# Part III.—Bibliography and Epitome.*

AN attempt is being made to provide as far as possible a complete bibliography compiled from the specialist journals dealing only with Psychiatry and Neurology (which are really inseparable) and their ancillary subjects, psychology, anatomy of the nervous system, criminology, etc.

A number of titles may appear to have a very remote relation to psychiatry, but they are included for the sake of completeness.

If any reader can add the names of journals to the following list, which it is hoped to publish each year in the January number, the addition will be gratefully received and acknowledged.

Those journals which are available in the Library of the Royal Medico-Psychological Association are marked " 1," those available in the Library of the Royal Society of Medicine are marked " 2," those in the Library of the British Psychological Society are marked " 3," and those in the Library of the British Medical Association are marked " 4."

The titles of these journals are mostly in the form given by the Board of Editors of Publications of the American Psychological Association, January, 1939. Contributors are requested to use the exact form given below.

## PSYCHIATRIC JOURNALS.

*Abh. Neur. Psychiat.*
*Abh. Psychother.*
*Acta Española Neur. y Psiquiat.*
2, 4 *Acta Psychiat. et Neurol.*
3 *Acta Psychol., Hague.*
*Acta Psychol., Keijo.*
*Aliéniste Français.*
2 *Allg. Ztschr. f. Psychiat.*
*Altersprobleme.*
*Am. Imago.*
*Am. J. ment. Def.*
2, 3 *Am. J. Orthopsychiat.*
1, 2, 3, 4 *Am. J. Psychiat.*
2, 3 *Am. J. Psychol.*
*An. Bras. Hig. Ment.*
*An. Istit. Psicol. Univ. B. Aires.*
*An. Psicotec., Rosario.*
*Anal. Inst. Neurol. Montevideo.*
*Analele Psihol. (Rumania).*
*Anales de psicologia, Buenos A.*
2, 3 *Année Psychol.*
2 *Ann. Méd. Psychol.*
2 *Ann. Osp. psichiatri., Perugia.*
*Arb. Psychiat. Inst., Sendai.*
*Arch. Anthrop. crim.*
2 *Arch. Argent. de Neurol.*
*Arch. Argent. Psicol. norm. pat.*
2 *Arch. Bras. de Neur. e Psiquiat.*
*Arch. Brasil Hig. Ment.*
*Arch. Chilenos de Crim.*
*Arch. Ital. di Studi Neuropsich.*
1, 2 *Arch. Neurobiol.*
4 *Arch. Neurol., Paris.*
*Arch. de Neurol. de Bucarest.*
1 *Arch. de Neurol.*
2 *Arch. di Antropol. Crim.*
*Arch. di Criminol. Neuropsiquiatria y Disciplinas Conexas, Quito.*
2, 4 *Arch f. Psychiat.*

* A number of abstracts in this section are reproduced from *Chemical Abstracts and Psychological Abstracts*. To the Editors of these two Journals we extend our grateful thanks.

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2, 3 *Arch. ges. Psychol.*
*Arch. Internationale de Neurol.*
*Arch. Ital. Psicol.*
*Arch. Krim. Anthrop.*
1, 2, 3, 4 *Arch. Neurol. Psychiat.*
*Arch. Neur. Psychiat., Mex.*
2, 3 *Arch. Psicol. Neurol. Psichiat. e Psicoter.*
2, 3 *Arch. Psychol., Genève.*
3 *Arch. Psychol., N.Y.*
*Arch. Relig. psychol.*
*Arch. Speech.*
*Arquivos da Assist. a Psicopatas de Pernambuco.*
*Arq. de Neuro-psiquiat., Brasil.*
3 *Austr. J. Psychol. Phil.*

*Beih. Z. angew. Psychol.*
*Beih. Zbl. Psychother.*
*Bol. Inst. Psiquiatria, Rosario.*
2, 4 *Brain.*
3 *Brit. J. Educ. Psychol.*
2, 4 *Brit. J. Inebriety.*
2, 3, 4 *Brit. J. Med. Psychol.*
2, 3, 4 *Brit. J. Psychol.*
3, 4 *Brit. J. Psychol. Monogr. Suppl.*
*Bull. Canad. Psychol. Ass.*
2, 4 *Bull. de la Soc. de Psychiatrie de Bucarest.*
2 *Bull. de la Soc. Roumaine Neur. Psychiat. Psychol. Endocrin.*
*Bull. du Groupement Français d'études de neuro-psychopathologie infantile.*
2 *Bull. Los Angeles Neur. Soc.*
*Bull. Menninger Clin.*
*Bull. Soc. Psihol. med., Sibiu.*

