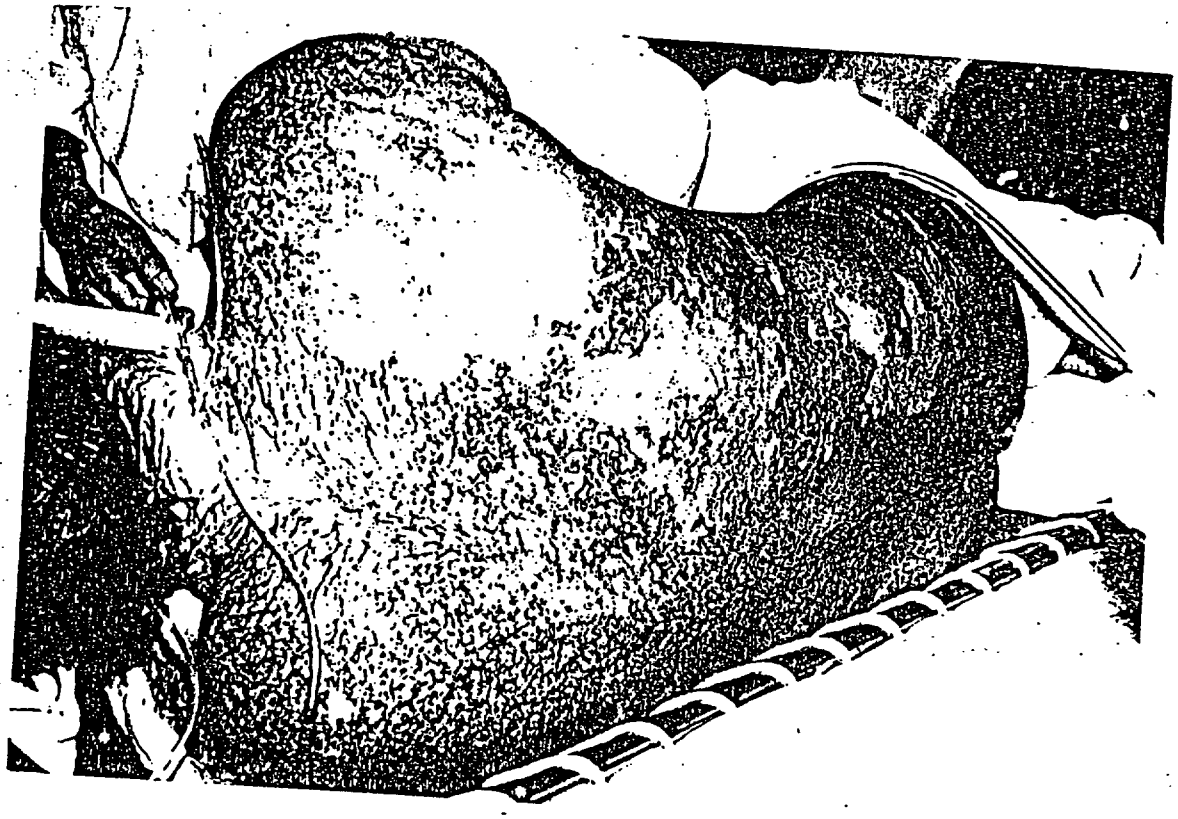

Figure 3

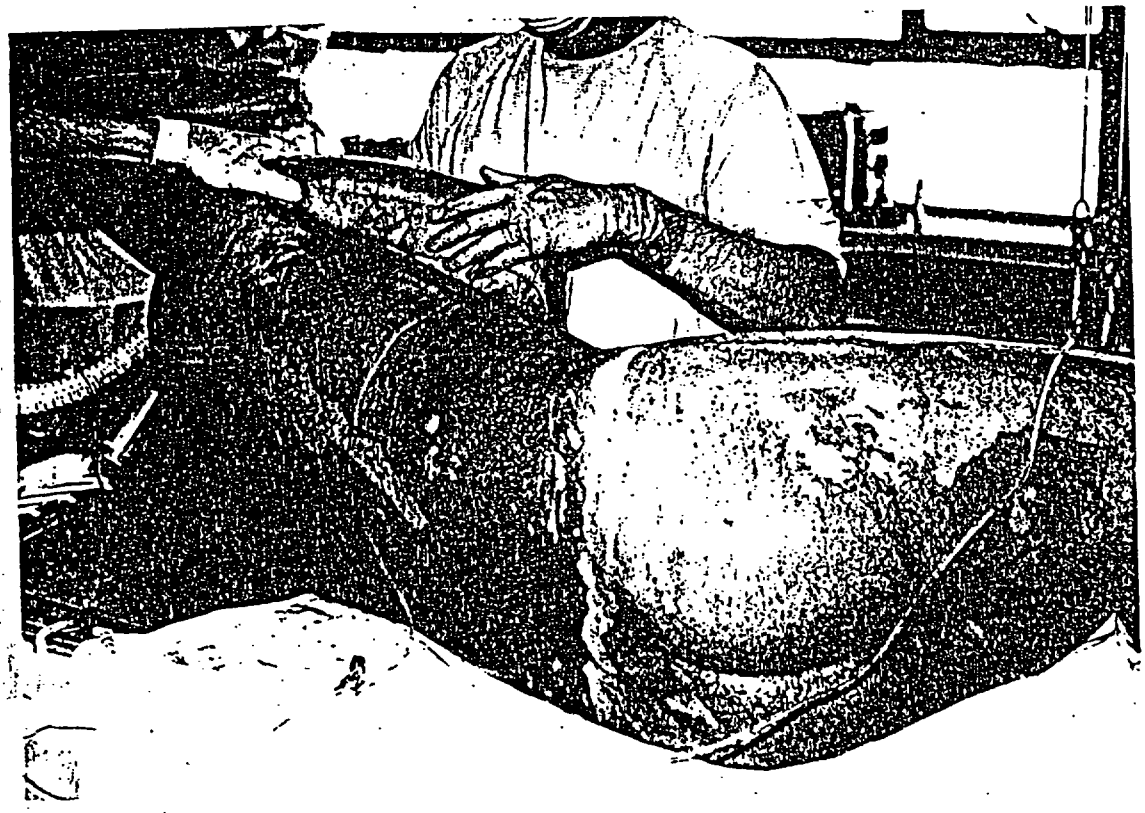

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Figure 4

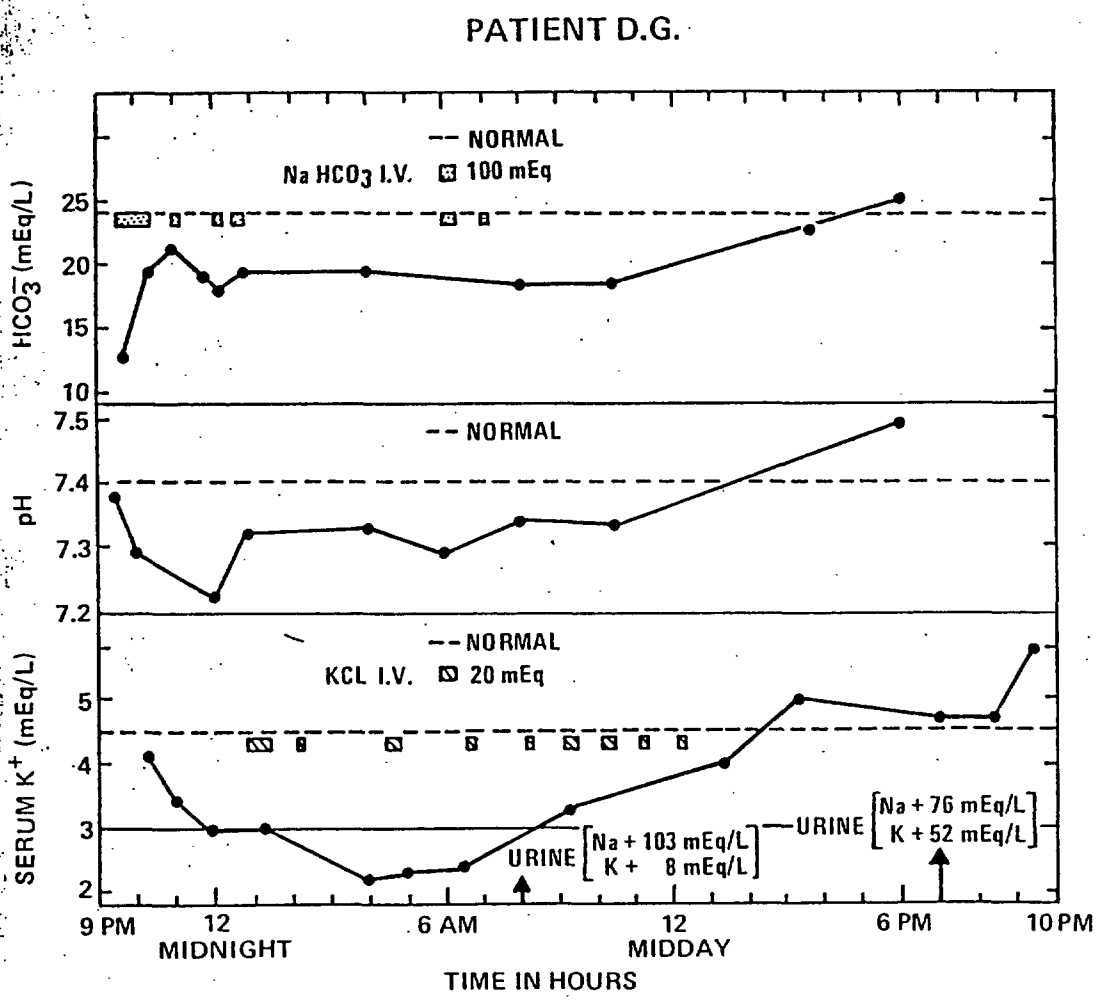
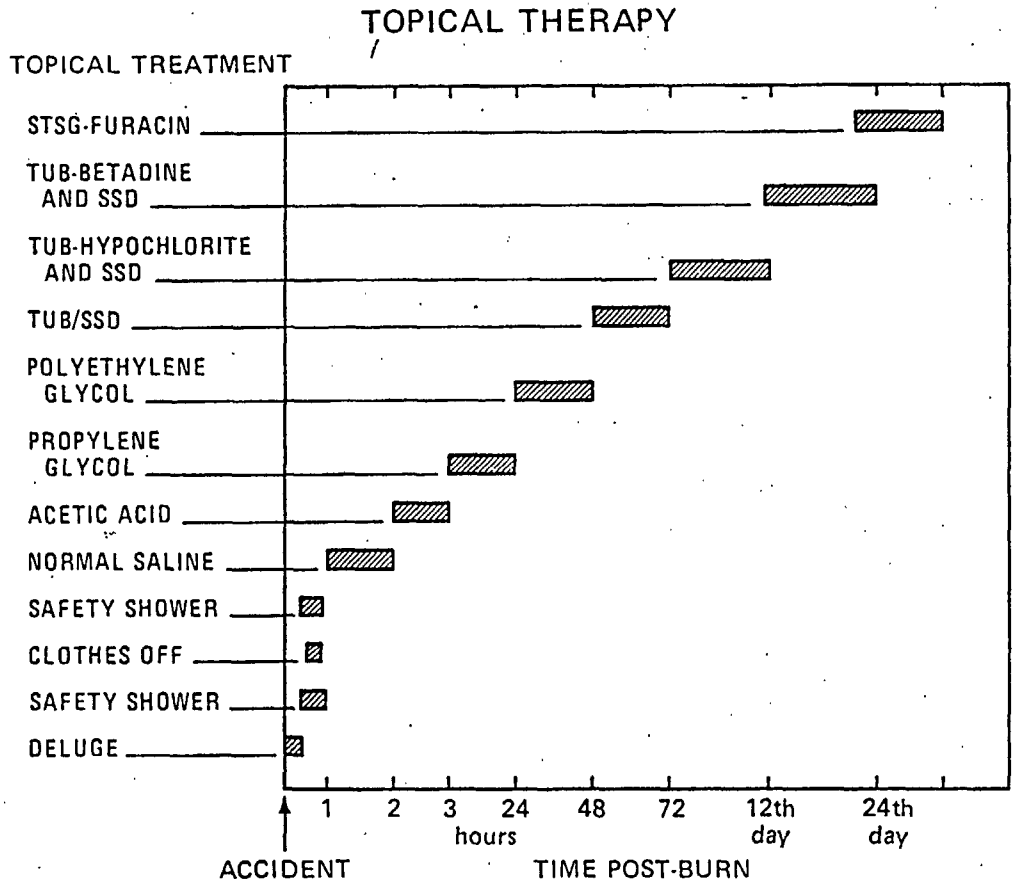

PATIENT D.G.
-- NORMAL
Na HCO3 I.V. 100 mEq
HCO3- (mEq/L)
25
20
15
10
7.5
