of the same order in white matter, gray matter, and peripheral nerve, but the concentration of PNA is much greater in gray matter. The concentration of the inositide P is much greater in white matter. These results are discussed in relation to the cellular constituents of these 3 tissues and the suggestion that the inositide P is derived from neurokeratin, a constituent of the myelin sheath.

A. PAPINEAU-COUTURE (Chem. Abstr.).

Isolated Nuclei of Cells of the Cerebral Cortex. Richter, D., et al. [*Abstracts Communs., 1st Intern. Congr. Biochem.*, 279 (1949).]

The enzymes of animal brains and of normal and pathological human brains are discussed. W. C. TOBIE (Chem. Abstr.).

Inhibition of Brain-slice Respiration In Vitro *by Alkyl Phosphates. Locker, A., et al.* [*Experientia*, **8**, 302–3 (1952).]

Tissue respiration of brain slices *in vitro* was inhibited by Parathion, Pestox III (bisbis [dimethylamino] phosphoric acid anhydride), Pestox XIV (bis [dimethylamino] fluophosphine oxide), and Pestox XV (bis [isopropylamino] fluophosphine oxide) in concentrations of 10^{-5}–10^{-4} M. The order of activity is Pestox XIV > Parathion > Pestox III > Pestox XV. With the exception of Pestox III the inhibitory action of these compounds on brain cholinesterase is greater than the effect on brain-slice respiration. D. S. FARNER (Chem. Abstr.).

2. Pharmacology and Treatment.

Action of Hydergin on the Circulation of the Brain. Taeschler, M., et al. [*Helv. Physiol. et Pharmacol. Acta*, **10**, 120–37 (1952).]

Hydergin decreased the cerebral vascular tone in cats but had little such effect in dogs. Whether the effect is central or directly on the musculature of the vessels is uncertain. L. E. GILSON (Chem. Abstr.).

Visual Disturbances due to Tridione. Dekking, H. M. [*Acta XVI° Concilium Ophthalmol. Britannia*, **1**, 465–7 (1950).]

The vivid sensations of glare and almost total disappearance of colour in brightly lighted objects which sometimes follows use of the drug is caused by a specific action on the retinal cones. It interferes with their light adaptation. Dark adaptation is not interfered with by this drug.

KARL F. URBACH (Chem. Abstr.).

The Anticonvulsant Activity of Ureides. Lubieńska, Jolanta. (*Polska Akad.*) [*Umiejetnosci Rozprawy Wydzialu Lekarskiego*, **12**, *No.* 6, 20 pp. (1951).]

The anticonvulsant activities were studied of the ureide derivatives of phenylacetate (I), α-phenylpropionate (II), β-phenylpropionate, α-phenylbutyrate (II), γ-phenylbutyrate, phenylcyanate, α-chlorophenylacetate, and α-bromophenylacetate. These compounds, practically insoluble in H_2O, were given as 10 per cent. suspensions in gum acacia. The most potent anticonvulsant was (I). For comparison purposes phenylethylbarbituric acid (IV) and the Na salt of diphenylhydantoin (V) were used. By the electric method on rabbits, (IV) and (V) were 4 times as potent as (I) in rats. (I) was 1·2 times stronger than (II) and (III) had minimum activity; the rest of the derivatives were without activity. In shocks produced by cardiac glycosides and coramine, (I) was 1·3 times as potent as (V); in coramine-induced shocks (I) was 1·9 times as potent as (V). (I) was effective in abolishing convulsions produced by malononitrile in white mice and protected frogs against picrotoxin-induced convulsions.

L. J. PIOTROWSKI (Chem. Abstr.).

Changes in the Sugar and Potassium of Blood During Electrometrazole-, and Insulin-shock in Man. Riechert, W., et al. [*Nervenarzt*, **23**, 212 (1952).]

The concentrations of glucose (I) and K were determined in blood taken before and at intervals after 6 electroshock treatments in 2 patients and 14 metrazole (II) shocks in 5 patients. K and (I) concentrations increased considerably immediately

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after electric shock and usually returned to the starting level half-an-hour later. A similar result was usually found after (II) shock, but there were some exceptions. Treatment with dihydroergotamine before (II) usually prevented changes in blood K but not of (I) concentrations; the occurrence of shock was not affected.
WARREN M. SPERRY (Chem. Abstr.).