4 *Canad. Journ. Occup. Ther.*
3 *Cath. Univ. of Amer. Studies in Psychol. and Psychiat.*
2 *Cervello.*
2, 3, 4 *Character and Per.*
2 *Child Develpm.*
*Child Develpm. Abstr.*
*Child Develpm. Monogr.*
*Child Study.*
*Chin. J. Psychol.*
3 *Comp. Psychol. Monogr.*
2 *Confinia Neurol.*
*Contr. del Lab. di Psichologia.*
*Contr. psychol. Theor.*
*Criança Portuguesa.*

2 *Deutsche Ztschr. f. Nervenh.*
*Dis. Nerv. Syst.*

*Educ. psychol. Measmt.*
1, 2, 3, 4 *L'Encéphale.*
2 *Epilepsia.*
*Evolut. Psychiat.*

*Fiziol. Th. S.S.S.R.*
2 *Folia Neuropath. Esthon.*
2 *Folia Psychiat. et Neurol. Japonica.*
2 *Fortsch. Neur. Psychiat.*

3 *Genet. Psychol. Monogr.*
*Giornale di Psich. e di Neuropat.*

4 *Hum. Factor.*
2, 3, 4 *Hyg. Ment,*

*Illinois Psychiat. J.*
*Index Neurol. y Psiquiat., Buenos Aires.*

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3 *Indian J. Psychol.*
*Industr. Psychol.*
*Industr. Psychotech.*
1, 2, 3, 4 *Int. J. Psychoanal.*
2, 3 *Int. Z. Psychoanal. u. Imago.*

*Jap. J. appl. Psychol.*
*Jap. J. Exp. Psychol.*
*Jap. J. Psychol.*
1, 2, 3 *J. Abnorm. Soc. Psychol.*
*J. Am. Soc. Psychic. Res.*
3 *J. App. Psychol.*
2, 4 *J. Belge Neur. Psychiat.*
2 *J. Comp. Neur.*
1, 3 *J. Comp. Psychol.*
*J. Consult. Psychol.*
*J. Crim. Law and Criminol.*
*J. Crim. Psychopathol.*
*J. de Psychiatrie Infantile.*
3 *J. Educ. Psychol.*
*J. Except. Child.*
3, 4 *J. Exp. Psychol.*
2 *J. f. Psychol. u. Neurol.*
3 *J. Gen. Psychol.*
*J. Genet. Psychol.*
*J. Juvenile Res.*
1, 2, 3, 4 *J. Ment. Sci.*
1, 2, 3 *J. Nerv. Ment. Dis.*
1, 2, 3, 4 *J. Neurol. Neurosurg. Psychiat., London.*
*J. Neuropath. and Psychiat., Leningrad.*
1 *J. Neuropath. Ex. Path.*
2, 4 *J. Neurophysiol.*
*J. Neuropsychiatrique du Pacifique.*
*J. Neurosurg.*
*J. Parapsychol.*
*J. of Psychical Research.*
*J. Psihoteh.*
2 *J. Psycho-Asthenics.*
3 *J. Psychol.*
*J. Psychol., Moscou.*
*J. Psychol. Neurol., Leipzig.*
2 *J. Psychol. Norm. Path.*
*J. Soc. for Psychical Research.*
3 *J. Soc. Psychol.*
*J. Speech Disorders.*

*Kriminalistik.*
3 *Kwart. Psychol.*

*Mag. psychol. Szle.*
*Ment. Health.*
*Ment. Health Obs.*
2, 4 *Ment. Hyg., Lond.*
*Ment. Hyg., N.Y.*
*Ment. Hyg. Rev.*
*Ment. Hyg. Bull., Indiana.*
3 *Mind.*
*Mschr. Krim. Biol.*
2 *Mschr. Psychiat. Neurol.*

*Ned. Tijdschr. Psychol.*
2 *Neopsichiatrica.*
2 *Nervenarzt.*
3 *Neue psychol. Stud.*
*Neurbiol., Pernambuco.*
*Nevrasse.*
*Nevropath. i. Psikhiat.*
*Note e Riv. di Psichiat.*
*Nowiny Psychjaryczne.*
*Nuova Riv. di Clin. ed Assistenza Psichiatrica.*

