was mixed with diphtheria toxin (0.2 ml.) and incubated with antitoxin at 37° for 5-6 days in medium 199. The min. toxin concn. detd. by the metabolic pH-indicator test using phenol red was 10^{-4}-10^{-5} Lf/ml. The test was applied using a range of inoculum sizes, vols., HCO_3^- concn. and antitoxin concn. Variance was 20% compared with 7% in the cytopathogenicity test, but the method was easier and was suitable for serial dilu. application. Peter M. Molton

59105g Toxicological investigation of cyanides. Ortega, Manuel; Vallejo, E. (Inst. Nac. Toxicol., Madrid, Spain). *An. Real Acad. Farm.* 1966, 32(4-5), 479-87 (Span). Blood (1-2 ml.) or minced viscera (2-3 g.) was placed in a Conway cell for 4 hrs. with 4-5 drops of 10% H_2SO_4 and HCN evolved was absorbed in 0.1 *N* NaOH. To the latter were added 2 ml. *M* NaH_2PO_4 and 1 ml. 0.25% chloramine-T, then after 2-3 min., 3 ml. reagent (3 g. barbituric acid, 15 ml. pyridine, 3 ml. HCl, and water to 50 ml.). The red to reddish purple color was read at 580 mμ after 10 min. Beer's law was obeyed for 0.5-2 γ KCN/ml. With time, cadaver CN^- is converted to CNS^-. To det. this, blood or viscera was deproteinized by adding an equal amt. of H_3PO_4, heating to boiling for 10 min., and adding 50 mg. of powd. picric acid/g. of tissue. After 12-24 hrs. at room temp., it was filtered and 1/3 vol. 5% $K_2Cr_2O_7$ and 2/3 vol. 50% H_2SO_4 were added to the filtrate in a Conway cell. Detn. of CN^- was as above. Recovery of added CNS^- was 54% (7% if not deproteinized). Disappearance of CN postmortem is approx. expotential and the amt. present at death cannot be accurately calcd. from subsequent detns. J. R. Gwilt

59106h Identification of hashish (Cannabis sativa). Chambon, Paul (Fac. Med. Pharm. Lyon, Lyons, Fr.). *Bull. Trav. Soc. Pharm. Lyon* 1968, 12(1), 43-6 (Fr). A sample of hashish was identified using color reactions and thin-layer chromatog. on silica gel G. The principal constituents were cannabinol, cannabidiol, and tetrahydrocannabinol.

Dorothy J. Buchanan-Davidson

59107j Identification and determination of nonvolatile organic poisons by infrared spectroscopy in forensic toxicology. Corneteau, H.; Monnet, R.; Boiteau, H. L. (Fac. Mixte Med. Pharm. Nantes, Nantes, Fr.). *Med. Leg. Domm. Corpor.* 1968, 1(4), 352-9 (Fr). Eleven alkaloids and 9 neuroleptic agents were identified and estd. by ir spectrometry after extn. and sepn. by thin-layer chromatog. on silica gel. The chromatogram eluates were concd. on KBr micropellets. Spots contg. 10 γ of compd. could be estd. with a precision of 8-15%.

L. A. Dehennin

59108k Utilization of an anti-γ-globulin immune serum for determining the human origin of blood stains. Tran Van Ky, Philippe; Lenoir, L.; Muller, P. (Inst. Med. Legale Lille, Lille, Fr.). *Med. Leg. Domm. Corpor.* 1968, 1(4), 392-5 (Fr). Extn. of a blood spot with isotonic phosphate buffer of pH 7.4 exts. the antigen. Monospecific antisera are obtained from rabbits immunized with human γG-globulins. The immunologic tests are performed by double diffusion on gelose.

