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was not indicative insofar as exposure to low concns. of $PhNO_2$ was concerned. On the other hand, even with low concn. of $PhNO_2$ and $PhNH_2$ in the breathed air, urinary I and II levels were often too high. Detn. of urinary I and II was therefore suggested as a routine check in poisoning prophylaxis. Jerzy Lange a

Semi-quantitative determination of urobilinogenuria as an early diagnostic test for liver impairment due to poisoning. Henryk Bomski, Irena Kodura, and Halina Bomska (Zaklady Przemyslu Barwnikarskiego Boruta, Zgierz, Poland). *Med. Pracy* 14(2), 119-27(1963). In 136 workers exposed to amino and nitro compds., urobilinogen (I) was detd. in the urine according to Watson, *et al.* (*CA* 39, 2085). Abnormal values (over 1.5 units/2 hrs.) were found in 22 cases, of which 80% was diagnosed as toxic impairment of the liver. Detn. of I was suggested as a routine test in occupational exposures of the type referred to. Jerzy Lange b

Methanol determination in expired air and in urine in acute poisonings. K. Antczak and J. Piotrowski (Inst. Med. Pracy, Lodz, Poland). *Med. Pracy* 14(4), 321-33(1963). The air expired during 5 min. is bubbled through 30 ml. H_2O, 1 ml. of this soln. is mixed with 0.2 ml. H_2SO_4 (1:1) and 5 drops 2% $KMnO_4$, left 5 min., treated with 2 drops satd. Na_2SO_3, 0.2 ml. 2% chromotropic acid, and 4 ml. concd. H_2SO_4. In the presence of MeOH the mixt. turns purple and is assayed by colorimetry at 570 mμ. The sensitivity of the detn. is 1 γ/ml., which is equiv. to 6 γ/min. To det. MeOH in urine, a 50-ml. sample is acidified with 5 ml. 6*N* H_2SO_4 and distd. until 10 ml. is collected, 0.2 ml. of this distillate is dild. to make 1 ml., treated with 0.2 ml. H_2SO_4 (1:1) and 5 drops 2% $KMnO_4$, the further procedure being as above. EtOH interfered with the detn. to some extent, but its concn. has to be 1000 times higher to produce the color reaction of comparable intensity. Jerzy Lange c

Effect of cadmium oxide and nickel salts upon the upper respiratory tract. Barbara Pazolowa (Przychodnia Med. Przemyslowej, Poznan, Poland). *Med. Pracy* 14(5), 407-11(1963). Examn. of 78 workers of a Cd-Ni storage battery plant revealed symptoms of poisoning wherever the CdO and Ni salts concns. in the air exceeded permissible limits (0.1 mg. Cd/m.³ and 0.5 mg. Ni/m.³). The typical symptoms included irritation of nose and throat, edema of the palatal tonsil and posterior palatal arches, inflammatory changes in the larynx, and others. Jerzy Lange d

Histological pattern of endocrine glands in chronic furfural poisoning. Bronislaw Giedosz (Akad. Med., Krakow, Poland). *Med. Pracy* 14(6), 455-8(1963). Rats were poisoned by breathing, 1-4 months, air contg. 0.132 mg. furfural (I)/l., 30 mg. I being simultaneously added to the daily fodder. Distinct histol. changes were noted in the pituitary, thyroid, adrenal cortex, and ovaries. Jerzy Lange

Metabolism of γ-hexachlorocyclohexane. II. γ-HCH determination in urine by the method of Armstrong. Władyslaw Rusiecki, Halina Bronisz, and Bozena Wysocka (Akad. Med., Warsaw). *Med. Pracy* 14(6), 459-65(1963); cf. *CA* 61, 6307*f*. In quant. tests with urine 95.4% of γ-HCH (I) added to the urine was detd. by the method of A., *et al.* (*CA* 45, 7740*f*). In rats poisoned with I (125 mg./kg.) 0.33-0.55% of the intake was detected in urine. Urinary elimination of I was at the peak on the 5th-8th day of the expt. (up to 42 γ/animal/day) and no I was detected in urine after 17-18 days. Jerzy Lange e