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*Obshch. Klin. Nevropat.*
3 *Occup. Psychol.*
2 *Occup. Ther. and Rehabil.*
2 *Onderzoekingen Psychiat-Neur. Klin., Utrecht.*
2 *Ospedale Psichiatrico.*

*Person. J.*
*Pisani.*
*Polsk. Arch. Psychol.*
*Prace Psychol.*
*Proc. Amer. Assoc. Stud. Ment. Def.*
*Proc. A. Research Nerv. and Ment. Dis.*
*Psichotec.*
*Psicoterapia* (*Cordoba*).
*Psyche, Cambs., Mass.*
*Psychiat. Monogr.*
2 *Psychiat. en Neurol. Bl., Utrecht.*
2 *Psychiat. et Neurol. Japonica.*
2 *Psychiat. Neurol. Wchnschr.*
*Psychiatry.*
1, 2, 4 *Psychiat. Quart.*
2, 3 *Psychoanal. Quart.*
1, 2, 3 *Psychoanal. Rev.*
2, 3 *Psychol. Abstr.*
3 *Psychol. Bull.*
*Psychol. Clin.*
*Psychol. Exch.*
3 *Psychol. Forsch.*
*Psychol. Index.*
*Psychol. Monogr.*
*Psychol. Rec.*
3, 4 *Psychol. Rev.*
3 *Psychol. Rev. Monographs.*
*Psychol. Stud. Univ. Bp.*
*Psychol. wychow.*
3 *Psychometrika.*
*Psychometr. Monogr.*
*Psychosom. Med.*
*Psychosom. Med. Monogr.*

*Quart. J. Speech.*
*Quart. J. Stud. Alcohol.*

*Rass. Neurol. Veget.*
*Rass. Studi Psichiat.*
*Rev. Argent. Neurol. Psiquiat.*
*Rev. de Psicoanal., Argentina.*
*Rev. di Neur. e Psychiat., S. Paolo.*
*Rev. di Psiquiatria, Chile.*
*Rev. di Psiquiatria y Crim.*
3 *Rev. Franç. Psychoanal.*
*Rev. Ibero-Amer. de Anal. Biblio. de Neurol. y Psiquiat.*
*Rev. Mex. Neurol. Psiquiat.*
1, 2, 4 *Rev. Neurol.*
*Rev. Neurol. de Buenos-Aires.*
*Rev. Neurol. Psychiat., Praha.*
*Rev. Neuro-psiquiatr., Lima.*
*Rev. Oto-Neur.-Oftal. Cir. Neur. Sud-Am.*
*Rev. Neuropsiquiatr.*
*Rev. Psicol. Pädag.*
*Rev. Psihol.*
*Rev. Psiquiat. Crim., B. Aires.*
*Rev. Psiquiat., Uruguay.*
*Rev. Psiquiat. y Disc. Con., Chile.*
*Rev. Sudam. Psicol. Pedag.*
*Rev. Tchèque de Neurol. et de Psychiat.*
*Ricerca Psichica.*
2, 3, 4 *Riv. di Neurol.*
*Riv. di Neuro-psiquiatria, Peru.*
3 *Riv. di Psichologia.*
*Riv. Ital. di Endocrin. e Neurochir.*

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2 *Riv. Patol. nerv. ment.*
*Riv. Psichol. Norm. Pat.*
2, 4 *Riv. Sper. Freniat.*
1 *Rocznik Psychjatryczny.*
*Rorschach Res. Exch.*

1 *Schizofrenie.*
2 *Schweiz. Arch. Neurol. Psychiat.*
*Skand. Arch. Psychol.*
*Sovet. nervropatol., psichiatri, psichoguoguia.*
*Sovet. Psichoneurol.*
*Sovet. Psikhotekh.*
*Speech Monogr.*

*Tohoku Psychol. Folia.*
*Tr. Am. Neurol. A.*
*Tr. Beritov. Inst., Tiflis.*
*Tr. Kostchenko Ment. Hosp., Moscow.*
*Trud. fiziol. Lab. Pavlova.*
*Trud. tsentral. psikhonevrol. Inst.*

*Univ. Calif. Publ. Psychol.*
*Univ. Iowa Stud. Psychol.*
*Untersuch. Psychol. Phil.*