7.4
7.3
7.2
pH
-- NORMAL
-- NORMAL
KCL I.V. 20 mEq
SERUM K+ (mEq/L)
5
4
3
2
URINE [Na + 103 mEq/L, K + 8 mEq/L]
URINE [Na + 76 mEq/L, K + 52 mEq/L]
9 PM
12
MIDNIGHT
6 AM
12
MIDDAY
6 PM
10 PM
TIME IN HOURS

Figure 5



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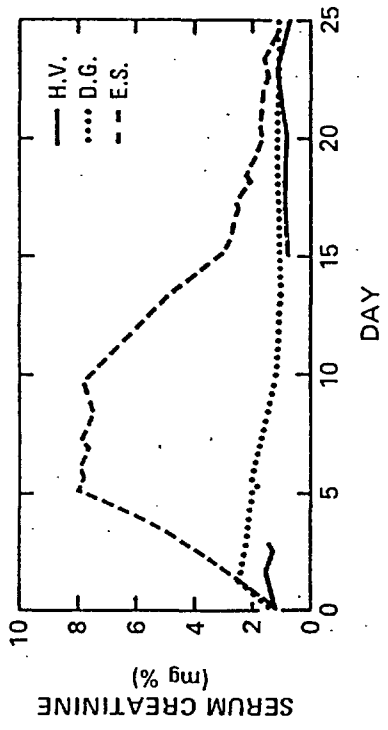


