

FILE NAME: Owens Illinois Library (OWL)

DATE: 1929

DOC#: OWL006

DOCUMENT DESCRIPTION: Relevant Abstracts from The Journal of Industrial Hygiene

THE JOURNAL OF INDUSTRIAL HYGIENE

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VOLUME XI
JANUARY, 1929—DECEMBER, 1929

PUBLISHED BY
HARVARD SCHOOL OF PUBLIC HEALTH
Boston, Mass.

THE JOURNAL OF INDUSTRIAL HYGIENE

POISONOUS HAZARDS AND THEIR EFFECTS: GASES, CHEMICALS, ETC.

FATAL POISONING PROBABLY DUE TO ARSENIURETTED HYDROGEN. *Larde. Abstr. as follows from Soc. de éd. légale de Paris, Nov. 14, 1927, in nn. d'hyg., May, 1928, N. S. vol. pp. 300-301.*

Two workmen who had to clean out tank containing sulphuric acid and were apparently exposed to arseniuretted hydrogen. The men had been working for only twenty minutes. One said he felt ill; he went home, and died of acute edema of the lungs in forty-eight hours. The second developed bronchitis and gastro-intestinal trouble. In searching for a cause of poisoning, the sulphuric acid was found to contain 1 part of arsenic per thousand; this metal was recovered from the viscera of the deceased. As neither of the men had been taking any medicine containing arsenic, the author holds that the sulphuric acid in the tank, acting on the sludge, generated arseniuretted hydrogen. Once it gets into the lungs it is transformed into arsenious acid which causes considerable blood destruction. E. L. C.

BENZOL POISONING AS A POSSIBLE HAZARD IN CHEMICAL LABORATORIES. *J. Bloomfield. U. S. Pub. Health p., July 20, 1928, vol. 43, pp. 1896-97.*

Blood examinations of the workers exposed to benzol vapor in the chemical laboratories visited by the writer did not show an abnormal white blood cell count. However, the blood of one of the men showed a departure in

the relationship between the various types of white blood cells. Such a blood picture, coupled with a definite exposure to solvent vapors of a concentration considered toxic, according to present day standards, tends to indicate that the possibility of benzol poisoning in chemical laboratories must be considered.

Many industrial plants still use benzol, since the claim is made that the cost of suitable substitute solvents is prohibitive. Certain chemical laboratories use a sufficient amount of benzol to be considered as a potential hazard, but not enough of this solvent is utilized to justify its retention on the above claim. It is felt that in practically all chemical laboratories benzol could be replaced by some solvent known to be less toxic, such as toluol, xylol, or Hiflash naphtha. It is recommended at this time that in case the use of benzol in chemical laboratories is continued, it should be confined to the testing of materials only, and should not be employed as a cleansing agent. Also, in order to detect incipient benzol poisoning at a stage when its effects can be minimized, all laboratory workers exposed to benzol fumes should be given a thorough medical examination before employment, and should be reexamined, with systematic blood counts, once every month or two thereafter. Any worker whose blood picture, on reexamination, shows a marked departure from the normal (obtained from a previous examination) should be promptly excluded from benzol exposure.

ABSTRACT

CHRONIC CARBON MONOXIDE POISONING. *J. C. S. Battley. Canadian Med. Assn. Jour., Aug., 1928, vol. 19, p. 157.*

LEAD POISONING AS AN OCCUPATIONAL DISEASE. *Langelez. Bruxelles Méd., 1928, vol. 8, pp. 1579-1586.*

This article is issued by the Chief of the Industrial Medical Service in order to help doctors in carrying out their duties connected with the extension of the Compensation Acts in Belgium to certain industrial diseases, including lead poisoning. The usual accepted signs and symptoms of lead poisoning

are specified below based on conclusions from lead poisoning and inhalation of lead dust.

