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Industrial Hygiene.

INDUSTRIAL BENZOL POISONING IN MASSACHUSETTS.

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BENZOL, or benzene, is used extensively in the industries of this State, and has given rise to much ill health and has caused several deaths. It is an unstable, very volatile, colorless fluid with a heavy aromatic odor. It is a derivative of coal tar distillation, and is frequently confounded in literature with *benzine*, a petroleum derivative with which it has many properties and uses in common. Benzol is decidedly more poisonous than benzine.

It contains many poisonous substances, such as hydrogen sulphide, toluene, and bisulphide of carbon (50 per cent., often). Much of the toxic effects noted is due to these substances. Poisoning by benzol depends upon individual susceptibility and immunity (natural or acquired), as well as upon the quantity and proportion of impurities in the fumes inhaled.

The principal uses of benzol (benzene) are:

Manufacture of colors and dyes; carborizing motor gas and illuminating gas; refining and dissolving fat and grease; lacquer (acid proof) colors and varnish work; rubber industry; as a cement in auto tire-building; in anti-rust, anti-corrosive and rapid drying paints. It is also used in the manufacture of pharmaceutical preparations, *e. g.*, aspirin, antipyrine, etc., as well

as in the preparation of perfumery scents. Lately, benzol has been extensively used as a substitute for benzine and oil of turpentine, because of its great power in dissolving resin.

Benzol, like its homologues and other hydrocarbons of coal tar, has a specific affinity for the central nervous system and a general action on the protoplasm of the organic cell. The remarkable property possessed by benzol of reducing the number of leucocytes in the blood has given to it a place in medicine in the treatment of myelogenous leukemia. Female workers, particularly at the age of puberty and at the menstrual period, are especially susceptible to benzol poisoning. Symptoms of benzol poisoning vary much with the mode of poisoning; *i. e.*, whether in the form of vapor through the respiratory organs, or by absorption through the skin, or through the digestive tract.

The following cases reported to the State Board of Labor and Industries are typical of the symptoms and result of benzol toxemia.

ACUTE BENZOL POISONING.

Two steamfitters were employed in repairing pipes inside a benzol still that had been drawn off three days previously. These men worked together inside the still. This retort was 5 ft.x8 ft. and was entered by a manhole opening 11 in.x15 in. A stream of compressed air under sixty pounds' pressure was fed into the still by a 2 in. pipe while the men were engaged at this work.

One workman, 35 years of age, entered the still about 11 a. m. and worked until 12 noon. He and another workman, 23 years of age, entered the still at 12:30 p. m. At 1:15 p. m. the older man became greatly excited, laughing, singing, gesticulating and

acting "as if he had gone crazy." He soon became unconscious and was lifted through the manhole by his fellow worker. It required twenty minutes to get his body through the opening of the retort. It was then discovered that the younger man was unconscious on the floor of the still. He was removed and all efforts to resuscitate him failed. No autopsy was held.

The older man was resuscitated by artificial respiration, oxygen inhalation, lung motor and stimulants. Several days later he was able to report for work.

ACUTE NITRO-BENZOL POISONING.

A man, sixty-five years of age, employed as a teamster for a soap factory, on July 7, 1916, while delivering a five-gallon flask of oil of mirbane (nitro-benzol) accidentally loosened the cork of the flask, spilling the chemical onto his clothes. A fellow workman noticed a pale, sick appearance and before he could reach the teamster he had grown dizzy, nauseated, cyanotic about the lips, mentally confused and was soon unconscious. Artificial respiration failed to resuscitate him and he was taken to the hospital. He soon developed Cheyne-Stokes respiration, slow, weak pulse, blood pressure 90 systolic, 50 diastolic. Blood showed methaemoglobin; later convulsion and coma developed, followed by death three and one-half hours after the accident. Autopsy findings: Poisoning by nitro-benzol.

CHRONIC BENZOL POISONING.

In view of the little information available on this subject in textbooks, the five chronic cases of benzol poisoning (three fatal) reported in Massachusetts are given in detail. All of these cases occurred in the auto tire-building department of a rubber establishment in which benzol cement was used. The process was essentially as follows: The benzol was poured from a can which was rigged with a ball-cock arrangement and a trigger handle so that it was necessary to pull the trigger in order to get a flow of benzol from the can. The benzol was poured onto a cloth and wiped around the tire, preparatory to vulcanizing. This process was repeated about every half hour, or approximately eighteen times in an eight-hour day.