Figure 6

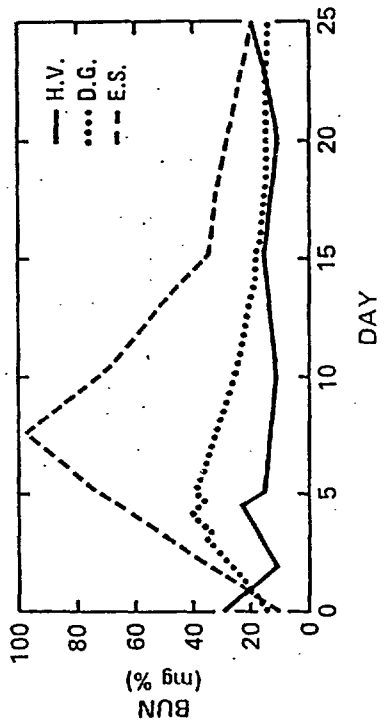


Figure 7

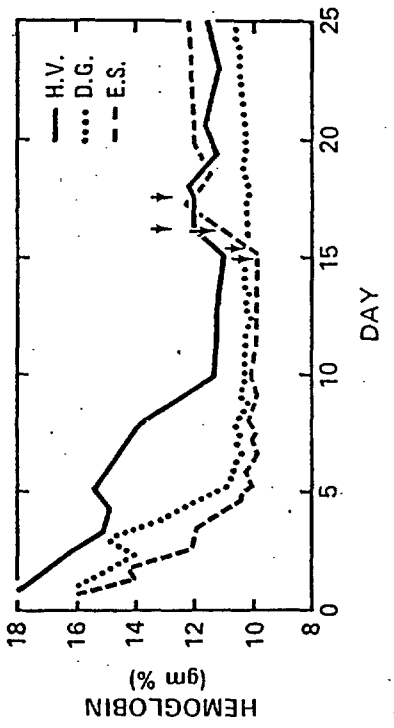


Figure 8

Figure 9

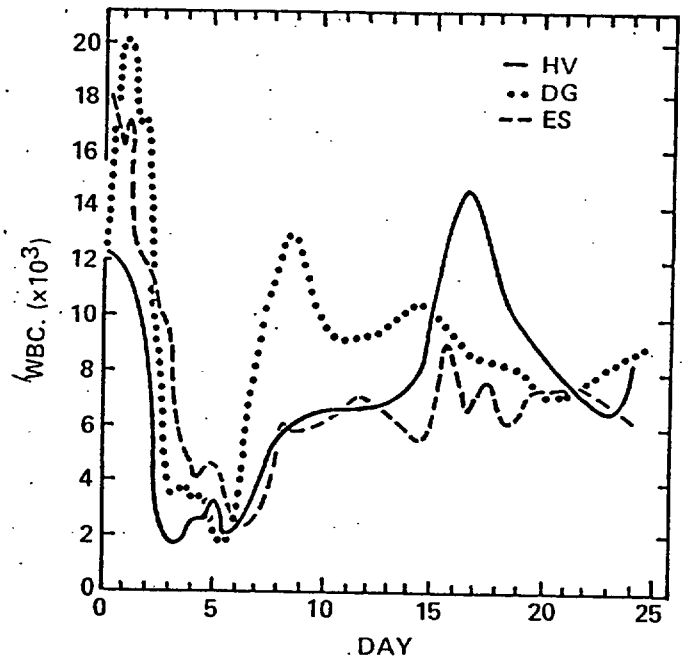
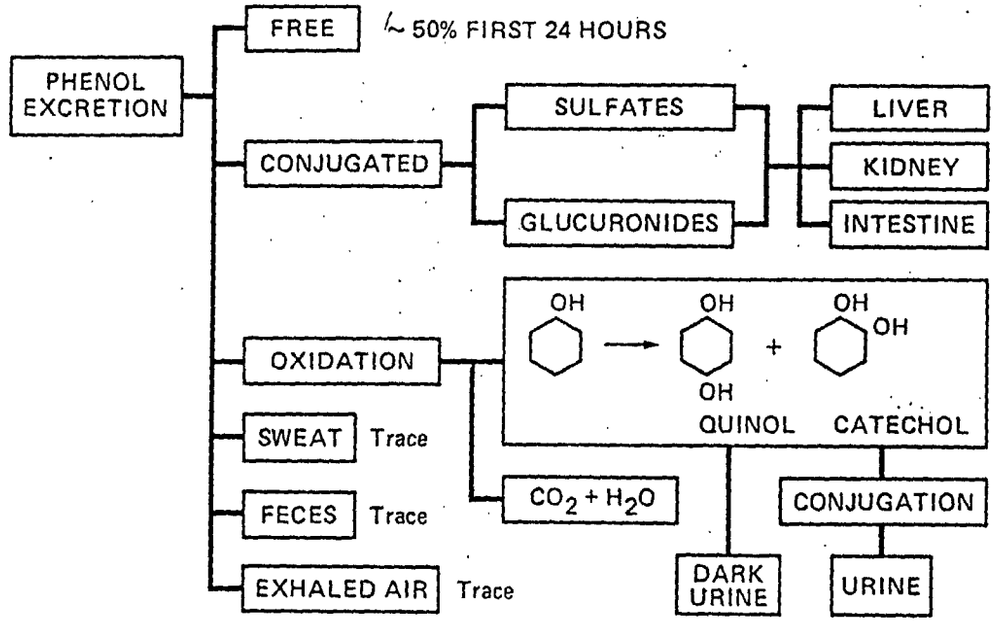


Figure 10



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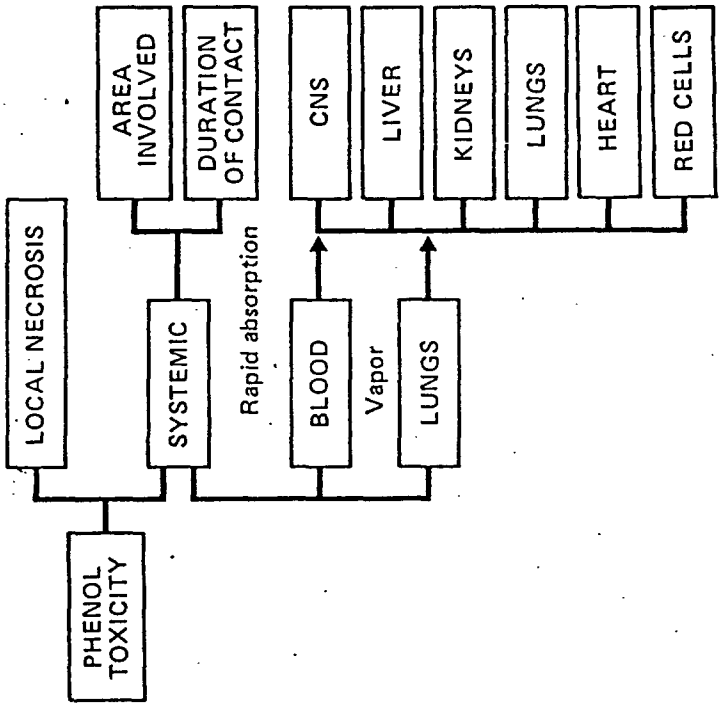
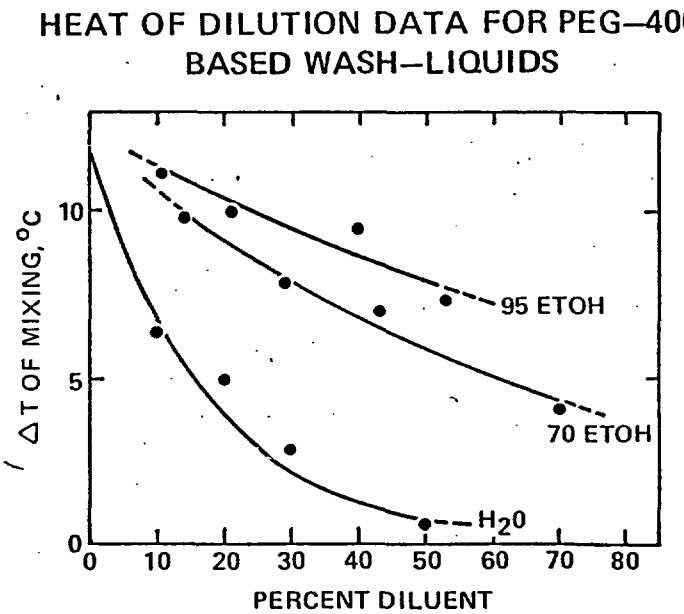

PHENOL TOXICITY
LOCAL NECROSIS
SYSTEMIC
AREA INVOLVED
DURATION OF CONTACT
Rapid absorption
BLOOD
LUNGS
Vapor
CNS
LIVER
KIDNEYS
LUNGS
HEART
RED CELLS

Figure 11

Figure 12


HEAT OF DILUTION DATA FOR PEG–400
BASED WASH–LIQUIDS
Δ T OF MIXING, °C
10
5
0
0
10
20
30
40
50
60
70
80
PERCENT DILUENT
95 ETOH
70 ETOH
H20

REFERENCES

ABRAHAMS R. (1900) A fatal case of carbolic acid poisoning in an infant, due to absorption by the skin. Pediatrics 9, 241-242.