DUST HAZARDS AND TREATMENT

ANTHRACOSIS AND PULMONARY TUBERCULOSIS. *T. Wedekind. Abstr. as follows from Klin. Wchnschr., May 6, 1928, vol. 7, p. 380, in Jour. Am. Med. Assn., July 21, 1928, vol. 91, p. 214.*

In a series of experiments on rabbits, Wedekind succeeded in producing healing of tuberculosis of fatal type by intravenous injection of a suspension of coal dust. His theory is that irritation of the cells of the vascular endothelium by the particles of coal gave rise to migration of large numbers of phagocytes into the lung tissue.—K. R. D.

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Pulmonary Cook, vol. 2. The gestic bodies life to of as besto: These and e mean incub asbest may, ambl.

THE IMMEDIATE DIAGNOSIS OF PULMONARY ASBESTOSIS AT NECROPSY. *M. J. Stewart. Brit. Med. Jour., Sept. 15, 1928, vol. 2, p. 509.*

Juice from lungs fibrosed by asbestos dust exhibits under the microscope myriads of the "brown bodies" characteristic of this disease. Subsequent histologic examination revealed them

NOXIDE POR-
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1928, vol. 19,

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are reviewed, and fairly stated, with special attention directed toward the blood condition and the presence of basophil red corpuscles. The author considers these an effort of the hemato-poietic system to replace poisoned red cells rather than as degenerated cells. Somewhat unfortunately he ascribes lead poisoning to absorption through the skin, then through the digestion, and only lastly by the lungs; in fact inhalation of lead dust is of course the chief cause, and the skin, in occupation, may be entirely neglected as a portal of entry.—E. L. C.

HAZARDS AND THEIR EFFECTS

LMONARY TU-
nd. *Abstr. as*
hnschr., May
in Jour. Am.
1928, vol. 91, p.

nents on rab-
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dering on fibrotic areas, and, in smaller numbers, within the fibrotic patches. These bodies have been described only in asbestosis. In this case the worker, a woman, aged 34, had made asbestos mattresses for sixteen years; but had not worked for two years before death. Both lungs were fibrosed and anthracotic.—E. L. C.

PULMONARY ASBESTOSIS. W. E.
Cooke. Brit. Med. Jour., Sept. 29, 1928,
vol. 2, p. 585.

The writer counters Stewart's suggestions (*vide supra*) that the "curious" bodies found in lungs exposed during life to asbestos dust are (a) diagnostic of asbestosis, (b) derivatives of asbestos, and (c) not vegetable in origin. These bodies do not exist in asbestos and cannot be produced by chemical means from asbestos. But organisms incubated with a colloidal solution of asbestos change in appearance and may, it is thought, be made to resemble the curious bodies.

GNOSIS OF PUL-
AT NECROPSY.
Med. Jour., Sept.

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the microscope
bodies" charac-
Subsequent
revealed them

with lead oxides. The lead fume given off behaves like a gas, is absorbed into the finest bronchial tubes, and is today a common source of poisoning where the foregoing are practised. He describes the various combinations of gas flames used to produce a high temperature, laying stress on the fact that the higher the proportion of oxygen used the more lead is given off into the atmosphere as, e.g., with the oxy-acetylene flame. He believes that the sole real protection of the person employed using this flame is the "fresh air respirator," i.e., a facepiece with suitable tubing attached through which the wearer can breathe fresh air from a distance of, say, 30 feet. Provided the work is carried out in lofty, well-ventilated rooms, danger to other persons is not great as, owing to its weight, the amount of lead rapidly diminishes at 1 or 2 meters' distance from the burner. In Froboese's opinion no one should be allowed to do autogenous soldering who is not wearing a fresh air breathing apparatus, whether locally applied exhaust ventilation is present or not.

Thirteen experiments altogether are described, together with a minute account of the aspirating apparatus used and the method of determining the amount of lead found. Each experiment lasted for two hours and from 3 to 5 cu. m. of air were aspirated.

In one experiment (repair work by burning off the lead with a flame of hydrogen and oxygen in excess) 23.3 mg. in 10 cu. m. were found; in another (metallization) 17.3; in two others 5.3 and 5.2, respectively; and in the remainder from 0.2 to 4.5 mg. per 10 cu. m.—T. M. L.