L. A. Dehennin

59109m Evaluation of postmortem blood sugar levels and their correlation with the glycogen content of the liver. Ramu, M.; Robinson, Ann E.; Camps, F. E. *Med. Sci. Law* 1969, 9, 23-6 (Eng). Satisfactory blood glucose estns. may be made by using blood samples collected into tubes contg. F^- up to 48 hrs. after death, provided that a specific assay procedure is used. The blood glucose concn. for samples from the left side of the heart and the upper and lower extremities statistically belong to 1 group whereas the right heart blood and the liver blood belong, statistically, to 2 different groups. There were no significant variations in the blood glucose concn. with the time interval 12-48 hrs. after death. There is a significant inverse relation between liver blood glucose concns. and the glycogen content of the liver as assayed chem. Irving Sunshine

59110e Relations between the levels of alcohol in the blood and in the breath. Paolucci, G. *Riv. Med. Aeronaut. Spaz.* 1968, 31(3-4), 401-9 (Ital). Tests were made with 20 subjects (2 female) of av. age 25 who consumed, in a single dose, 0.50 g. EtOH/kg. in the form of cognac. Exhaled air was collected every 15 min. up to 2 hrs. after ingestion and blood was sampled at 45 and 120 min. in 15 of the subjects and every 15 min. in the remaining 5. To det. whether differences existed between the air exhaled in 1 profound expiration and alveolar air, the latter was collected in equal vol. immediately after the former. The blood EtOH increased regularly up to 60 min., then decreased irregularly up to 2 hrs. The ratio of EtOH in 50 ml. exhaled air to that in 1 ml. blood was about 1:2 for the first hr. after ingestion, varying with the individual. Blood EtOH is detectable up to 2 hrs. in subjects with considerable adipose tissue but has practically disappeared after 1 hr. in lean subjects.

J. I. M. Jones

59111f Fractionation analysis of metallic poisons. Kryleva, A. N. *Vopr. Sudebnoi Med., Min. Zhravookhr. SSSR* 1968, 314-21 (Russ). From *Ref. Zh., Khim.* 1969, Abstr. No. 2G175. A comparison of the accepted H_2S method and the new fractionation method of analyzing human organs during autopsy is made, with relation to a group of 12 poisons (Pb, Ba, As, Sb, Bi, Hg, Cu, Cd, Ag, Zn, Cr, and Mn). Qual. and quant. anal. methods take 1-2 working days instead of the 8 required for the H_2S method, with greater sensitivity and accuracy. MQRK

59112g Separation of copper and cadmium by extraction in forensic chemical investigations. Krylova, A. N. *Vopr. Sudebnoi Med., Min. Zhravookhr. SSSR* 1968, 322-7 (Russ). From *Ref. Zh., Khim.* 1969, Abstr. No. 2G176. A method of extn. and complexometric detn. of Cu and Cd in human organs is described. Mineralize the sample with HNO_3-H_2SO_4, add 20 ml. 20% K Na tartrate, 10 ml. glycerol (1:10), and 30% KOH until neutral and 5 ml. in excess. Add 10 ml. 1% Na diethyldithiocarbamate (I) and 10-15 ml. $CHCl_3$, shake, and repeat the extn. until a colorless $CHCl_3$ layer is obtained. Reext. Cd from the combined ext. by shaking with 10 ml. *N* HCl (3 times). Dil. the Cd ext. to 100 ml. with H_2O and bring to pH 8. Titrate Cd with 0.01 *N* EDTA until the red-violet color changes to dark blue. Wash the $CHCl_3$ ext. 3 times with 10 ml. 5 *N* HCl (to remove Bi and Tl) and then with H_2O until neutral reaction. Ext. Cu by shaking with 1% $HgCl_2$ until colorless, and det. in the ext. by titrn. with EDTA in the presence of murexide and 1-2 g. KI at pH 8 (color change from yellow to violet). MQRK