ALLAWALA N.A. and RIEGELMAN S. (1954) Phenol coefficients and the Ferguson principle. J. Am. Pharm. Ass. (Sci. ed.) 43, 93-97.

ANGEL A. and ROGERS K.J. (1972) An analysis of the convulsant activity of substituted benzenes in the mouse. Toxicol. Appl. Pharmacol. 21, 214-229.

BENNETT I.L., JAMES D.F. and GOLDEN A. (1950) Severe acidosis due to phenol poisoning. Report of two cases. Ann. Int. Med. 32, 324-327.

BROWN A.M., KAPLAN L. and BROWN M.E. (1960) Cutaneous alterations induced by phenol. A histologic bio-assay. J. Int. Coll. Surg. 34, 602-612.

BROWN V.K.H., BOX V.L. and SIMPSON B.J. (1975) Decontamination procedures for skin exposed to phenolic substances. Arch. Enviorn. Health 30, 1-6.

CAPEL I.D., FRENCH M.R., MILLBURN P., SMITH R.L. and WILLIAMS R.T. (1972) Species variations in the metabolism of phenol. Proc. Biochem Soc. 127, 25p-26p.

CHAN T.K., MAK L.W. and NG R.P. (1971) Methemoglobinemia, Heinz bodies and acute massive intravascular hemolysis in lysol poisoning. Blood 38, 739-744.

CIRKSENA W.J., BASTIAN R.C., MALLOY J.P. and BARRY K.G. (1964) Use of mannitol in exogenous and endogenous intoxications. New Engl. J. Med. 270, 161-166.

CONNING D.M. and HAYES M.J. (1970) The dermal toxicity of phenol: an investiagation of the most effective first aid measures. Brit. J. Ind. Med. 27, 155-159.

COOPER E.A. (1912) On the reactions of phenol and meta-cresol to proteins. A contribution to our knowledge of the mechanism of disinfection. Biochem J. 6, 362-387.

CRONIN T.D. and BRAUER R.O. (1949) Death due to phenol contained in Foille. JAMA 139, 777-779.

DEICHMANN W.B. (1944) Phenol studies V. The distribution, detoxification and excretion of phenol in the mammalian body. Arch. Biochem. 3, 345-355.

DEICHMANN W.B. and WITHERUP S. (1944) Phenol studies VI. The acute and comparative toxicity of phenol and O-, M- and P- cresols for experimental animals. J. Pharmacol. Exptl. Therap. 80, 233-240.

DEICHMANN W.B. (1949) Local and systemic effects following skin contact with phenol. A review of the literature. J. Ind. Hyg. Toxicol. 31, 146-154.

DEICHMANN W.B. and KEPLINGER M.L. (1963) Industrial Hygiene and Toxicology, New York, Interscience, Patty F.A. (Ed.), p. 1363-1408.

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EDITORIAL (1974) Lancet 2, Oct. 1974, p. 879.

EGGLESTON F.C. and FEIERABEND T.C. (1975) The early acidosis of burns. Its relationship to extent of burn and management. Surgery 77, 641-647.

FERGUSON J. (1939) The use of chemical potentials as indices of toxicity. Proc. Roy. Soc. Med. 127B, 387-403.

FISHER B. (1955) The significance of Heinz bodies in anemias of obscure etiology. Amer. J. Med. Sci. 230, 143-146.

GELLIN G.A., POSSICK P.A. and PERONE V.B. (1970) Depigmentation from 4-tertiary butyl catechol - an experimental study. J. Invest. Derm. 55, 190-197.

GEROGIEV A., HADJEVA S.T. and VELISAROV A. (1972) Morphologic changes in kidneys after acute and chronic poisoning with phenol. Eksper. Med. Morf. 11, 80-84.

GILES C.H., MacEWAN T.H., NAKHWA S.N. and SMITH D. (1960) Studies in adsorption XI. A system of classification of solution adsorption isotherms and its use in diagnosis of adsorption mechanisms and in measurement of specific surface areas of solids. J. Chem. Soc. 3973-3993.

GLEASON M.N., GOSSELIN R.E. and HODGE H.C. (1963) Clinical Toxicology of Commercial Products, 2nd edition, Baltimore, Williams and Wilkins, p. 189-191.

GRIFFITHS G.J. (1973) Fatal acute poisoning by intradermal absorption of phenol. Med. Sci. Law 13, 46-48.

GOODMAN L. and GEIGER A.J. (1935) Therapy in carbolic acid poisoning, with special reference to the use of oil antidotes. Amer. J. Med. Sci. 190, 206-220.

GOODMAN L.S. and GILMAN A. (1975) The Pharmacological Basis of Therapeutics, 5th edition. New York, MacMillan, p. 990-991.

HUGO W.B. (1967) The mode of action of antibacterial agents. J. Appl. Bact. 30, 17-50.

KAHN G. (1970) Depigmentation caused by phenolic detergent germicides. Arch. Derm. 102, 177-187.

KIMBROUGH R.D. (1973) Review of recent evidence of toxic effects of hexachlorophene. Pediatrics 51, 391-394.

KOCH R. (1881) Ueber desinfection. Kaiserlichen Gesundheitsamte Mitteheilugen 1, 234-282.

LEMAIRE J. (1865) De L'acide Phenique de son Action sur les Vegetaux les Animaux les Ferments..., 2nd edition. Paris, Germer-Bailliere, p. 754.

LISTER, J. (1867) On a new method of treating compound fracture, abcess, etc. Lancet 1, 326-329, 357-359, 387-389, 507-509.

McGUIRE J. and HENDEE J. (1971) Biochemical basis for depigmentation of skin by phenolic germicides. J. Invest. Derm. 57, 256-261.

MERLISS R.R. (1972) Phenol marasmus. J. Occup. Med. 14, 55-56.

MILLER F.G. (1942) Poisoning by phenol. Canad. Med. Assoc. J. 46, 615-616.

PIOTROWSKI J.K. (1971) Evaluation of exposure to phenol: absorption of phenol vapour in the lungs and through the skin and excretion of phenol in urine. Brit. J. Industr. Med. 28, 172-178.

POWELL G.M., MILLER J.J., OLAVESEN A.H. and CURTIS C.G. (1974) Liver as major organ of phenol detoxication? Nature 252, 234-235.

RIDEAL S. and WALKER J.T.A. (1903) Standardization of disinfectants. J. Sanit. Inst. 24, 424-441.

ROBERTS M.S. and ANDERSON R.A. (1975) The percutaneous absorption of phenolic compounds: the effect of vehicles on the penetration of phenol. J. Pharm. Pharmac. 27, 599-605.

RUEDEMANN R. and DEICHMANN W.B. (1953) Blood phenol level after topical application of phenol-containing preparations. JAMA 152, 506-509.

SCHEUPLEIN R.J. and BLANK I.H. (1973) Mechanism of percutaneous absorption - IV. Penetration of nonelectrolytes (alcohols) from aqueous solutions and from pure liquids. J. Invest. Derm. 60, 286-296.

TURTLE W.R.M. and DOLAN T. (1922) A case of rapid and fatal absorption of carbolic acid through the skin. Lancet 2, 1273-1274.

VON OETTINGEN W.F. (1949) Phenol and its derivatives: the relation between their chemical constitution and their effect on the organism. Nat. Inst. Health Bull. No. 190, 3-56.

WALKLEY J.E., PAGNOTTO L.D. and ELKINS H.B. (1961) The measurement of phenol in urine as an index of benzene exposure. Amer. Indust. Hyg. Ass. J. 22, 362-367.

Fatal Acute Poisoning by Intradermal Absorption of Phenol

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Summary

THE HISTORY, post-mortem findings, and the qualitative chemical screening at autopsy are described in a case of acute intradermal phenol poisoning. The forensic estimate of phenol in the blood is described. A discussion of the immediate and later treatment of such a case is given.