Acute Frenolon intoxications. Imre Lazar (Tanacs Koranyi Frigyes Sandor Korhaz, Budapest, Hung.). *Orv. Hetilap* 105 (41), 1943-6(1964)(Hung). Frenolon [*N*-(β-hydroxyethyl)-*N'*-[γ-(3-chloro-10-phenothiazinyl)propyl]piperazine 3,4,5-trimethoxybenzoate difumarate] produced in 22 cases a 2-phase acute intoxication, with tachycardia, dryness of the mouth, and dizziness, in addn. to the tranquilizing effect. Then, independent of the intoxicating dose, dystonia of extrapyramidal origin developed. GGJH f

Fundamental parameters influencing the accumulation and elimination of carbon monoxide by adult human beings. T. H. Allen and R. W. Allard (Army Med. Res. & Nutr. Lab., Denver, Colo.). *U.S. Dept. Com., Office Tech. Serv., AD* 265,519, pp.(1961). The basis of a math. system for accumulation-elimination of CO by humans is described. The system can be solved by algebraic methods, and is able to predict the level of carboxyhemoglobin as a function of time from the initial level of carboxyhemoglobin, the concn. of inspired CO and O, expiratory flow rate, total body hemoglobin, and total pressure of gas breathed. These findings suggest that future physiol. investigations, using CO as a tracer, should include the measurement of further parameters than often included. Examples are given of the method of calcn., and this is used to illustrate the importance of each parameter. Although scarcely any data are available on elimination of CO it is shown how the system may predict the rate of elimination, esp. when using the newest method of treating CO poisoning by means of artificial ventilation with pure O at a total ambient pressure of 2 atm. 31 references. From *U.S. Govt. Res. Rept.* 37(2), 52-3(1962). TCTT g h

Exaltation of toxicity of sympathomimetic amines by thyroxine. Bernard N. Halpern, Carola Drudi-Baracco, and Denise Bessirard (Univ. Paris). *Nature* 204(4956), 387-88(1964); cf. *CA* 60, 2244*d*. DL-Thyroxine, 200 γ or 1 mg./kg., injected intraperitoneally daily into mice, increased the toxicity of subsequent injections of DL-amphetamine sulfate and ephedrine hydrochloride, depending on dosage and duration of hormonal treatment. BBJN

Panmyelopathy from exposure to toluene; comments. R. Hoschek. *Zentr. Arbeitsmed. Arbeitsschutz* 14(8), 189(1964) (Ger). Pure toluene contg. <0.03% benzene has no appreciable hepatotoxic activity. Infectious etiology is suggested for a case of alleged poisoning. **Concluding remarks.** H. Gattner. *Ibid.* 190. Polemic. Andrew L. Reeves