3 *Z. angew. Psychol.*
*Z. Arb. Psychologie.*
*Z. Berufsbildung des Pflegepersonals.*
*Z. Individ. Psychologie.*
2, 4 *Z. ges. Neurol. Psychiat.*
3 *Z. Pädag. Psychol.*
*Z. Parapsychol.*
*Z. Psych. Hyg.*
*Z. Psychoanalyse* (*Tokyo*).
3 *Z. psychoanal. pädag.*
2, 3 *Z. Psychol.*
*Z. Psychother. med. Psychol.*
*Z. Tierspsychol.*
2 *Zbl. Neurochir.*
1, 2 *Zbl. ges. Neurol. Psychiat.*
*Zbl. Psychotherap.*

## ACTA ESPAÑOLA NEUR. Y PSIQUIAT.

1940.

Psychopathological Modalities of Delirious Ideas. *Lopez Ibor, J.* . . . . No. 1

## ACTA PSYCHOL., HAGUE.

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Genetic Studies on the Comic. *Piret, R.* . . . . . 103

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Paraphrenia, Paranoia and Querulents. *Moura, J. de* . . . . . Nos. 3–4

## ARCH. INT. MED.

VOL. LXXIII. JUNE, 1944.

Late Cerebral Sequelae of Rheumatic Fever. *Bruetsch, W. L.* . . . . . 472

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Integration of the EEG. *Roberts, C. S., and Turner, W. J.* . . . . . 581
Physiologic and Clinical Aspects of EEG. *Putnam, T. J., and Hoefer, F. A.* . . . . 581

*Prolonged Insulin Coma in Treatment of Schizophrenia.*

An evaluation of the data in 51 cases of prolonged coma occurring in 38 patients during the course of insulin shock therapy leads to the assumption that some factor within the patients themselves, rather than the technic of treatment alone, was responsible for those prolonged comas.

A method suggested for the management of this complication is based on the experience gained in treating these patients, and the physiologic soundness of this method rests on a presented explanation of the mechanism of this complication. With emphasis on the dynamic nature of the action of insulin shock and insulin shock stupor progressing to the irreversible phase of prolonged coma and with free use of the contemporary literature, an attempt is made to explain the varying pathologic pictures in the light of progressive cellular hydration.

(Authors' abstr.)

*Fatal Circulatory Failure Caused by Electric Shock Therapy.*

There are probably at least two mechanisms by which fatal circulatory failure may be precipitated by electric shock therapy. In the first the acute cardiac dilatation and failure are

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caused by the tremendous muscular exertion of the convulsion. The likelihood of such an occurrence undoubtedly bears a direct relation to the severity of already existing heart disease. In the second the circulatory failure may occur without recognizable heart disease, and possibly results from the electrical stimulation of hyperexcitable central cardio-regulatory or vasomotor centers. The existence of this mechanism should be regarded as hypothetic rather than proved.

Of 3 patients who died of circulatory failure after electric shock treatment, 2 had advanced arteriosclerotic heart disease and the other acute myocarditis.

Electric shock therapy of persons with heart disease or of persons suspected of unusual susceptibility to disturbances of the central vasomotor or the central cardiac centres should not be undertaken without full realization of the potential hazard. (Author's abstr.)

VOL. LII. JULY.

Lesion in Peripheral Nerve Resulting from Compression by Spring Clip. *Denny-Brown, D., and Brenner, C.* . . . . . 1
Dystonia. III. Pathology and Conclusions. *Herz, E.* . . . . . 20
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*The EEG of Criminals. *Silverman, D.* . . . . . 38
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*Synthesis of Hippuric Acid in Dementia Praecox. *Michael, S. T., et al.* . . . . 57
*Abolition of Bulbocapnine Catalepsy in the Cat by Ac-tetrahydro-β-naphthylamine. *Kerman, E. F.* . . . . . 61
*Histamine Content of the Blood during Insulin Shock Therapy. *Billig, O., and Hesser, F. H.* . . . . . 65

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Functional Organization of the Medial Surface of the Primate Cortex. *McCulloch, W. S., and von Bonin, G.* . . . . . 81

PHILADELPHIA PSYCHIATRIC SOCIETY.