The Pharmacological Properties of Laudolissin—a Long-acting Curarizing Agent. Collier, H. O. J., et al. [*Brit. J. Pharmacol.*, **7**, 398 (1952).]

Decamethylenebis (1, 2, 3, 4-tetrahydro-6, 7-dimethoxy-1-(3′, 4′-dimethoxybenzyl)-2-methylisoquinolinium salts), laudolissin (I), and decamethylenebis (1, 2, 3, 4-tetrahydro-6, 7, 8-trimethoxy-2-methylisoquinolinium iodide), Compound 15 (II) were more potent curarizing agents than *d*-tubocurarine (III) in the rabbit and cat but less potent in the rat and mouse. In equipotent doses (I) was longer acting and (II) shorter acting than (III). Paralysis caused by (I) or (II) was antagonized by neostigmine, and succinylcholine hastened recovery after (I) or (III). (I) and (II) acted synergistically with ether. (I) blocked autonomic ganglia less readily than (II) which in turn was less effective than (III). Both (I) and (II) elicited the release of histamine, as does (III). The comparative effect depended on the preparation tested. RICHARD F. RILEY (Chem. Abstr.).

Evaluation of Two Synthetic Curarizing Agents in Conscious Volunteers. Bodman, R. I. [*Brit. J. Pharmacol.*, **7**, 409 (1952).]

Laudolissin (I) was similar in action and lack of side effects to *d*-tubocurarine (II). (I) was half as potent as (II) when measured by reduction in hand-grip, but its duration of action was longer. The amount of histamine released by (I) was comparable to that released by (II). Compound 15 released excessive amounts of histamine. RICHARD F. RILEY (Chem. Abstr.).

The Use of Amphetamine in Barbiturate Poisoning. Dick, H., and Lenox H. [*Am. J. Med. Sci.*, **224**, 281 (1952).]

Eleven cases of barbiturate poisoning were successfully treated with amphetamine as the major analeptic drug. MARION HORN PESKIN (Chem. Abstr.).

Effects of Liver Injury and Nephrectomy on the Anticonvulsant Action of Phenacemide (Phenacetylurea, Phenurone). Weaver, L. C., et al. [*Proc. Soc. Exptl. Biol. Med.*, **80**, 744–6 (1952).]

The prevention of electroshock seizures in rats by the compound was studied. Liver injury (from CCl_4) significantly increased the potency and the duration of anticonvulsant action; bilateral nephrectomy had no such effects. This suggests that the liver is the principal organ for the destruction of the compound.
L. E. GILSON (Chem. Abstr.).

Anoxia Therapy. I. Psychic Action of Nitrogen Inhalation and its Therapeutic Application. Bach, W. [*Nervenarzt*, **19**, 449–64 (1948); *Abstracts World Med.*, **6**, 109 (1949).]

Physiological and psychic effects of inhaling N for 50-60 seconds were studied in 42 patients. Results in different psychiatric states are discussed. Anoxia with N is mainly effective in depressions, in which it compares favorably with electroshock therapy. W. C. TOBIE (Chem. Abstr.).

Influence of the Diethylaminoethyl Ester of Phenylcyclopentanecarboxylic Acid (Parpanit) on the Increased Metabolism in Parkinsonism. Mahaux, J., et al. [*Schweiz. med. Wochschr.*, **78**, 1013 (1948) (*in French*); *Abstracts World Med.*, **6**, 105 (1949); *cf. C.A.*, **43**, 4373*i*; **44**, 1605*a*.]

Parpanit usually produces a fall in the increased metabolism of Parkinsonism (within 30 minutes) and provides a means of assessing the action of other drugs.
W. C. TOBIE (Chem. Abstr.).

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Central Depressant Actions of Certain Myoneural Blocking Agents. *Ellis, C. H., et al.* [*J. Pharmacol. Exptl. Therap.*, **106**, 353 (1952).]

Some central effects (especially respiratory arrest) of large doses of *d*-tubocurarine, decamethonium, and succinylcholine in cats are reported and discussed.
L. E. GILSON (Chem. Abstr.).