Reading through the literature on acute phenol poisoning, little has been written on the subject of intradermal absorption of phenol. Standard forensic reference books (Camps, 1968; Simpson, 1966) have drawn attention to this mode of poisoning with reference to past and present literature. Recently a paper on the toxicity of phenol absorption in animals and an effective first-aid measure in treatment have been published (British Chemical Industries Safety Council of Chemical Industries Association, 1970; Conning and Hayes, 1970).

CASE REPORT

A young man of 23 years was employed as a technical assistant to a firm in Derbyshire engaged in processing asbestos using liquid phenol in the manufacture of brake linings. The dismantling of the plant following a report of any fault in its working is done by a skilled tradesman on the instruction of a works supervisor. Safety measures are laid down regarding the type of clothing to be worn and notices of danger to be displayed at the site of the dismantling. It is not the duty of the technician to dismantle the apparatus, his work is solely to inspect when the work of dismantling is complete.

Some 10 days prior to Wednesday, 5 October, 1971, the day of the fatal accident, the maintenance staff had been off work owing to an industrial dispute. On the morning of the accident the deceased had reason to visit the plant and found the flow of phenol was retarded. At 2 p.m. that day, in company with a trainee supervisor, they visited the pump house. The deceased started and stopped the pump. There was a clanking noise which became steadily less and finally disappeared on closing a valve. They then went to the filter and the deceased began dismantling the buckles of the filter, when suddenly a spray of liquid phenol covered his thighs—he was in a crouching position. At the time the deceased was not wearing the regulation safety clothing. He was wearing a warehouse coat, which came to his knees, ordinary trousers, socks, pants, and shoes. There were no 'display of danger' notices. The supervisor called at the inquest stated that the deceased would not be aware of the method employed to dismantle the buckles, and if he had there would be no spray of phenol. The trainee immediately splashed water over the thighs from a nearby tap and went to raise the alarm. In the meantime the deceased had removed his shoes, socks, and trousers. He was taken to the automatic safety shower platform. This shower did not work and from evidence at the inquest the water supply was in the off position. No explanation was given for this. He was finally taken to the ambulance room, some 100 yards from the scene of the accident. On the way there he collapsed. He was

Dr. G. J. Griffiths, Pathologist, High Peak Mortuary, Silverlands, Buxton, Derbyshire.

411538 0359

given oxygen and a heart stimulant. On the arrival of the works doctor he was dead. The time factor for this incident must have been at the minimum ten minutes from the occurrence of the spray to the time of death.

Post-mortem Findings

External Appearances

The patient was a male, of good physique, height 5 ft. 10 in. and weighing 11 stone. At 8½ in. below the right pubic ramus there was an area 12 in. long covering the front of the right thigh, extending to the outer side of the thigh, the front, and inner surface of the thigh. Likewise, 7 in. below the left ramus was an area 8 in. long on the inner side of the left thigh. The area on the right continued downwards to cover the front, outer, and inner side of the right leg and to some degree the inner surface of the left leg. These areas presented a dry greyish-brown corrosive appearance on the right thigh and the inner side of the left thigh, whilst that below the right thigh had a more reddish-white appearance, and to a lesser degree this was the same on the left leg. On the back of the left hand was an area measuring 2 in. by 1½ in., being red, with some degree of corrosion. It was estimated that the area involved was 25 per cent of the total skin surface. The rest of the body was not involved.

Internal Examination

SKULL AND BRAIN

Skull and scalp were intact. There was acute congestion of the brain and meninges, with scattered petechiae in the brain substance. The blood in the basal vessels was coagulated.

THORAX

Oedema of the mucosa was seen in the larynx and pharynx.

There was acute haemorrhagic congestion of both lungs, with blood dripping on section. Some blood mucus was seen in the lower end of the trachea.

ABDOMEN

Some degree of congestion was seen in the stomach and intestines. The liver and spleen were friable with acute haemorrhagic congestion. There was intense oedema of the kidneys, with scattered haemorrhage in the kidney substance. No haemorrhage was present in the adrenal gland. The bladder was completely empty and there was no inflammation of the mucosa of the bladder. *Fig.* 1 shows the distribution of the phenol on the thighs and legs.

Further Investigations

These were carried out at the High Peak Mortuary, which was built and completed in August, 1970, fulfilling all the requirements of the Home Office and was provided with a screening laboratory for preliminary investigations at the time of an autopsy.

Examination of the Right Thigh

The thigh was incised a length of 7 in. and the incision carried downwards to the right femur. The area was widened in the form of a cleft. The superficial skin was free from odour as it had been washed in attempts at diluting the phenol at the works. The smell of phenol was present in the deep tissues.

To determine depth of phenol absorption a Whatman's filter-paper was impregnated with a solution of ferric chloride, allowed to stand for a few minutes, and then folded in half. The folded



FIG. 1.—Distribution of phenol on thighs and legs of the deceased.

end was placed deep in the wound and pressed towards the sides of the incised muscles. A blue-violet colour was obtained, indicating the presence of phenol. It was also noticed that there was coagulation of the blood in the wound. A similar test with the incised kidney gave a similar positive test for the presence of phenol.

Blood was collected from the left femoral vessels. The following qualitative tests were made. The blood was centrifuged and the supernatant fluid removed and tested as follows:—

1. To 1 ml. of fluid was added a few drops of ferric chloride. A blue-violet colour was obtained, disappearing on the addition of excess of chloride.
2. To 1 ml. fluid, 2 drops of alcohol were added, then 2 drops of chloride. There was no colour.
3. To 1 ml. fluid, 2 drops of ammonia were added, then 2 drops of chloride. There was no colour.

These three tests confirmed the presence of phenol in the blood.

Unfortunately, in this case, owing to suppression of urine, there was no urine to demonstrate the presence of blood, casts, and the presence of hydroquinone giving rise to a dark urine on standing.

Forensic Laboratory Findings

The following were sent for examination to the Director, Forensic Laboratories, Nottingham:—

1. Blood sample from deceased.
2. Clothing of deceased.
3. Control sample of phenol.

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The following report of Mr. P. O. Rees, a Principal Scientific Officer was as follows:—

Clothing consisted of a white warehouse coat, trousers, pants, socks, tie, and a pair of brown shoes. All the articles had a strong odour of phenol. Parts of the warehouse coat and the trousers were cut out as representative of this specimen and the presence of phenol on these two garments was confirmed by chemical tests.

The control sample of phenol consisted of solid crystalline substance. Analysis showed this contained 98·4 per cent of phenol.

The blood specimen contained 7 mg. per cent of phenol.

The cause of death was cardiac and respiratory depression due to acute phenol poisoning.

DISCUSSION

Mr. Rees, on receiving the above, communicated with the author and informed him that this was the first case the laboratories had seen of intradermal phenol poisoning. When one considers the population and area which this laboratory covers, this form of poisoning must be unique.

From this fatality the importance of first-aid treatment to prevent the phenol reaching the skin surface is indicated. The clothing in this case was totally inadequate. Technicians working with phenol should be suitably clothed with boots with extensions to cover the thighs, non-absorbent gloves with gauntlets, non-absorbent apron with a sleeved gown of similar non-absorbent material, and goggles to protect the eyes. At the site, water in the form of a spray should be available and there should be a telephone in communication with the first-aid room. A second person, similarly dressed, should be present ready for emergencies. Should an emergency arise, this person will lie the victim in the prone position to prevent the flow down the clothing, apply the spray of water, and cut off the clothing with scissors provided. Immediate help should be summoned and the victim removed to the first-aid room. There, complete removal of clothing should be done and the site of the phenol spray again washed with water. The application of alcohol is not advisable in view of the fact that phenol is partly soluble in alcohol. For slight phenol splashes the treatment indicated by Cumming and Hayes (1970) may be used, using glycerol, polyethylene glycol, but this treatment is not feasible for a victim with extensive involvement. It is better to continue the water spray until medical help arrives. Should there be respiratory failure, resuscitation is indicated. Following medical examination the victim is transferred to hospital. If renal failure is imminent, haemodialysis is indicated (Matthew and Lawson, 1970). In addition, 100 mg. hydrocortisone should be given intravenously six-hourly. To combat water loss an intravenous infusion of sodium lactate should be given (Cumming, 1961). The area involved should be treated as a third-degree burn.