Newer Applications of Narcosis Therapy in War Psychiatry. *Palmer, H. D.* . . . 82
Clinical and Biochemical Observations on Intravenous Administration of Insulin in Shock Therapy. *Revitch, E.* . . . . . 83
Psychic Death. *Walters, M. J.* . . . . . 84

*The EEG of Criminals.*

In a study of the EEG of 411 prisoners, including " normal " criminals, psychotic and psychopathic criminals and offenders with " organic " disease, the most significant observation was the high percentage (75) of borderline and abnormal patterns among the psychopathic criminals. The EEG is a potentially important tool in the study and management of the psychopath, but not of the normal criminal. (Author's abstr.)

*Synthesis of Hippuric Acid in Dementia Praecox.*

Studies of the rate of synthesis of hippuric acid and of concomitant changes in the excretion of uric acid and in the levels of pyruvic and lactic acids in the blood of 18 schizophrenic patients and in 9 normal control subjects revealed that the average amount of hippuric acid synthesized in four hours was approximately the same in the two groups. Three schizophrenic patients synthesized amounts considerably less than the lowest value for the normal subjects, while 3 patients exceeded the range of the normal subjects. Unlike the normal group, several of the schizophrenic patients showed initial retardation of the rate of synthesis and 2 patients excreted unusually large amounts of hippuric acid during the first hour of synthesis. The ingestion of sodium benzoate caused a retention of uric acid which was slightly higher in the patient group. The synthesis of hippuric acid was accompanied by changes in the level of pyruvic acid of the blood. These changes were somewhat correlated with the intensity of the synthesis. (Authors' abstr.)

*Abolition of Bulbocapnine Catalepsy in the Cat by ac-tetrahydro-β-naphthylamine.*

A state of catalepsy was produced in cats by the administration of bulbocapnine. A state of excitement and stimulation of the sympathetic nervous system was produced by the administration of ac-tetrahydro-*B*-naphthylamine. The cataleptic state produced by bulbocapnine was completely abolished by the administration of ac-tetrahydro-β-naphthylamine. When bulbocapnine and ac-tetrahydro-β-naphthylamine were administered simultaneously in proper dosage, no symptoms characteristic of either drug appeared, the animal behaving in a normal manner throughout the period of observation. (Author's abstr.)

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*Histamine Content of the Blood during Insulin Shock Therapy.*

In a study of the histamine concentration in venous blood during various clinically recognizable phases of the insulin reaction in six psychotic patients receiving insulin shock therapy, it was found that values for histamine in whole blood before the administration of insulin corresponded closely to those reported by other investigators for normal persons. Early in the insulin reaction a slight rise in the histamine level of the blood was observed. The concentration of histamine increased, together with the depth of shock, late in the insulin reaction, reached its peak during the period of deepest shock, and returned toward normal after the reaction was interrupted by intravenous administration of dextrose. Values for the concentration of histamine with plasma, though less than those for whole blood, apparently fluctuated in similar fashion. The average maximal increase in the histamine concentration of the blood was 456 per cent. for all patients; the average maximal increase for patients in deep shock was 684 per cent. Changes in the histamine level corresponded to spontaneous fluctuations in depth of shock. Administration of a small amount of dextrose intravenously to one patient reduced the depth of shock and the histamine level concomitantly. Comparison of histamine levels during subshock, medium shock and deep shock revealed that the concentration of histamine with circulating blood apparently corresponded to the intensity of clinical shock observed during the insulin reaction. (Authors' abstr.)

## ARCH. PATH.

VOL. XXXVII. JUNE, 1942.

Encephalitis Complicating Virus Pneumonia. *Ingleby, H.* . . . . . 359
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Personality Traits of Jewish and Non-Jewish Students. *Shuey, A. M.* . . . No. 290

## BRIT. J. PSYCHOL.

VOL. XXXV. SEPTEMBER, 1944.

On Some Sound Values in English. *Orr, J.* . . . . . 1
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Penetrating Wounds of the Skull. *Patterson, G. H.* . . . . . 106
The Repair of Traumatic Cranial Defects. *Reeves, D. L.* . . . . . 112

*Electroencephalographic Changes Due to Cerebral Trauma.*

1. The place of electroencephalography in the study of head injury is similar to that of other laboratory procedures and, to be most useful, must be interpreted in the light of clinical manifestations.

2. In patients with cerebral "concussion" there is first a momentary absence of cerebral

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electrical activity, followed by runs of slow waves suggestive of cortical depression. The return of normal wave pattern is gradual during a ten- or fifteen-minute period immediately following the concussion.