Effect of Thyroxine, Thyroidectomy, and 6-Propyl-2-Thiouracil on Brain Function. *Woodbury, D. M., et al.* [*J. Pharmacol. Exptl. Therap.*, **106**, 331 (1952).]

Differences in susceptibility to electroshock seizures and metrazole-induced convulsions in normal, thyroxine-treated, thyroidectomized, and propylthiouracil-treated rats are reported. L. E. GILSON (Chem. Abstr.).

Pharmacological Studies of Phenylacetylurea (Phenurone), an Anticonvulsant Drug. *Everett, G. M., et al.* [*J. Pharmacol. Exptl. Therap.*, **106**, 303–13 (1952).]

Phenurone has low oral toxicity. It causes ataxia and coma in high doses. Fatal doses are in the range of 3–5 gm./kgm. for mice, rats, and cats. It has outstanding anticonvulsant action against supramaximal and psychomotor electroshock seizures in mice, and against metrazole, strychnine, and thujone convulsions. The duration of anticonvulsant action is 4–5 hours normally, and longer in animals with kidney damage from CCl_4. Phenurone is rapidly absorbed from the intestine but not from the stomach. In mice, double nephrectomy did not increase the duration of action. No phenurone was detected in human urine after ingestion of 2 gm. Chronic toxicity studies in rats and dogs for 3 years showed no ill effects. Phenurone is effective in various types of human epileptic seizures. The following compounds were also studied: (α-phenylpropionyl)urea, 1-methyl- and 3-methyl-1-(phenylacetyl)urea, 1, 3-dimethyl-1-(phenylacetyl)urea, (α-phenylbutyryl)urea, diphenylacetylurea, isobutyrylurea, 1, 3-dimethyl-1-isobutyrylurea, cyclohexylacetylurea, tolylacetylurea, chlorophenylacetylurea, and thienylacetylurea (position of substitution not stated in last 3 compounds). All had a depressant action, but only the first 5 were effective against electroshock and only the first 3 against metrazole shock. L. E. GILSON (Chem. Abstr.).

The Effect of Antabuse on the Electroencephalogram. *Busse, Ewald W., et al.* [*Am. J. Med. Sci.*, **223**, 126–30 (1952).]

A toxic influence on the brain, as demonstrated by alterations in the electroencephalogram, was exerted by antabuse (I) in 18 of 30 patients being treated with (I) for alcoholism. The changes were more frequent and intense in patients with abnormal, than in those with normal, electroencephalograms before (I) therapy.
MARION HORN PESKIN (Chem. Abstr.).

Effects of Alcohol in Small Doses and Tetraethylthiuram Disulfide (Antabuse) on Cerebral Blood Flow and Cerebral Metabolism. *Hine, C. H., et al.* [*J. Pharmacol. Exptl. Therap.*, **106**, 253 (1952).]

Studies of cerebral blood flow (I) and cerebral metabolism (II) were made on 10 human alcoholics. (I) was decreased about 15 per cent. after ingestion of a moderate dose of alcohol, but the significance of this decrease is questionable. (II) changes were in line with the decrease in (I). Antabuse alone decreased (I), and (II) was altered accordingly. Alcohol in the presence of antabuse significantly raised (I). The brain metabolizes MeCHO, as was indicated by a difference in arterial and venous cerebral blood content. Evidence was obtained that antabuse interferes with the metabolism of MeCHO by human brain *in vivo*.
L. E. GILSON (Chem. Abstr.).

Massive Picrotoxin Therapy in Treatment of Acute Barbiturate Poisoning. *Newman, E. A., et al.* [*Arch. Internal. Med.*, **81**, 690–5 (1948); *Abstracts World Med.*, **6**, 19 (1949).]

Some 30 cases are reported. The chief drug used was picrotoxin (I) in an average dose of 15 mgm. intravenously every 15 minutes until muscular twitching just below the convulsion level occurred. There is no arbitrary dose of (I) in acute

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barbiturate poisoning, and there is no toxic dose until the preconvulsive stage is reached. (I) acts mainly on the lower brain centers, and amphetamine is a valuable adjuvant to stimulate the cortex. W. C. TOBIE (Chem. Abstr.).

The Anticonvulsive Action of Several New Derivatives of Barbituric Acid. Hofmann, H., et al. [*Pharm. Zentralhalle*, **91**, 273–80 (1952).]