Acknowledgements

I am most grateful to Mr. H. Hartley, H.M. Coroner, High Peak, Derbyshire, for access to the inquest dossier, and to Mr. W. Stansfield, Chief Constable, Derby County and Borough Constabulary, for permission to publish the photograph in this case, and to other members of the Constabulary for their valuable help.

REFERENCES

British Chemical Industries Safety Council of Chemical Industries Association (1970), *First Aid Treatment of Phenol Splashes on the Skin—A New Approach*. London: Chemical Industries Association.

Casus, F. E. (ed.) (1968), *Gradwohl's Legal Medicine*, 2nd ed., p. 609. Bristol: Wright.

Cumming, D. M., and Hayes, M. J. (1970), *Br. J. ind. Med.*, 27, 155.

Cumming, G. (1961), *The Medical Management of Acute Poisoning*, p. 107. London: Cassell.

Matthew, H., and Lawson, A. A. (1970), *Treatment of Common Acute Poisonings*, 2nd ed., p. 128. Edinburgh: E. & S. Livingstone.

Simpson, K. G. (ed.) (1966), *Taylor's Principles and Practice of Medical Jurisprudence*, Vol. II. 12th ed., p. 250. London: J. & A. Churchill.

411538 0361

Rea Magnet Wire Company, Inc.
3600 East Pontiac Street
Fort Wayne, Indiana 46806
219 743-6032

1978 August 25

Dr. Kenneth S. Lane
Union Carbide Corporation
270 Park Avenue
New York, NY 10017

Dear Dr. Lane:

We at Rea Magnet Wire Company are contacting you due to our common interest in treating Phenol burns. We are interested in any information that you may have available on the use of Polyethylene Glycol for the treatment of Phenol burns.

Rea Magnet Wire Company has been using Polyethylene Glycol 200 for the removal of magnet wire enamels and thinners from the eyes and skin of Rea employees for the last 18 months. We discovered the use of Polyethylene Glycol by researching available literature on the treatment of Phenol burns, including a British article[1] presenting data on the removal of Phenol from the skin with various solvents or diluents. It was their conclusion that the best and most effective method for removing Phenol from the skin was by swabbing for a minimum of ten minutes with Glycerol, Polyethylene Glycol or a Polyethylene Glycol/Methylated Spirit[2] mixture.

The British researchers used Polyethylene Glycol 300, but we decided to use Polyethylene Glycol 200 because of its lower viscosity and its increased solvency power.

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1 "The Dermal Toxicity of Phenol: an Investigation of the Most Effective First-Aid Measures"; D.M. Conning and M.J. Hayes, Brit. J. Industr. Med. (1970) 27, 155-159.

2 70% Polyethylene Glycol/30% Denatured Alcohol

A typical magnet wire enamel contains approximately 30% resin solids while the remaining 70% consists of a mixture of aromatic Hydrocarbons (Xylene, C-9s and C-10s with a boiling point range of 138°C - 180°C) and Cresylic Acid (Phenol, Cresol, and Xylenols with a boiling point range from 182°C - 225°C). These enamels cannot be diluted or dissolved with water. The standard thinners are also not miscible with water. Because of this, we are using the Polyethylene Glycol as purchased. Also, we don't dilute the Polyethylene Glycol with alcohol because it is used to remove enamel from the eyes.

The Polyethylene Glycol has a slight warming effect (5 - 10°C.) and slight discomfort (when used in the eyes) but this is very mild when compared to an Alcohol/Polyethylene Glycol mixture. Diluting the Polyethylene Glycol with water decreases the discomfort to the eyes but nullifies its ability to dilute and dissolve the enamel.

I am enclosing the following list of information pertaining to the treatment of Phenol burns.

1. "Removal of Cresylic Acid, Thinners, and Enamels from the Skin", by Harold Otis, Rea Magnet Wire, R & D, 1976-03-02 with a copy of the British report cited previously.

2. "Glycol Solvent Used in Phenol Burn Therapy" Medical Tribune Report (Dr. Russel Pardoe).

3. "Treatment of Phenol Burns" (Dow Communique) by H. C. Scharnweber, M.D. and Corporate Medical Director, Dow Chemical Company Volume I, Number 4, 1977-09-05, with "Comparison of Decontamination Procedures for Acute Dermal Phenol Exposure in Swine", by T.G. Pullin, et al, Texas Division Medical Department.

4. Letter to Judy Aeschliman, RN from Dr. Russell Pardoe, Santa Clara Valley Medical Center, containing his comments on the Dow Communique.

Our comment on Dr. Scharnweber's conclusion that water is the preferred treatment "because of the universal availability of water" is that we have made Polyethylene Glycol 200 available throughout our plants and have it in areas where water is not available.

Our Fort Wayne plant dispensary has "copious quantities" of Polyethylene Glycol 200 available for the rapid removal of enamel or thinner from the eyes and skin.

We have shown a "sound on slide" presentation to all employees that might come into contact with materials containing Cresylic Acid (or Phenol). This was part of a safety program to make these employees aware that immediate action is necessary to prevent serious burns (and possible death) when any material containing Cresylic Acid contacts the skin. At this time they were introduced to the use of Polythylene Glycol 200 for the treatment of Phenol burns.

We realize that Union Carbide Corporation might not choose to officially go on record by recommending the use of the Polyethylene Glycol for the treatment of Phenol burns. However, we hope you and Union Carbide would not endorse the strong negative position of Dr. Scharnweber and Dow Chemical.

We have been informed that the Manufacturing Chemists Association will be issuing a document on the use of Polyethylene Glycol for the treatment of Phenol burns.

Yours sincerely,

Rea Magnet Wire Company, Inc.

Dr. Alan Richards, M.D.
Corporate Medical Director

Enclosures

AR/eb

Santa Clara Valley Medical Center

owned and operated by The County of Santa Clara
affiliated with Stanford University School of Medicine

751 South Bascom Avenue
San Jose, California 95128
293-0262 Area Code 408

September 21, 1976

Alan D. Richards, M.D.
Medical Director
Rea Magnet Wire Company, Inc.
3600 E. Pontiac Street
Ft. Wayne, Indiana 46806

Dear Doctor Richards:

Thank you for your letter of August 5. I apologize for the delay in replying, but I have been absent in Australia during August.

An article entitled "Phenol Burns" has been accepted for publication by the journal "Burns" which is the Journal of International Society for Burn Injuries and published by John Wright & Sons in Bristol. At this stage I am uncertain as to which issue it will be included in. If you have any problem obtaining a reprint of this article I will be happy to post you one when they come out or if you need any information in the meantime, I could send you the bibliography and a photostat copy of our work.

With thanks.

Sincerely,

Russell Pardoe

Russel Pardoe, M.D., Chief
Plastic & Reconstructive Surgery

RP:vph

SCHENECTADY CHEMICALS, INC.

P.O. Box 1046 Schenectady, N.Y. 12301 Area Code 518 346-8711 Cable Schenvar

July 5, 1978

Ms. Barbara Paris, Occupational Nurse
Rea Magnet Wire Co., Inc.
3600 East Pontiac St.
Fort Wayne, Indiana 46806

Dear Ms. Paris:

Per our conversation of last week relative to P.E.G. 200 for phenol and cresylic acid burns, would you please forward any information that you may have available regarding the application of P.E.G. 200 on the foregoing burns.

We would appreciate hearing from you as soon as conveniently possible.

Very truly yours,

SCHENECTADY CHEMICALS, INC.