1. (Fatal) A man, 33 years of age, had been employed in the rubber industry since 1907. On April 20, 1915, he was transferred to the tire-building department. He soon complained of severe headaches, often lasting two or three days at a time. In March, 1916, he noticed that he was spitting blood, especially after brushing his teeth. This condition of spongy, bleeding gums was noticed almost daily, and later in March he noticed bluish-green spots on his left thigh, followed in a few days by similar spots on his left arm. Soon similar spots appeared in rapid succession on various parts of the body. On May 3rd he gave up work on account of extreme weakness and dyspnoea on slight exertion. On this date he had a severe nose-bleed, followed by a similar attack on May 7th, when he was admitted to the hospital.*

Physical examination showed spongy, bleeding gums, pale mucous membrane of throat and con-

* The clinical records of these three cases of chronic benzol poisoning were furnished by St. Elizabeth's Hospital, Brighton.

junctiva, ulceration in posterior nares. Spleen not palpable. Knee jerks present and equal. No Babinski, no ankle clonus. Eyes react to light and to distance, no nystagmus. Breath, foul odor. Free bleeding from right nostril. Speculum examination showed bleeding point on septum about $\frac{3}{4}$ inch back and near floor of nares. Patient conscious, slight tremor of head, no glandular enlargements, lungs normal, heart sounds weak, regular, no murmur, spleen not felt, liver 2 cm. below costal margin, no masses in abdomen, no spasm, no tenderness. Ecchymotic spots on skin, most marked on outer side of left arm near shoulder and on outer side of each leg. Family history and personal history good. No purpura nor haemophilia in family. Pulse 124, weak. Vertigo and visual disturbance.

Haemoglobin	60%
Leukocyte count	5,000
Red count	2,288,000

Transfusion of ten ounces of blood, followed by uncontrollable epistaxis. Nose-bleeds daily, severe and uncontrollable. Gums spongy and bleed on least irritation. Headache, vertigo, dizziness, restlessness, delirium, talkative, loss of power of arms and legs, coma, convulsions and death on May 18th. Autopsy diagnosis: Benzol poisoning.

	MAY 7	MAY 11	MAY 12	MAY 14	MAY 16
Haemoglobin	60%	45%	40%	40%	35%
Leukocyte count	5,000	1,810	1,810	1,100	850
Red count	2,288,000	2,184,000	2,184,000	2,720,000	1,616,000
Polymorphonuclears	...	29	...	...	14
Large Mononuclears	...	14	...	...	20
Small Mononuclears	...	53	...	...	62
Eosinophiles	...	2	...	...	2
Myelocytes	...	...	...	...	...
Platelets	...	Dimin.	...	...	...
Achromia	...	Slight	...	...	Much dimin.
Poikilocytosis	...	Slight	...	...	Slight
Megaloblasts	...	Few	...	...	Few

2. (Fatal) A man, 40 years of age, had worked in a rubber factory since 1905. On May 21, 1915, was transferred to the tire-building department. In winter of 1915, noticed much loss of muscular power. Easily fatigued, dyspnoea on least exertion. He was not able to work continuously, was losing in weight, had repeated attacks of headache. In March, 1916, began to have bloody stools. April 1st, noticed tendency to have nose-bleeds and a constant cold in the head. About May 1st, had the first severe nose-bleed that he had difficulty in controlling. Gave up work on account of loss of muscular power, dizziness and shortness of breath.

Entered hospital May 3rd on account of severe nose-bleed.

Physical examination: Well developed and nourished, skin sallow, anemic mucous membrane, ulceration and bleeding spot in nose. Gums spongy, marked ecchymotic areas over lower and upper extremities, some areas being so large as to suggest trauma. No rigidity of neck, no glandular enlargement; lungs, heart and abdomen negative. No nystagmus nor strabismus, disc lemon color, teeth in poor condition, gums spongy, with blood oozing at junction of gums and teeth. Knee jerk present and equal on both sides. No ankle clonus, Babinski nor Oppenheim. No disturbance of sensation. Pulse 120; palpitation, vertigo, haemoglobin 65%; leukocyte count 10,000; red count 3,400,000; blood pressure 120 mg.

Patient failed steadily. Nose-bleeds frequent and more severe. Twitching of limbs and head. Paralysis, delirium, Cheyne-Stokes breathing, involuntary movements of bowels, coma. Death May 20th. Autopsy diagnosis: Poisoning by benzol.