Phosphorylated thiocholine and choline derivatives. I. General toxicology and pharmacology. Sten-Magnus Aquilonius, Torsten Fredriksson, and Anders Sundwall (Res. Inst. Natl. Defence, Sundbyberg, Swed.). *Toxicol. Appl. Pharmacol.* 6(3), 269-79(1964). Diethoxy, diisopropoxy, methylethoxy, and methylisopropoxy derivs. of (2-dimethylaminoethylthio)-phosphine oxide, phosphorylthiocholine, (2-dimethylaminoethyl)phosphine oxide, and phosphorylcholine were screened for acute toxicity in the mouse and rabbit, and for *in vitro* inhibition of cholinesterase activity of human erythrocytes and plasma. Twelve compds. prepd. according to Tammelin were used (*CA* 52, 13816*g*). Intraperitoneal $L.D._{50}$ values of the O analogs in mice ranged from 1000 to 1310 micromoles/kg.; the corresponding range for the S analog was 0.074-140 micromoles/kg. Anticholinesterase activities (human erythrocytes), expressed as the neg. log of the inhibitory $dose_{50}$ (pI_{50}) ranged from 4.6 to 4.7 for the O analogs, and from 5.8 to 9.4 for the S analogs. A correlation was evident between the pI_{50} and the $pL.D._{50}$ (neg. log of $L.D._{50}$ expressed in moles/kg.) of the S analogs. Effects on blood press., heart rate, respiration, and neuromuscular and ganglionic transmission in the anesthetized cat were investigated for 5 of the compds., viz. methylisopropoxy(2-dimethylaminoethylthio)phosphine oxide, methylisopropoxyphosphorylthiocholine, methylisopropoxy(2-dimethylaminoethyl)phosphine oxide, methylisopropoxyphosphorylcholine, and diethoxy(2-dimethylaminoethyl)phosphine oxide. The S analogs of this group gave essentially the same effects as did Sarin, though the effects developed much more slowly than after Sarin. The O analogs in this latter group evidenced pharmacol. effects attributable to direct actions on cholinergic receptors. Substitution of the S by O thus reduced the potency of the compds. and also produced a change in the mechanism of action. Frank A. Smith i

The acute oral toxicity of sodium chloride. Eldon M. Boyd and M. N. Shanas (Queen's Univ., Kingston, Can.). *Arch. Intern. Pharmacodyn* 144(1/2), 86-96(1963)(Eng). The median lethal dose of NaCl given orally to young adult albino rats on an empty stomach was 3.75 g./kg. body wt. The mean interval to death was 8.6 hrs. The reaction in males was similar to that in females. The clin. syndrome included convulsive movements, diarrhea, muscular rigidity, prostration, and death due to respiratory failure. At autopsy, dehydration and vascular congestion were present in most organs except skeletal muscle, lungs, and skin. Congestion was esp. marked in the meninges and brain. Columnar epithelium lining the gastrointestinal tract was lysed. Survivors had a brief convalescent anorexia, polyuria, fever, and acidosis; appearance, wts., and water contents of organs were essentially normal at autopsy 2 weeks after drug. The cause of death, therefore, was respiratory failure assocd. with an acute encephalopathy, accompanied by a fulminating gastroenteritis and dehydration and congestion of many organs, due to oral administration of a single lethal dose of NaCl. From *Biol. Abstr.* 45(19), Abstr. No. 81607(1964). TCCG j

The form and changes in the form of asbestosis bodies [in the lungs]. R. Rath. *Beitr. Silikose-Forsch.* No. 81, 3-10(1964) (Ger). Old and new data are given on the so-called asbestosis bodies occurring in the lungs in fatal asbestosis. Such pathol. bodies arise from the inhalation of fibers of the chrysotile (I) form of asbestos. Since I has the form of a tube, the formation of asbestosis bodies is interpreted in terms of the diffusion of dissolved Si, Fe, and Mg out of the ends of the tubes of I (whose chem. compn. is interpreted in relation to reactions in the lung tissues). The breakdown of the bodies may result from a breakup of I fibers arising from a local narrowing of the tube diam. caused by diffusion of sol. materials. 15 references. W. C. Tobie k

Lung lesions observed one month after intratracheal injections [in rats] of coesite containing 1.5 percent quartz. Heinrich Breiger and Paul Gross (Jefferson Med. Coll., Philadelphia, Pa.). *Beitr. Silikose-Forsch.* No. 81, 43-50(1964)(Eng). The title injections of coesite dust gave a marked, generally multifocal, cellular and stromal reaction. The coesite lesions contained much reticulin and collagen in contrast to lesions produced by quartz dust alone. The effects of coesite (<0.5 μ) were thus much more fibrogenic than quartz dust alone (1-3 μ). Production of alveolar thickening probably was related to the smaller size of coesite particles rather than to its crystn. compn. W. C. Tobie l

Mercury poisoning from an unsuspected source. M. Tamir, B. Bornstein, M. Behar, and M. Chwat (Beilinson Hosp.,