Alois A. Matousek
Alois A. Matousek
Director of Safety

AAM:ap

78. 7-14
Received by Dr. Richards
78-7-21
Response okd by G. Smith - Plant Personnel
J. Lyle - Corp Personnel

411538 0366

Rea Magnet Wire Company, Inc.
3600 East Pontiac Street
Fo.t Wayne, Indiana 46806
219 743-8032

July 19, 1978

Mr. Alois A. Matousek
Director of Safety
Schenectady Chemicals, Inc.
P.O. Box 1046
Schenectady, New York 12301

Dear Mr. Matousek:

Dr. Alan Richards, our Corporate Medical Director, has reviewed your letter of July 5, 1978, and I am now forwarding to you most of the accumulated reference material upon which we based our pilot PEG 200 program.

We have also resourced Dr. J. Young of the Manufacturing Chemist Association at 1825 Connecticut Ave., N.W., Washington, D.C.,for the treatment recommended by M.C.A. They are preparing a revised document on phenol burns, and their care, and I'm sure would be happy to share this with you. Dr. Young may also be reached by phone - (202) 483-6126.

Dr. Russell Pardoe at Stanford University Medical Center has been a pioneer in the use of PEG in the care of phenol burns and has been very supportive to us in implementing this program.

Our plant has approximately fourteen of the burn baskets in high risk areas, and the baskets have approximately two 16 oz. wide mouth bottles of PEG 200 on each one. Field scissors developed in Vietnam, which cut through clothing and pads,are also in the basket. Simple instructions are attached for quick reference.

I maintain these supplies and a quality control program. I check various bottles at periodic intervals for culture at Lutheran Hospital and, to date, all cultures have been negative.

Education of the work force involved is my primary goal. I, on each and every occasion, stress immediate decontamination with either water or PEG, or both.

Rea has developed an eleven minute sound-on-slide presentation which has been shown to most of our work force. I have met with individual work groups and discussed the technique of using the burn basket prior to placing the basket in the area.

Finally, on one occasion, we had a significant contamination, above 30% of skin area which received immediate benefit of the program. This patient recovered completely in less than twelve days without complicatio This went a long way towards winning the compliance of any doubters.

As a result of this emergency, I presented our program to our local hospital emergency rooms along with an initial supply of PEG 200. I, also, asked for a briefing of all local E.M.S. personnel, since the ambulance attendants did protest the modality of my care for this patient. I accompanied this patient to the local emergency room, taking with us 2 gallons extra of PEG 200, and we continued care for in excess of 1½ hours.

If we can assist you any further, please feel free to call or write.

Very truly yours,

REA MAGNET WIRE COMPANY, INC.

Barbara Paris, RN

/fmt

ESSEX GROUP

CHEMICAL PROCESSING

1601 Wall Street, Fort Wayne, Indiana 46804 • Phone: (219) 461-4000

78-10-16

October 12, 1978

Bob - Please advise us of corporate position on this recurring question - B Paris, Jr

Dr. Allen Richards
REA Magnet Wire Co., Inc.
Medical Department
3600 E. Pontiac
Fort Wayne, IN

Dear Dr. Richards:

As Safety Officer for the Essex Chemical Processing Plant, I am interested in becoming better educated on the treatment of Phenol burns. It was recently learned that REA Magnet Wire developed the Phenol burn treatment now used by the St. Joseph Hospital in Fort Wayne. In a telephone conversation with Barb Paris of REA, I was informed that REA has developed a program for the handling of Phenol and that parts of this program could possibly be obtained by writing you.

I would greatly appreciate any information you could pass on to me that has been found to be beneficial in improving the safe handling of Phenol and in the treatment of Phenol burns.

Please contact me if you have any questions about my request.

Sincerely,

Lewis M. Brown

Lewis M. Brown
Plant Engineer
Ph. #461-4487

LMB:ej

10-20
Barb - Thought we all agreed to pass along this info to friends and competitors. Suggest you advise Jr.

OK - To release
Dom

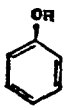
Subsidiary of
UNITED TECHNOLOGIES.

411538 0369

ANTIPRURITICS AND LOCAL ANESTHETICS 84:08

Phenol U.S.P. Carbolic Acid

Phenol, Liquefied, U.S.P.



Phenol in dilute solution is used by topical application on the skin or mucous membrane for its local anesthetic action and antiseptic properties. It is an effective antipruritic and for this purpose it is often incorporated in such lotions as calamine. Liquefied phenol, containing 88 percent phenol, is more readily incorporated into compounded prescriptions.

Dosage

For their antipruritic effect preparations containing 0.5 to 2 percent of phenol are applied locally several times a day. Phenol in glycerin may be used in 5 percent concentration since glycerite of phenol is less caustic than aqueous mixtures. The glycerite may be employed in the local management of itching, applied to superficial burns, and used as a dressing for painful ulcerations. Liquefied phenol is used as a caustic to cauterize necrotic tissue and as a germicide in badly infected wounds; this preparation must be used with great caution to avoid accidental contact with normal tissue, from which it may be removed with alcohol or vegetable oils.

Preparations

LIQUEFIED PHENOL

U.S.P. (88% phenol)

Glycerite 5%

Liquefied Phenol	5.	ml.
Sodium Citrate	2.5	Gm.
Purified Water	2.5	ml.
Glycerin, to make	100	ml.

92:00 UNCLASSIFIED THERAPEUTIC AGENTS

APPENDIX

INDEX

86:00 SPASMOLYTICS

88:00 VITAMINS

411538 0370

DOW CHEMICAL U.S.A. MIDLAND MICHIGAN 48640 EMERGENCY PHONE: 517-636-4400

EFFECTIVE DATE: 10 NOV 75 DATE PRINTED: 1 APR 77 PRODUCT CODE: 66417

PRODUCT NAME: POLYGLYCOL E-200 MSD: 0096

INGREDIENTS (TYPICAL VALUES)	%
POLYETHYLENE GLYCOL, ESSENTIALLY	100

SECTION 1 PHYSICAL DATA

BOILING POINT: DECOMPOSES : SOL. IN WATER: COMPLETELY MISCIBLE
VAP PRESS-MMHG @ 20C: LOW : SP. GRAVITY: 1.12 25/25C
VAP DENSITY (AIR=1): NOT APPL. : % VOLATILE BY VOLUME: NOT APPL.
APPEARANCE AND ODOR: COLORLESS/BLAND LIQUID.

SECTION 2 FIRE AND EXPLOSION HAZARD DATA

FLASH POINT: 360-480F : FLAMMABLE LIMITS (STP IN AIR)
METHOD USED: CLEVELAND OPEN CUP : LEL: NOT DETER. UEL: NOT DETER.
EXTINGUISHING MEDIA: WATER FOG, ALCOHOL FOAM, DRY CHEMICAL.
SPECIAL FIRE FIGHTING EQUIPMENT AND HAZARDS: ----

SECTION 3 REACTIVITY DATA

STABILITY: STABLE.
CONDITIONS TO AVOID: ----
INCOMPATIBILITY: OXIDIZING MATERIAL.
HAZARDOUS DECOMPOSITION PRODUCTS: ----
HAZARDOUS POLYMERIZATION: MAY OCCUR.
CONDITIONS TO AVOID: ----

SECTION 4 SPILL, LEAK, AND DISPOSAL PROCEDURES

ACTION TO BE TAKEN (USE APPROPRIATE SAFETY EQUIPMENT): SHOULD NOT BE LEFT ON FLOOR - MAKES IT SLIPPERY. SCOOP INTO DRUMS. SOAK UP WITH ABSORBENT MATERIAL.
DISPOSAL METHOD: SALVAGE OR BURN, IN ACCORDANCE WITH FEDERAL, STATE AND LOCAL LAWS.