Six new derivatives of barbituric acid are tested for their anticonvulsive activity on the white rat injected with pentamethylenetetrazole. 5-Methyl-5-(methylpropylamino) barbituric acid, m. 169–71°, SLD (protective dose against convulsions) 100 mgm./kgm.; 5-methyl-5-(methylisobutylamino) barbituric acid, m. 215°, SLD 25 mgm./kgm., has weak, long-lasting narcotic action; 5-methyl-5-(methylbutylamino) barbituric acid, m. 179–81°, SLD 60 mgm./kgm., weak narcotic action; N-isopropyl-5-methyl-5-ethylbarbituric acid, m. 134–50°, SLD 20 mgm./kgm., a weak narcotic action; N-isopropyl-5, 5-diethylbarbituric acid, m. 112–14°, SLD 20 mgm./kgm.; and 5-ethyl-5-crotylbarbituric acid, m. 111–12°, SLD 7 mgm/kgm., sol. in H_2O (15°) 1 per cent. The last compound is much superior to luminal. K. SCHOEN (Chem. Abstr.).

Influence of Anticonvulsant Medication on Hyperthermia Produced by 2, 4-Dinitrophenol in the Guinea-pig. Frommel, E., et al. [*Helv. Physiol. et Pharmacol. Acta*, **10**, 288–91 (1952).]

Phenobarbital at the time of its maximum anticonvulsant action augments the hyperthermia induced by dinitrophenol. This action is probably connected with a decrease in pulmonary ventilation. Phenylacetylurea and phenylethylacetylurea, which have no influence by themselves on the respiratory exchanges, also augment the hyperthermia, possibly through the sympathicomimetic effect which they have on cerebral stimulation in guinea-pigs injected with dinitrophenol.
L. E. GILSON (Chem. Abstr.).

Temperature and Electroshock; Influence of Anticonvulsant Medication. Frommel, E., et al. [*Helv. Physiol. et Pharmacol. Acta*, **10**, 292–4 (1952).]

Oral administration of phenylethylurea 3 hours previously prevents the drop in body temperature during an electroshock crisis in guinea-pigs. It also prevents the increases in blood Ca, inorganic P, and sugar, the decrease in plasma alkali reserve, and the changes in the hemogram and blood concentration which accompany the electroshock. L. E. GILSON (Chem. Abstr.).

The Nature of Narcosis. Leipold, W. [*Deut. Dentist Z.*, **4**, 138, 140–2 (1950); *Chem. Zentr.* (**11**), 194 (1950).]

After a definition of narcosis and a discussion of its history and theory, etc., the manner of action of the following is described: $CHCl_3$, ether, ethylene chloride, N_2O, C_2H_4, narcylene, avertin, pernoston, evipan, and eunarcon.
M. G. MOORE (Chem. Abstr.).

Convulsive and Relaxant Agents for Muscles, and Phosphatase Activity of Serum. Inoue, K. [*Kagaku* (*Science*), **22**, 363–4 (1952).]

The intravenous injection of several drugs, which cause muscle convulsion and relaxation, such as myanesin 50 mgm., *d*-tubocurarine chloride 0·2 mgm., strychnine 0·1 mgm., or guanidine 150 mgm. per kgm. body weight does not have any significant effect upon the activities of acid and alkaline phosphatases in rabbit serum. T. KOBAYASHI (Chem. Abstr.).

Factors Affecting the Potentiation of Barbiturate Action by Tetraethylthiuram Disulfide. Graham, W. D., et al. [*J. Pharm. Pharmacol.*, **4**, 615 (1952).]

Neither Na ascorbate nor reduced glutathione will decrease the potentiating effect of tetraethylthiuram disulfide (I) on barbiturate-induced narcosis in rats. Repeated administration of (I) failed to make rats more sensitive to Na cyclural than did a single dose. S. W. GOLDSTEIN (Chem. Abstr.).

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Effects of Liver Injury and Nephrectomy on the Anticonvulsant Activity of 2, 4-Oxazolidinediones. Swinyard, E. A., et al. [*J. Pharmacol. Exptl. Therap.*, **105**, 365 (1952); *cf. C.A.*, **46**, 6740*f*.]