THE OCCURRENCE OF LEAD IN THE EGG OF THE DOMESTIC HEN. PART III. W. B. S. Bishop. *Med. Jour. Australia*, June 15, 1929, vol. 1, pp. 792-806.

Articles reporting upon Parts I and II of this extended research appeared on April 21, 1928, and Jan. 26, 1929. (See *This Jour.*, 1928, vol. 10, Abstr. Section, pp. 216-217, and 1929, vol. 11, Abstr. Section, p. 158.) This considerable article concerns weights of eggs, the loss of weight during incubation, and the weight of embryos. Throughout, the methods used are carefully described, and the facts ascertained are stated in tabular form. The lead naturally present in the white-yolk was found to diminish during incubation from 0.08 mg. at 6 days to 0.005 at 20 days; meanwhile it increased in the embryo from 0.0008 mg. at 6 days to 0.08 at 20 days. The toxicity of lead compounds was tested by injecting them into the air lock of the egg; the fat-soluble compounds were found to be the least toxic, the order of decreasing toxicity being: the chloride, carbonate, colloidal lead, sulphide, phosphate, stearate, oleate, glyceride, and the lecithin compound. Lead was found to be present normally in egg yolk in two forms, as lead oleate and as lead lecithin compound.—E. L. C.

THE TOXICITY OF LEAD SULPHATE IN COMPARISON WITH THAT OF CARBONATE OF LEAD (WHITE LEAD). L. Schwarz and F. Sieke. *Abstr. as follows from Klin. Wchnschr.*, 1928, vol. 7, pp. 1838-1839, in *Bull. Hyg.*, May, 1929, vol. 4, p. 424.

The authors were asked to give a definite opinion as to the toxicity of

lead sulphate and, therefore, carried out comparative experiments with rabbits and cats feeding them with the sulphate and carbonate. Analysis of the sulphate revealed 72.7 per cent. sulphate and 26.7 per cent. lead oxide, i.e., 73.3 per cent. total lead. The sample of white lead contained total lead equal to 79.2 per cent.

The amount given in the food was 0.1 gm. per kilogram of weight daily for thirteen weeks; administration was then stopped as loss of weight, diminu-

DUST HAZARDS AND

DUST. C. O. Sappington. *Health Practices Pamphlet No. 4, Nat. Safety Council*, 1929, pp. 7.

A popular review of the conclusions of various investigators with regard to the industrial dust problem—in particular: the inhalation of free silica, the importance of particle size and of the toxicity of the material breathed, the mechanism of dust absorption by the lungs, the percentage retention of the dust inhaled, the prevention of dust formation and diffusion, the removal and collection of dusts and fumes, the advantages and limitations of masks and respirators, and the dust explosion hazard.

The procedure followed by the United States Public Health Service in their studies of the dusty trades is given.—T. H.

HISTOCHEMICAL STUDY OF ANTHRACOSIS LUNG. A. Policard and S. Doubrow. *Presse méd.*, July 10, 1929, pp. 905-907.

The authors report further on their

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The amount given in the food was 0.1 gm. per kilogram of weight daily for thirteen weeks; administration was then stopped as loss of weight, diminu-

tion in the hemoglobin content of the blood, and more or less considerable punctate basophilia occurred in the animals. In other experiments larger doses were given.

No significant differences were observed and the authors conclude that "sulphate of lead and carbonate of lead, when given with the food, or absorbed from deposits placed in the subcutaneous cellular tissue, have, for practical purposes, equal toxicity."—T. M. L.

DUST HAZARDS AND THEIR EFFECTS

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method of micro-incineration. A lung section placed on a slide is incinerated in a small electric furnace after which only inorganic cinders, still in their original position, are left. Such a section is now compared with an unincinerated fellow, when the exact relation of inorganic matter to lung pigment can be determined. The conclusion is drawn that some lung pigment is derived from iron in the blood and is endogenous, but, especially in those exposed to occupational dusts, most pigment originates as inhaled dust and is exogenous. Hence, evidence is obtained that dust is inhaled directly into the lungs, and that tubercle bacilli may be taken in by the respiratory route. The article is illustrated.—E. L. C.

A CASE OF PULMONARY ASBESTOSIS.