SECTION 5 HEALTH HAZARD DATA

INGESTION: LOW. LD50 RANGE: 14.1 - 34.0 G/KG. (RABBITS, RATS, GUINEA

(CONTINUED ON PAGE 2)

DOW CHEMICAL U.S.A. MIDLAND MICHIGAN 48640 EMERGENCY PHONE: 517-636-4400
PRODUCT CODE: 68417
PRODUCT (CONT'D): POLYGLYCOL P-200 MSD: 0096

SECTION 5 HEALTH HAZARD DATA (CONTINUED)

INGESTION: (CONTINUED)
PIGS). ESTIMATING FROM THESE DATA, THE LETHAL DOSE FOR A 100 LB. PERSON MAY BE GREATER THAN A PINT.
EYE CONTACT: SLIGHT PAIN AND CONJUNCTIVITIS (RESPONSE TRANSITORY). RESEMBLES MILD SOAP.
SKIN CONTACT: NO SIGNIFICANT SKIN IRRITATION OR SENSITIZATION.
SKIN ABSORPTION: ----
INHALATION: NOT A FACTOR. NO TLV SUGGESTED.
EFFECTS OF OVEREXPOSURE: NONE EXPECTED UNLESS LARGE QUANTITIES ARE INGESTED.

SECTION 6 FIRST AID--NOTE TO PHYSICIAN

FIRST AID PROCEDURES: CAUTION - NEVER GIVE FLUIDS OR INDUCE VOMITING IF PATIENT IS UNCONSCIOUS OR HAVING CONVULSIONS.
EYES: FLUSH WITH PLENTY OF WATER. NO INJURY IS TO BE EXPECTED BUT IF PERSISTENT IRRITATION DEVELOPS, GET MEDICAL ATTENTION.
SKIN: NO INJURY EXPECTED. IF LARGE AMOUNTS ARE SPILLED ON SKIN, WASH OFF WITH WATER AND SOAP.
INHALATION: NOT LIKELY A PROBLEM.
INGESTION: IF LARGE AMOUNTS ARE INGESTED, INDUCE VOMITING AND GET MEDICAL ATTENTION.
NOTE TO PHYSICIAN: NO SPECIFIC ANTIDOTE KNOWN. TREATMENT DEPENDS ON THE SOUND JUDGMENT OF THE PHYSICIAN AND THE INDIVIDUAL REACTIONS OF THE PATIENT.

SECTION 7 SPECIAL HANDLING INFORMATION

VENTILATION: GOOD ROOM VENTILATION USUALLY ADEQUATE FOR MOST OPERATIONS.
RESPIRATORY PROTECTION: ----
PROTECTIVE CLOTHING: ----
EYE PROTECTION: NOT NORMALLY NECESSARY.

SECTION 8 SPECIAL PRECAUTIONS OR COMMENTS

PRECAUTIONS TO BE TAKEN IN HANDLING AND STORAGE: DO NOT STORE ABOVE 110F - PREFERABLY BELOW 95F. DO NOT CONTACT WITH HOT IGNITION SOURCE, SUCH AS AN ELECTRIC ELEMENT. OBSERVE REASONABLE CARE AND CLEANLINESS.

ADDITIONAL INFORMATION: ----

LAST PAGE

THE INFORMATION HEREIN IS GIVEN IN GOOD FAITH, BUT NO WARRANTY, EXPRESSED OR IMPLIED, IS MADE.

THE DOW CHEMICAL COMPANY QUALITY ASSURANCE 68417
SALES SPECIFICATION

DATE PRINTED: 1 APR 77 PAGE: 1
PRODUCT CODE: 68417 EFFECTIVE: 27 DEC 66

NAME: POLYGLYCOL E-200

DESC:A VISCOUS LIQUID, SLIGHTLY HYGROSCOPIC, MISCIBLE WITH WATER
APPL GOVT/IND STDS:SEE RELATED FDA REG.

TEST ITEM	: UNIT :	LIMITS	: TEST METHODS
MOLECULAR WT.	AVG.	190-210	ADD-A-1
VISCOSITY AT 210 F	CKS	4.1-4.8	ADD-J-1
ACIDITY (AS ACETIC ACID) MAX	%	.02	ADD-I-1
BASICITY (AS NAOH) MAXS	%	.02	ADD-I-1
FREEZE RANGE	C	SUPERCOOLS	ADD-N
WATER SOLUBILITY AT 25 C		COMPLETE	ADD-O
COLOR, MAX, APHA		25	ADD-F
ASH, MAX	%	.01	ADD-P
PH (5% AQUEOUS SOLUTION)		4.0-7.0	ADD-C-1

PI:1098060 BULK
PI:1102060 55 GAL DRUM
PI:1100064 5 GAL DRUM
PI:1464213 1 GAL DRUM

LAST PAGE

411538 0373

EMERGENCY TREATMENT FOR PHENOL BASED ACID OR ENAMEL BURNS

EMERGENCY SERVICE VEHICLES

Upon contact from Rea Magnet Wire requesting emergency service, prepare for rapid transport to Lutheran Hospital Emergency Room. Cooperation with plant personnel should take priority over routine vital signs and history. Application of PEG 200 en route is treatment of choice. Treat all clothing as contaminated. Remove clothing as rapidly as possible. Jewelry, glasses, belts, and shoes should be removed as these may cover skin and prevent PEG contact. Life support may include:

1) Ventilation - via ambu bag or mouth-to-mouth/CPR

2) Preparation for convulsions - control of emesis

At least one (1) gallon of PEG 200 should accompany each victim to the emergency room. Application of PEG is best achieved by gently swabbing the contaminated skin area with any available clean, non-abrasive applicator (i.e., pads, cloths, sponges, etc.). When transporting burn victims, if multiple burns are present, apply PEG 200 to burns in sequence so that none are neglected and all receive some application with priority to soft tissues such as eyes, neck, underarms, genitals, groin, abdomen, etc. (PEG may be used in eyes without difficulties.) When time and personnel permits, routine history and vital signs can be established. Phenol exposure may be lethal within ten (10) minutes of contact, depending upon body surface area exposed and chemical make-up. Death may occur in the first 15 minutes due to central nervous system depression, and respiratory arrest.

EMERGENCY TREATMENT FOR PHENOL BASED ACID OR ENAMEL BURNS

HOSPITAL EMERGENCY ROOM

Upon receiving phenol acid or enamel burn victims from Rea Magnet Wire, if no treatment has been instituted, IMMEDIATE deluge with water is urged. If PEG 200 is available (check for supply in emergency room) alternate water and PEG for at least twenty (20) minutes to contaminated skin area. If no PEG 200 is on hand in your emergency room, call the hospital pharmacy for PEG - diluted 1:1 with H_2O (water), which may be substituted. (PEG may be used in the eyes without difficulties.) If treatment has begun at the site or en route, continue treatment until the victim no longer complains of skin burning and smell is completely gone from the contaminated skin. This may take more than an hour and treatment may have to be repeated at intervals for comfort. Treat all clothing as contaminated. Remove clothing as rapidly as possible. Jewelry, glasses, belts, and shoes should be removed as these may cover the contaminated skin and intensify burns and absorption. Application of PEG to contaminated skin surfaces may be done with any suitable applicator such as soft cloths, gauze, pads, etc. Care should be taken not to abraid the skin. Priority must be given to the victim with the greatest amount of total skin surface exposure. PEG should be applied to all contaminated skin areas giving priority to those most permeable such as eyes, neck, underms, abdomen, groin, genitals, etc. In treating multiple burns, adapt a technique of sequential PEG application so that all burns receive attention. Speed is essential as deaths have been reported within ten (10) minutes of contamination. Death may occur from central nervous system depression and respiratory arrest. The following life support systems may be

instituted upon admission if respiratory difficulties are noted:

1) Ventilation - Mask or E-Tube

2) Treatment - Shock

3) Treatment of Acidosis

4) Maintenance of high urinary output

A urine sample to lab for hemoglobin or red cells should be routine. If patient's condition permits, transport to physical therapy for Hubbard tank treatment in 90-degree water for twenty (20) minutes to cleanse skin. If contaminated areas are greater than 20% or more of total body surface, observation is indicated for at least the first twenty-four (24) hours. No dressings are indicated if skin is not broken - occlusive dressings may enhance absorption of phenolic acid.