Trimethadione and paramethadione were tested at various time intervals after intraperitoneal injection in normal, liver-injured, and nephrectomized rats for ability to prevent the tonic extensor component of maximum electroshock seizures. Liver injury (CCl_4-induced) increased the potency and duration of action of both drugs. Bilateral nephrectomy increased the effect of paramethadione only. These results suggest that trimethadione is destroyed principally in the liver, while both liver and kidney are concerned in the degradation and elimination of paramethadione. L. E. GILSON (Chem. Abstr.).

Changes in Glucose Tolerance as an Index of Improvement after Electroshock Therapy. Altschule, M. D., et al. [*Proc. Soc. Exptl. Biol. Med.*, **80**, 436 (1952).]

Changes in sugar tolerance varied in direction and degree; there was no correlation with clinical improvement of the psychotic patients.

L. E. GILSON (Chem. Abstr.).

Demonstration of Functional Changes in the Central Nervous System after Electrically and Chemically Induced Convulsions. Kewitz, H., et al. [*Arch. exptl. Pathol. Pharmakol*, **215**, 93 (1952).]

Convulsions in groups of rats elicited by electric stimulation effective in all controls were partially inhibited by a single oral dose of 200 mgm./kgm. β-hexachlorocyclohexane (I) while the α, γ and δ-isomers were ineffective. Maximum inhibition of convulsions was observed 4 days after (I), and no effect remained after 10 days. When 100 mgm./kgm. of γ-hexachlorocyclohexane was given 4 days after the 200 mgm./kgm. of (I) inhibition of convulsions was noted as long as 21 days. Control experiments with phenobarbital showed maximum inhibition in 2 hours and with diphenylhydantoin in 1·5–2 hours. Electroshock did not inhibit the effect of a similar shock 15 minutes later but increased the threshold for metrazole, picrotoxin, and coramine convulsions. KARL F. URBACH (Chem. Abstr.).

A New Thiobarbiturate for Anesthesia. G. Colucci. [*Minerva med.*, **39** (11), 570–3 (1948); *Abstracts World Surg., Obstet. and Gynaecol.*, **5**, 300 (1949).

The new intravenous anesthetic thionarcon (I) (5-ethyl-5-(butylmercapto)-methyl)-2-thiobarbituric acid) shows a wide margin between the hypnotic and lethal doses and probably acts on the respiratory center and the sleep center in the hypothalamus. Results in a number of cases (given in detail) indicate that a maximum of 2 gm. of (I) can be given with safety, owing to its rapid breakdown.

W. C. TOBIE (Chem. Abstr.).

Side Effects of Curare and Synthetic Muscle Relaxants and their Therapeutic Abolition. Kootz, F. [*Anaesthetist* (**1**), 13–15 (1952).]

Intravenous injection of 60 mgm. of flaxedil or 12 mgm. of curare in 12 human volunteers increased clotting time and antithrombin titer significantly during the period of muscle relaxation. Protamine sulfate (3 mgm. of 1 per cent. solution, intravenous) promptly returned the clotting mechanism to normal; presumably by counteracting liberated heparin. Head-drop doses of curare and flaxedil increased a fluorescein excretion into the chamber fluid of rabbit eyes, which effect could be counteracted by antistine. This action is presumably by counteracting the effect of liberated histamine on the capillaries. KARL F. URBACH (Chem. Abstr.).

Tetanal Intoxication and Metabolism of Central Nervous Tissue. II. Activity of Diphospho pyridinenucleotide- and triphospho pyridinenucleotide-splitting Systems in the Brain of Mice Intoxicated with Tetanal Toxin. Asakura, S., et al. [*Med. J. Osaka Univ.* 2, *No.* 3, 35–42 (1951) (*in English*); *cf. C.A.*, **46**, 4108*f*.]

Diphosphopyridinenucleotidase activity, measured by the method of Mann and Quastel, dropped to about 50 per cent. of normal in brains and livers of mice showing general tetanus as a result of injection of a culture filtrate of *Clostridium tetani*. The toxin had no effect on activity of the enzyme *in vitro*. Brains from intoxicated mice showed normal triphosphopyridinenucleotidase activity.

FRED. H. SNYDER (Chem. Abstr.).