W. B. Wood and D. S. Page. *Tubercle, July, 1929, pp. 457-460.*

The clinical description and post-mortem findings are given of a woman, aged 34, who worked from age 21 for

itory air because of the constantly alternating inspiratory and expiratory movements and because of electric charges of the particles; form of the particles also must be taken into consideration: flat scale and ell-like particles are more easily kept in suspension. The sedimentation proceeds in direct ratio to the size of the particles and thus to the degree of humectation of the particles to the slowing of the respiratory movements in the smaller bronchi. In the large bronchi the mucosa is protected by ciliary epithelium and a layer of mucus, and the particles deposited on it are rapidly expectorated, without having penetrated into the pulmonary tissue; but in the small bronchioles there is no mucous secretion and the epithelium is thin and easily traumatized by deposited particles, which thus come in contact with the underlying connective tissue and are carried away by the macrophages or forced into various parts of the lungs by a continuous pulling of connective tissue caused by the expansion and the retraction of the lungs during respiration. Thus the mechanism of penetration of the mineral particles into the pulmonary tissue seems to be of the same nature as the mechanism of migration of a needle introduced under the skin.—K. R. D.

PULMONARY ASBESTOSIS. W. B. Wood. *Tubercle*, May, 1929, pp. 353-358.

Some description is given of the clinical condition of fifteen workers who came under observation after being exposed for varying periods of time to dust inhalation at an asbestos factory. Asbestos is a fibrous and

crystalline mineral composed mainly of magnesium silicate. The author deals chiefly with radiographic appearances and gives twelve illustrations. He considers that pulmonary asbestosis is a definite condition characterized by progressive dyspnea which becomes extreme; it is accompanied by cyanosis giving a leaden hue to the appearance. Wasting is a notable feature, while cough is a variable symptom. Clubbing of the fingers may be present. Prognosis appears to be grave owing to intervention of such diseases as pneumonia or bronchitis, or even tuberculosis, although the evidence is not quite definite as to whether a patient with asbestosis is more liable than a normal person to contract tuberculous infection. Skiagrams present shadows suggesting a diffuse fibrosis affecting chiefly the lower two-thirds of the lungs. The striations form a fine network, like that of a cobweb, which lacks the coarse quality of silicosis.—E. L. C.

THE PRESENCE OF THE ASBESTOS FIBRE IN THE LESIONS OF ASBESTOS WORKERS. S. R. Gloyne. *Tubercle*, June, 1929, pp. 404-407.

The author first examined powdered asbestos fibers, and then looked for them in workers. He found them in asbestos corns or skin thickenings which are common among the workers, in nose swabs, in mouth washes, and in sputum. But in no case were the "curious bodies" seen found by others in lungs. He found them however in profusion in the lungs of one worker. Careful examination of these "curious bodies" with concentrated sulphuric acid showed that they were material—possibly blood pigment—deposited

round an asbestos fiber. How they are formed is not, however, discussed but a further communication dealing with cellular reaction to asbestos in living tissues is promised.—E. L. C.

DEMONSTRATION OF THE PECULIAR BODIES OF PULMONARY ASBESTOS ("ASBESTOSIS BODIES") IN MATERIAL OBTAINED BY LUNG PUNCTURE AND IN THE SPUTUM. M. J. Stewart and A. C. Haddow. *Abstr. as follows from Jour. Path. and Bact.*, 1929, vol. 32 p. 172, in *Chem. Abstr.*, May 20, 1929, vol. 23, p. 2482.

The peculiar characteristic golden yellow bodies were found in two cases

OCCUPATIONAL AFFECTIONS C

ASEPTIC NECROSIS BY AN ANILINE PENCIL. P. Wilmoth. *Presse méd.* May 29, 1929, vol. 57, pp. 700-701.