59113h Biochemical modifications induced by acute and subacute intoxication with white phosphorus in the rat. I. Action of repeated parenteral doses. Truhaut, Rene; Claude, Jean R.; Warnet, Jean M. (Fac. Pharm., Paris, Fr.). *Ann. Pharm. Fr.* 1969, 27(1), 17-23 (Fr). Repeated administration of white P to rats, parenterally, decreased body wt. A dose of 2.5 mg./kg. decreased liver wt. without visible steatosis; liver triglycerides were increased and phospholipids decreased, leaving the total lipid content nearly const. In the blood both total lipids and triglycerides were lowered. With 5 mg./kg. several rats died after 7-8 days following the 3rd injection, and all showed varying degrees of liver steatosis with increase in triglycerides and in total lipids. Since the activity of glucose-6-phosphate dehydrogenase remained normal it is unlikely that the biosynthesis of fatty acids and triglycerides was increased. Ruth M. Chester

59114j Toxicity of pyrolysis products of vinyl plastics. Cornish, Herbert H.; Abar, Ellen L. (Sch. of Public Health, Ann Arbor, Mich.). *Arch. Environ. Health* 1969, 19(1), 15-21 (Eng). Poly(vinyl chloride) (I) and vinyl chloride–vinyl acetate polymers (II) were pyrolyzed in air by gradually raising the air temp. from ambient to 600°. Rats were exposed to the pyrolysis products air stream dild. with room air to twice its initial vol. Exposure to air contg. the pyrolyzed products from 1-2 g. I resulted in death to 50% of the animals. Most deaths were due to CO, and carboxyhemoglobin levels correlated well with the amt. of pyrolyzed I. Little histol. evidence of lung damage was evident. When the air stream was dild. with O, pulmonary edema and interstitial hemorrhage developed. The lungs of animals exposed to high levels of II pyrolysis products showed focal edema and intraalveolar hemorrhage. I formulations contg. additives and inert materials were generally less toxic per g. of sample pyrolyzed. BZJN

59115k Validity of a critical blood level for prevention of dieldrin intoxication. Keane, William T., Jr.; Zavon, Mitchell R. (Agr. Chem. Div., Shell Chem. Co., New York, N.Y.). *Arch. Environ. Health* 1969, 19(1), 36-44 (Eng). In dogs given a single dose of 5 mg./kg. of dieldrin, dieldrin concn. in the blood reached a max. in 2-4 hrs. and declined after 10 hrs. to a relatively const. level which persisted for the following 22 hrs. All intoxications should therefore occur within 2-4 hrs. after feeding dieldrin. In dogs given 1.0 mg./kg. of dieldrin for 5 days, 0.2 mg./kg. for the next 57 days, and then 2 mg./kg. daily until intoxication occurred, the 1st muscular spasm occurred at a baseline concn. of 55 μg./100 ml. of whole blood. In both acute and subacute intoxication, the baseline concn. prior to intoxication was 62 μg./100 ml. After administration of the daily dose and subsequent appearance of intoxication, the blood concns. of dieldrin were, resp., 74 and 83 μg./100 ml. blood in the acute and subacute groups. The difference was not significant. Correlation between dieldrin concns. in blood and body fat was 0.84. The results support the hypothesis of a threshold level for dieldrin in blood which, when exceeded, results in intoxication independently of the type or duration of exposure.

Raymond Zehnpfennig

59116m Alteration of the locomotor activity of mice poisoned with sublethal doses of lead acetate. Terzin, A. L.; Vujkov, V. V. (Med. Fac., Novi Sad, Yugoslavia). *Arh. Hig. Rada Toksikol.* 1968, 19(4), 403-511 (Engl). Injection of $Pb(OAc)_2$ (0.4-20 mg. i.p.) into female mice (21-33 g.) produced dose-dependent changes in locomotor activity. Single doses produced depression of locomotor activity while chronic poisoning increased locomotor activity. Single or multiple i.p. injections of saline did not alter locomotor activity. Animals poisoned with lethal doses of $Pb(OAc)_2$ developed stippled cells during the 2nd or 3rd week while significant changes in locomotor activity were evident 1 day after the last injection. Mice poisoned with sublethal doses (0.4-1.0 mg.) showed no stippled cells but display changes in locomotor activity. P. H. Reiern