	BLOOD RECORD.					
	MAY 3	MAY 10	MAY 12	MAY 13	MAY 16	
Haemoglobin	65%	45%	45%	45%	40%	750
Leukocyte count	10,000	4,800	2,300	800	1,742,000	27
Red count	3,400,000	1,872,000	1,632,000	1,552,000	1,742,000	13
Polymorphonuclears	...	38	...	...	...	54
Large mononuclears	...	10	...	...	...	5
Small mononuclears	...	48	...	...	...	1
Eosinophiles	...	3	...	...	...	
Mast cells	...	1	...	...	...	

3. (Fatal) A man, 30 years of age, had worked in a rubber factory about two years (since 1914) in tire-building department. No history of nose-bleed, purpura, no hemophilia, either individual or family. Late in 1915, noticed some "red spots" on face and neck. These persisted for about three weeks. During the spring, 1916, noticed that he was much weaker and not able to do his work so well as formerly. Had headaches and dizzy spells. On May 6, 1916, consulted a physician who reported symptoms as follows: Pallor, general weakness, loss of weight, dyspnea, pulse 100-110, palpitation, weakness of gait, headache, dizziness, tinnitus, low white and red count, dry cough. Man returned to work but had to quit again on May 27th. Returned to work again on June 12th.

On July 6th had nose-bleed from left nostril, lasting six hours. The symptoms increased in sever-

ity: Pallor, muscular weakness, loss of weight, dyspnea, weakness of gait, tinnitus and nose-bleed. Brought to hospital on July 20th on account of severe uncontrollable bleeding from left nostril.

Physical examination: Pale, anemic, mucous membrane pale. Patient conscious, pupils equal, mouth and teeth in good condition. Gums bleeding, lungs normal, heart rapid, pulse regular, systolic murmur at apex, spleen not palpable, liver 1 cm. below costal margin, no spasm, no tumor, no tenderness. Brownish-yellow stains and evidence of ecchymotic areas on anterior surface of both thighs; knee jerks lively, equal on both sides. Patient grew worse daily and on July 30th developed lobar pneumonia with edema of lungs. Died August 2nd. Autopsy diagnosis: Acute lobar pneumonia, edema of lungs, anemia, exhaustion, consequent on poisoning by benzol.

	BLOOD RECORD.					
	JULY 20	JULY 23	JULY 26	JULY 28	JULY 30	
Haemoglobin	40%	40%	30%	35%	30%	
Leukocyte count	2,000	2,100	2,200	1,840	1,600	
Red count	2,136,000	2,048,000	1,364,000	960,000	944,000	
Polymorphonuclears	29	...	30	...	...	
Large mononuclears	7	...	13	...	...	
Small mononuclears	62	...	55	...	...	
Eosinophiles	2	...	1	...	...	
Masts	Few	...	1	...	...	
Platelets	Dimin.	...	...	...	...	
Achromia	Mod.	...	...	...	...	
Poikilocytosis	Mod.	...	...	...	...	
Congulation time	...	18	...	...	...	

4. (Non-Fatal) Man, 20 years old, employed in a rubber factory since February, 1915. His duties consisted in receiving tires after they had been cured and apply to them a benzol cement. In March, 1916, he manifested symptoms of benzol poisoning, and in May reported to the hospital for asymmetry of face which he ascribed to the extraction of a tooth. Blood examination showed a haemoglobin of 75%, leukocyte count of 6000, red count 4,480,000. He had headache, nausea, general weakness.

5. (Non-Fatal) Man, 29 years old, employed for more than two years in automobile tire-building in which he wiped benzol to the fabric. For past three months has had a papular eruption size of pin head on extensor and flexor surfaces of arms, feet, ears and neck. Later this eruption developed into blebs. Very itchy and accompanied by fever. Stated that 16 or 18 men among the 120 employed in that department had similar eruption. Disappear on substituting naphtha for benzol.

A chemical examination of the benzol used by these men showed it to be free from impurities and of the pure benzol grade. In one of the cases the presence of arseniuretted hydrogen in large quantities in the urine raised a doubt as to possible arsenic poisoning. This was traced, however, to treatment by Fowler's Solution that the man was taking for anemia. The use of the benzol as a cement at the factory, in the tire-building process, was discontinued and naphtha substituted. No further cases of benzol poisoning have been reported to the State Board of Labor and Industries since this change.