3. Cerebral contusions of the coup-contrecoup type are often multiple and extensive. In these cases multiple lesions may give rise to a disturbed electrical pattern closely simulating diffuse cerebral dysrhythmia. If repeated tracings are taken during the course of recovery most of the lesions can be localized and their progress followed to complete clinical recovery, or a residual cortical defect may be demonstrated at a later date.

4. Extradural and subdural hemorrhages produce similar electroencephalographic changes characterized by low potential waves over the site of the lesion and slow waves of high voltage, and occasionally spikes at the margin of the lesion. The low potential activity has been considered due to short-circuiting of the waves by the accumulation of blood or fluid. In some other cases the low potential may be due to complete functional depression of cortical cells beneath the hematoma.

5. Gunshot wounds and other penetrating injuries of the brain may produce damage in areas far removed from the point of entrance of the missile. The extent of this distant damage may be well outlined electrographically.

6. Post-traumatic epilepsy may be due to irritation from a focal scar, or in other instances due to an inherent dysrhythmia in which the injury is merely a precipitating mechanism. It is possible in some cases to differentiate between the two types by electroencephalography.

7. Electroencephalographic study in doubtful cases may be of definite help in proving or disproving the presence of cerebral damage in minor head injuries. This type of study should be of value in industrial accidents where there is a question of compensation.

(Author's abstr.)

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*The Physical Examination of Two Thousand Cases of Neuroses.*

Of 2,288 consecutive admissions to a military hospital for neurosis, 3·4 per cent. had organic disease. The mean blood pressure of the remainder was found to be higher than accepted normal. The anomalous results of exercise tolerance test which were found are probably due to nervous factors. A list of the factors which may lead to the location of psycho-somatic symptoms is given. (Author's abstr.)

## J. NEUROPATH. EX. NEUROL.

VOL. III. JULY, 1944.

*Neurohistologic Findings in Experimental Electric Shock Treatment. *Winkelman, N. W., and Moore, M. T.* . . . . . 199
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*Reaction of Monkeys of Various Ages to Partial and Complete Decortication. *Kennard, M. A.* . . . . . 289

*Neurohistologic Findings in Experimental Electric Shock Treatment.*

Cats were given electro-cerebral shock; one group with the faradic current of Berkwitz, the other with the Cerletti-Bini type of house current. The experiments were conducted in close imitation of human electro-shock therapy, and in two animals excessive doses were given intentionally. The nutritional aspects of the animals were carefully controlled.

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Careful histologic studies of the brains and cords of all experimental animals were made, many on serial sections. No morphologic changes were observed in those animals receiving convulsive doses analogous to those given in humans. In the two animals receiving excessive electric shock doses, one small area of pericapillary hemorrhage was seen in one, and congestion of the smaller vasculature was observed in the other. In no case were subarachnoid hemorrhages or diffuse or extensive intracerebral hemorrhages encountered.

Explanations for the vascular changes reported in the literature in similar experiments are offered. (Authors' abstr.)

*Alterations in Brain Structure after Asphyxiation at Birth.*

Experimental Cesarean sections were performed under local anaesthesia in guinea-pigs at term. Asphyxia was induced by occluding the uterine or umbilical circulation of more than 70 fetuses; 50 litter-mate fetuses were delivered for controls without asphyxiation. Sixty-six of the newborn guinea-pigs were resuscitated and lived 30 minutes to 13 weeks. Brains of all were preserved and sectioned serially. A controlled method of staining with buffered (pH 4 to 5) solutions of thionin were used. This allowed exact comparison of staining qualities in control and experimental brains.

Multiple capillary hemorrhages and occasionally larger hemorrhages were found in all animals between 3 hours and 5 days after resuscitation, in half between 30 minutes and 2 hours and in none killed by asphyxia. Great variation in the amount of hemorrhage and in its distribution was encountered. Hemorrhage was not the primary cause of extensive neuron damage.

Edema appeared to be present in all but one brain between 8 hours and 4 days after resuscitation. After 5 days it had subsided.

Cytologic changes were observed in all specimens between one and one-half hours and 21 days after resuscitation.

The usual sequence of events was clouding of Nissl granule pattern starting between 1 or 2 hours, swelling of some neurons and shrinkage of others by 3 hours, loss of stainability with thiosin (pH4 to 5) starting at about 5 hours, typical chromatolysis by 1 or 2 days, complete lysis of neurons as early as 4 days, and hyperchromatic staining at least as early as 3 weeks. Some specimens showed generalized changes throughout most of the brain. Others were affected to a marked degree in rather circumscribed regions only. The cerebellum and corpus striatum were not markedly affected, while the thalamus, cerebral cortex, tegmentum and spinal cord were often severely damaged.