Iselin in 1927 reported in *La Presse Médicale* certain injuries arising from aniline pencils. Now P. Wilmoth describes a further case which was due to a portion of an aniline pencil penetrating the skin of the foot of a young girl who was walking barefoot across the floor. At first there was no pain; but ten days later the skin swelled and was found to be stained a violet blue. Finally the skin broke discharging purple fluid and leaving an ulcer with a necrotic blue-stained base. The ulcer showed no signs of healing until all the stained tissue had been excised. Apparently bits of aniline under these circumstances pass into solution and continue to kill the tissues until they are removed. In the present case no general symptoms were reported, although Iselin men-

d an asbestos fiber. How they formed is not, however, discussed; a further communication dealing cellular reaction to asbestos in g tissues is promised.—E. L. C.

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low bodies were found in two cases

of pulmonary disease in individual
persons who had worked with asbestos
for several years.—P. D.

THE ESTIMATION OF SILICA IN TIS-
SUES. *E. J. King. Jour. Biol. Chem.,*
Nov., 1928, vol. 80, pp. 25-31.

"A micro method for the estimation
of silica in animal tissues, which de-
pends on the production of yellow
silicomolybdic acid, has been de-
scribed. Evidence is produced to
show that the method gives accurate
results.

"The silica content of some tissues
has been estimated; values are listed
in tabular form."

OCCUPATIONAL AFFECTIONS OF THE SKIN AND SPECIAL SENSES

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ry 29, 1929, vol. 37, pp. 700-701.

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tioned that headache, loss of appetite,
and mild jaundice may occur. Nor
is reference made to using ultraviolet
rays to hasten healing of the wound—
treatment which Iselin found of value.
—E. L. C.

GROSS AND MICROSCOPIC CHANGES
IN THE SKIN PRODUCED BY X-RAYS
AND BY LIPOID SOLVENTS. *L. H.*
Jorstad and C. W. Lane. Arch. Der-
mat. and Syph., June, 1929, vol. 19,
pp. 954-967.

The conclusions are given as follows:

Active precancerous lesions histologi-
cally have been produced in the skin of the
white rat by repeated painting with a
diluted tar preparation. The same type of
lesion is produced by a single exposure of
unfiltered x-rays, or by combining the two
procedures. In observing the histologic
changes produced by these procedures at
different intervals throughout the period of
experiment, further evidence was gained
that coal tar, allied substances such as

Oswaldo. Abstr. as follows from Ann. Ottal., Feb., 1928, vol. 56, p. 154, in m. Jour. Ophth., Nov., 1928, vol. 11, 938.

A man having the care of war projectiles undertook by means of a lathe to cut off the point of a hand grenade which was filled with phosgene gas, causing it to explode. He received the full effect of the contents in his nose and eyes and was immediately enveloped in a mist of gas. The face and lids became red and swollen; the cornea opaque with necrosis of the epithelium such as commonly follows an acid burn. The opacity prevented view of the iris or pupil. In a few days the injury was followed by a deep infiltration, corneal ulceration, and hypopyon, with prolapse of the endothelial tissues and loss of the eye. At the end of the year there was symblepharon and adhesion of the orbital tissues. The result was so

much more serious than that usually following an injury from phosgene gas that the author concludes that the gas must have had in it some bromine and chlorine to produce so serious an effect.

SPLENOMEDULLARY LEUKAEMIA IN AN X-RAY WORKER. *W. H. Evans and R. E. Roberts. Lancet, Oct. 13, 1928, vol. 2, pp. 748-751.*

A case is reported of typical myeloid leukemia of fourteen years' standing in an X-ray worker. Then all evidence previously published associating radiations with leukemia is critically reviewed; eleven cases were found, including two in radium and thorium workers. Finally the authors hold that the small number of cases makes it impossible to exclude coincidence, and pronounce the case against radiations as a cause of leukemia to be "not proven."—E. L. C.

DUST HAZARDS AND THEIR EFFECTS

EXPLOSIBILITY OF SULFIDE DUSTS IN METAL MINES. *E. D. Gardner and E. Stein. U. S. Bur. Mines Rep. Investigations, Serial No. 2863, 1928, pp. 11; abstr. as follows in Chem. Abstr., Nov. 20, 1928, vol. 22, p. 4320.*

Explosions have occurred in blasting of massive sulphide ores of greater magnitude than could be accounted for by the charge of explosive used in making the blast. During regular blasting in such ores sulphide dust is produced and scattered about the mine, while sulphur dioxide and hydrogen sulphide appear in the atmosphere. In these occasional heavy blasts the concentration of sulphur dioxide in the after-atmosphere is very high.