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Study of Experimental Habituation to Morphine. (I) Review and Morphine Habituation in the Rat. Fichtenberg, D. G. [*Bull. Narcotics, U.N., Dept. Social Affairs* 3, *No.* 3, 19–42 (1951).]

The rat (but not the mouse) became tolerant to morphine (I) on repeated dosage and tolerant rats withstood, subcutaneously, amounts of (I) 400 per cent. greater than normal rats. The increase in tolerance was developed very rapidly. Tolerant rats withstood daily subcutaneous doses of (I) 100 per cent. greater than the normal LD_{50} dose for 4 or 5 weeks. The LD_{50} subcutaneous/LD_{50} intravenous was 2·5 for normal rats and 4·8 for habituated rats, suggesting that the habituated rat converts (I) to a less toxic compound more rapidly than the normal. The analgetic effect of (I) decreased after the 3rd day of treatment and disappeared after 15 days. Habituated rats showed no cross tolerance to evipan or soneryl. The habituated rat withdrawn from (I), ceased to tolerate previously non-toxic high doses of (I) on the 5th day and the analgetic effect of the initial dose of (1) returned after 25 days. The rat became tolerant to heroin (II) on repeated dosage and tolerant rats withstood, subcutaneously, 10-fold greater amounts of (II) than normals. The analgetic effect of (II) decreased. The LD_{50} subcutaneous/LD_{50} intravenous was 2·5 for normals and 14·2 for habituated rats. Rats habituated to (II) showed no cross tolerance to evipan.

(*II*) *Biological Assay of Morphine and Oxydimorphine.* [*Ibid., No.* 4, 16 (1951).]

To 5 to 6 gm. of blood, urine or feces, or organs homogenized with sand add 7 volumes of 1:1,000 HCl absolute alcohol, reflux 3 hours, cool, and filter. Evaporate the filtrate in a stream of air, dissolve the residue in 5 c.c. 0·005 N HCl, filter, adjust the filtrate to pH 6·3 with NaOH and make to 10 c.c. The extract is assayed for total (I) and Oxydimorphine (III) on the denervated leech muscle, by evaluating the decreased response to bromocholine chloride. (I) but not (III) in mixtures is measured by evaluating the potentiation of the acetylcholine response of the denervated leech muscle.

(*III*) *Fate of Morphine in the Organism.* [*Ibid.*, 28–52.]

(III) was not found in tissues of normal or habituated rats after administration of (I). Liver sections of normal and habituated rats conjugated 37 to 50 per cent. of added (I); conjugated (I) being represented by the difference in amount of (I) obtained from hot alcohol-HCl extraction with refluxing and cold alcohol-HCl extraction. Brain sections conjugated 20 to 30 per cent. of added (I). Muscle did not conjugate (I). Conjugated (I) was recovered from liver, kidney, brain, stomach, intestine, and muscles of normal and habituated rats and from the spleen of habituated rats only. The percentage of total (I) in blood and tissues was higher 20 minutes after injection in habituated rats than normals and was identical 1 hour or 24 hours after injection. Free (I) was identical for both groups killed 15 to 60 minutes after injection. In general, (I) disappeared or was transformed more rapidly in habituated rats than in normal rats. RICHARD F. RILEY (Chem. Abstr.).

Convulsion Therapy of Psychotics by Injection of Restamin into Carotid Artery. Yano, M. (*Jiundo Hosp., Tokyo.*) [*Med. and Biol.*, **23**, 14 (1952).]

Injection into carotid artery of restamin, an antihistaminic agent, of 2 mgm/kgm. body weight, provoked convulsions as well as over-excitation of parasympathetic nerves of the injected side of the face. Several repeats of this treatment was effective in the therapy of psychotics. M. NAKAMURA (Chem. Abstr.).

Pharmacology of Antabuse. Harper, J., et al. [*Lancet*, **263**, 144 (1952).]

Mild toxic side effects from the drug without an increased blood-AcH content occurred among 36 male patients before any alcohol was given. When a test dose of alcohol was given after the patient had received 1 gm. antabuse daily for 4 days, the severity of symptoms corresponded with the blood-AcH level in 75 per cent. of the cases. Severe reaction (fall in systolic and diastolic pressure and other signs of vasomotor collapse) occurred with an AcH level above 0·5 mgm./100 ml.
BARBARA R. MURRAY (Chem. Abstr.).

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