Asphyxia induced a transient microglia reaction between 2 days and 3 weeks. Thereafter glia assumed normal proportions.

Generalized or regional atrophy followed the loss of neurons in two-thirds of the severely asphyxiated and half the mildly asphyxiated guinea-pigs. As late as 13 weeks after resuscitation neurons of the experimental brains stained darker and were more shrunken than those of littermate controls. Areas of partial necrosis occurred in 8 animals. Loss of myelinated nerve fibers appeared to be secondary to cell loss.

It can be concluded that experimental asphyxiation followed by successful resuscitation of guinea-pigs at birth produced neuropathologic changes of variable degrees of severity in all specimens studied between one and one-half hours and 3 weeks after birth, and produced detectable permanent structural changes in more than half of those allowed to live more than 3 weeks. (Authors' abstr.)

*Reaction of Monkeys of Various Ages to Partial and Complete Decortication.*

The reactions following complete or partial decortication in infant and adult monkeys have been described and compared.

The decorticate adult has a motor performance limited to the simpler righting reflexes which alter the posture of its extremities as its position in space is changed. It is moderately spastic and relatively less motile than the decorticate infant.

The infant, decorticate during the first months of life, has a more complex pattern of motor performance. It rights, maintains standing posture and walks spontaneously. All the patterns of the performance, whether sucking, swallowing, vocalizing, clinging or progressing, are more complex and less stereotyped than those of the adult. There is no spasticity. The posture at rest is one of exaggerated flexion. One six-months-old animal, when decorticate, showed behavior intermediate between the above two. It was flaccid and maintained flexor posture. It was, however, unable to right and had the limited performance of the older animals. Removal of the following portions of the central nervous system of infant monkeys was seen to have some effect on reactions at that time, and hence indicates some cortical function at an early age: frontal association areas, areas 8, 6, 4, the parietal areas and the basal ganglia.

Destruction of large parts of the cortex at various ages has confirmed the observation that the infant cortex has a greater capacity for reintegration of motor activity than has the cortex of the adult.

Furthermore, the reorganization of the cortex injured in infancy always follows a pattern which is fixed both by the age of the animal and by the size and site of the lesion. (Author's abstr.)

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## J. NEUROPHYSIOL.

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*An Analysis of the Variability of Spinal Reflex Thresholds. *Denslow, J. S.* . . 207
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*An Analysis of the Variability of Spinal Reflex Thresholds.*

The threshold for reflex muscle contraction on applying moving pressure stimuli to spinous processes is relatively constant in given subjects. There are wide threshold differences among spinous processes in different subjects, and from segment to segment, and from side to side in the same subject. The evidence indicates that these differences might be due (i) to changes in the environment of deep pressure or stretch receptors in medium and high level areas and to that of other end-organs (particularly free nerve-endings) in low threshold areas, and (ii) to an imbalance of excitor-inhibitor influences, such as occurs in an enduring subliminal C.E.S. The mean threshold is slightly but significantly higher on the left side than on the right. No reason for this is evident.

In the absence of organic disease of neuromuscular and other systems, reflex threshold levels might provide a neurologic index of the organism's efficiency in coping with mechanical weaknesses and with environmental stresses. (Author's abstr.)

*Parasympathetic Regulation of High Potential in the EEG.*

If the inhibition of parasympathetic activity responsible for cardiac acceleration during hyperventilation is also responsible for concomitant slowing of the brain waves, section of parasympathetic pathways to the brain should increase hypocapnic effects.

The facial nerves containing parasympathetic pathways to the pial blood vessels were exposed in cats under ether. The animals were then paralysed with beta-erythroidin and given artificial respiration. Two minute periods of hyperventilation, which had no effect before sectioning the nerves, produced high potential, slow and spiked, activity after one or both facial nerves were cut. Stimulation of peripheral cut ends of the nerves reduced or prevented slow waves. Intravenous physostigmine in low concentration likewise prevented effects of hyperventilation, and atropine exaggerated effects previously counteracted by physostigmine. Atropine, in sufficient concentration, also produced high potential slow and spiked waves during hyperventilation without nerve section.

A parasympathetic influence on the electrical activity of the brain is demonstrated.