Original Articles

ESSENTIAL SHRINKING OF THE CONJUNCTIVA OCCURRING IN ACUTE AND CHRONIC PEMPHIGUS, WITH A REPORT OF TWO CASES.

BY FRANK A. CONLON, M.D., LAWRENCE, MASS.

ESSENTIAL shrinking of the conjunctiva is simply a descriptive name of the end-result of pemphigus of the conjunctiva, although some men,¹ failing at the time of their examination to find specific evidence of a general pemphigus, have considered it a separate and distinct disease.

After a careful review of the literature, I find that all of these cases, so considered, come under Thost's² classification of chronic pemphigus of the mucous membrane, of which he gives the following characteristics:

1. Affection exclusively of the mucous membrane.
2. Unusual participation of the ocular mucous membrane, which tends to form synechiae.
3. Tendency to adhesion of all mucous surfaces.
4. Chronic evolution without fever.
5. Treatment of no avail.
6. Cachexia and dystrophy, cutaneous and muscular.

The lesions in the mouth or nose may easily be overlooked, as my patient told me her eyes troubled her so much that she had entirely forgotten about the condition in her mouth.

Pemphigus of the conjunctiva was first described by White Cooper³ in 1858. It was described as syndesmitis degeneration by Stellwag⁴ in 1870, and as essential shrinking of the conjunctiva by Kries, in 1878, of von Graefe's clinic. Von Graefe, in 1879, propounded the identity of pemphigus and essential shrinking of the conjunctiva.

It is a very rare disease. Adam⁵ saw one case in eleven thousand ophthalmic cases; Franke,⁶ five cases in forty-five thousand; Pergens,⁷ two in twenty-two thousand, while the late Herman Knapp⁸ saw but one case in his long experience.

It occurs with equal frequency in both sexes and at all ages; Alt⁹ reporting one in a girl of four and Banysiekiew¹⁰ in a woman of seventy-six.

Etiology. In the majority of cases there is no predisposing condition or cause, yet in a few a septic²² wound or vaccination²³ has been given as its origin.

A specific organism has been found in the bullae by the following writers: Cirincione,¹¹ Demme,¹² Dahnhardt,¹³ and Blutrau.¹⁴ They describe it as a diplococcus, rather larger than the gonococcus, which forms round, white colonies on agar, grows rapidly on bouillon at 37; well on agar and serum. The cocci stain with aniline dyes and by Gram's.

The cocci are not present in the sac after cicatrization is complete, and do not cause shrinking when placed in the conjunctival sac of animals, but do cause a rather severe conjunctivitis. Demme found the diplococcus in the blood. Xerosis bacilli in large numbers have been frequently demonstrated, but this, undoubtedly, has been due to the favorable soil found in the degenerate corneal epithelium. By some the condition is regarded as due to the action of toxins upon the nerve centers, resembling herpes in this respect.

CASE 1. Mrs. G., fifty years old. Pemphigus lesions first observed in the mouth along the first of August, 1915. Patient seen December 20, 1915, at which time she presented large bullae all over her body, with involvement of both eyes. The right eye presented an elevated, sharply defined spot five millimeters from the corneal sclerae on the nasal side about three millimeters in diameter. There was a loss of epithelium in the center, evidently a ruptured vesicle. Another similar spot was seen on the ball on the temporal side. The left eye showed two spots on the bulbar conjunctiva in corresponding positions and of identical appearance. Patient said her first eye trouble occurred one month before and lasted a week, and that the present lesions appeared that morning. Palpable conjunctiva was slightly injected and there was a beginning obliteration of the lower cul-de-sac. This seems a remarkably short time for a noticeable shrinking to develop, yet Stieren¹⁵ saw an almost complete obliteration of the conjunctival sac develop in six weeks.

CASE 2. Mrs. K., 50 years old, American born; while never a robust woman, she always enjoyed good health. I first saw this patient May 15, 1915, when she complained of an irritation due to entropion of her left lower lid. My records showed at that time twenty-twentieths vision in each eye, both corneae normal, slight redness of the bulbar conjunctiva of the left eye. The lower conjunctival sacs of both eyes were from three to four millimeters in depth, with numerous vertical folds of glistening white conjunctiva. At this time there was no appreciable involvement in the upper fornix of either eye. In June, 1916, she developed an erosion covering the lower half of the left cornea (ruptured bulla) with an iritis. Within a month, there was a complete obliteration of the lower cul-de-sac. The condition of her right eye remained