Gallery tests show that sulphide dusts can be ignited by blasting and that the combustion may be violent enough to be considered an explosion but it was not shown if such explosions are self-propagating. To prevent these explosions, settled dust should not be allowed to accumulate. All working places should be thoroughly wetted by sprinkling before the bore holes are loaded. And bore holes should be steamed and tamped to the collar.

A CASE OF PNEUMOCONIOSIS. RESULT OF THE INHALATION OF ASBESTOS DUST. *H. E. Seiler. Brit. Med. Jour., Dec. 1, 1928, vol. 2, p. 982.*

A case is reported of extensive

pulmonary fibrosis ascribed to inhaling asbestos dust. The fibrotic condition resembled that produced by silica dust, when seen by radiograph. The patient was a man aged 40 years, who for twenty-two years had been employed in the asbestos industry. He sought advice on account of chest symptoms associated with lassitude,

OCCUPATIONAL AFFECTATIONS OF

INDUSTRIAL SKIN LESIONS FROM SALTS OF ANTIMONY IN THE TEXTILE INDUSTRY. *A. B. Selitsky. Abstr. as follows from Dermal. Wchnschr., 1928, vol. 86, pp. 723-727, in Bull. Hyg., Feb., 1929, vol. 4, p. 170.*

The author describes occurrence of some 200 cases (including relapses) of industrial skin lesions due to the use as a mordant of solutions of salts of antimony in cloth dyeing. The form assumed was a pustular, necrotic dermatitis commencing usually as a folliculitis and ending in an atrophic scar. Usually the arms were affected. A perifollicular abscess formation was found histologically.

Etiologically the author believes that acid intermediate products, which are formed when antimony salts are used for mordanting, were responsible for the trouble. The condition was much the worst in the summer months and, in order to neutralize the excess of acid intermediate products, the author recommended addition of chalk. This step was followed by notable remission. After four months the chalk was given up on technical grounds, when the cases again became more numerous.

Use of indiarubber gloves and smear-

pulmonary fibrosis ascribed to inhaling asbestos dust. The fibrotic condition resembled that produced by silica dust, when seen by radiograph. The patient was a man aged 40 years, who for twenty-two years had been employed in the asbestos industry. He sought advice on account of chest symptoms associated with lassitude,

loss of weight, and night sweating. No tubercle bacilli were found, and his clinical condition is ascribed to his occupational hazard.—E. L. C.

CLINICAL PICTURE OF PNEUMONOKONIOSIS. *G. Schellenberg. Beitr. z. Klin. d. Tuberk., June 29, 1928, vol. 69, p. 498.*

OCCUPATIONAL AFFECTIONS OF THE SKIN AND SPECIAL SENSES

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Use of indiarubber gloves and smear-

ing the skin with vaseline is naturally of value.—T. M. L.

LESIONS DUE TO COPYING-PENCILS. *G. Marsiglia. Abstr. as follows from Riforma med., 1928, vol. 44, pp. 1308, 1311-1313, in Bull. Hyg., Feb., 1929, vol. 4, p. 179.*

Erdheim, a surgeon of Vienna, was the first to call attention to the deleterious effects of aniline dyes on living tissues, in 1914 and 1920. By experiment on guinea-pigs, rats, and rabbits, he showed that when small fragments of sterilized copying-pencils were introduced beneath the skin of the abdomen, a cyst formed round the foreign body and the color was diffused into the surrounding tissues and produced a wide necrosis. Fragments of green pencils produced the least injury, the violet were the most severe, gentian violet much less than methyl violet. A fragment of 2 sq. mm. of the last caused a zone of necrosis 3 cm. in diameter. Cicatrization did not take place until the whole of the necrotized tissue had been eliminated. In addition to the local lesions signs of intoxication might also occur, if the dose was a large one, leading to a fatal issue, and, postmortem, lesions of