A cholinergic influence on cerebral mechanism and cerebral circulation is implied. A supplemental relationship between acetylcholine and carbon dioxide in homeostatic regulation of cerebral circulation is indicated. It is suggested that a neural mechanism is indicated by which emotionally induced, autonomic changes may produce "functional" effects on brain. (Authors' abstr.)

*The Afferent Path of the Pupillodilator Reflex in the Cat.*

The pupillodilatation resulting from sciatic, splanchnic and in some instances trigeminal nerve stimulation has been observed after various lesions of the spinal cord and brain in lightly anaesthetized cats. This response is known to result from inhibition of oculomotor constrictor activity; the present study adds the observation that destruction of parts of the brain above the oculomotor nucleus does not impair the effect, which is evidently completed at the midbrain level.

A common intramedullary pathway conveys afferent impulses from each of the sources studied to the midbrain. This pathway ascends through the lateral funiculus of the spinal cord, traverses the reticular formation of the medulla, gains a paramedian position in the dorsal pontile tegmentum, and ascends through the midbrain in or near the ventral central grey of the aqueduct. This oculomotor inhibitory pathway seems to be distinct from the lateral spinothalamic tract throughout its course, and pupillodilatation and pain evolved by stimulating afferent nerves would appear to bear only a coincidental relation to one another. (Author's abstr.)

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## 1. Biochemistry, Physiology, Anatomy, Pathology, Psychiatry, etc.

*EEG Studies of the Conditioning Mechanisms for Periodic After-images.* *Motokawa, K.* [*Iohoku J. Exp. Med.*, **40**, 48 (1941).]

While acting as subject in Mita's experiment, the author noted that after the alpha waves had been suppressed by a light stimulus, they went through a rather long period of recovery, during which time their amplitude fluctuated until they returned to their normal size. He noticed also that the after-image went through a similar period of fluctuation. He points out that Jasper and Cruikshank noted the same phenomenon. The present article is a further study of this fluctuation. In the curve of the Berger rhythm one notices two superimposed amplitudes, one of 8–15 sec. and one of 40–60 sec. The former coincides with the fluctuations of the after-images; the latter with that of winking. The alpha waves are, in general, suppressed in the after-image phase, and in their place appear delta waves. In the dark intervals between after-images there are alpha wave spindles, and these coincide satisfactorily with the after-images. The periodic after-images are ascribed to the rhythmic activity of the cortical visual center. T. V. Moore (Psychol. Abstr.).

*Action of Atropine on the Electroencephalogram.* *Markosyan, A. A.* [*Byull. Eksptl. Biol. Med.*, **13**, *No.* 3/4, 69–71 (1942).]

Intravenous injection of atropine leads to complete disappearance of waves of excitability of brain cortex. Hence it may be supposed that these waves are conditioned by changes of acetylcholine metabolism. G. M. Kosolapoff (Chem. Abstr.).

*The Action of $CO_2$ in Epilepsy and Schizophrenia on the Basis of Electric Findings on the Brain.* *Kornmüller, A. E.* [*Münch. med. Wochschr.*, **1**, 30 (1942); *Klin. Wochschr.*, **21**, 678 (1942).]

In certain cases of schizophrenia and epilepsy in which the cerebral bioelectrical findings (especially in the frontal region) reveal abnormally slow waves, a decrease in these variations could be produced by inhalation of a mixture of 5 per cent. $CO_2$ and 95 per cent. O. This effect of $CO_2$ is explained by its action on the cerebral circulation (in $CO_2$ excess the circulation would be increased, while in $CO_2$ deficiency, e.g. through hyperventilation, it would be decreased), as well as by the fact that $CO_2$ is an adequate stimulus for certain brain sections (especially vegetative). Finally, the role of $CO_2$ in respiration, especially tissue respiration, is considered. With respect to the quite prompt normalization of the bioelectrical findings produced by the $CO_2$, it is proposed to carry out therapeutic experiments with the administration of $CO_2$ in epilepsy, schizophrenia and other brain diseases. The possibility is discussed that the therapeutic success of metrazole and insulin treatment as well as of electric shock depends at least in part on the accumulation of $CO_2$, which appears in the brain and blood; it would also be conceivable that the success of the work and exercise therapy in various brain diseases depends in part on the same basis. Ruth Berggren (Chem. Abstr.).

*The Mechanism of Decurarization.* *Coppée, G.* [*Bull. classe sci. acad. roy. Belg.*, **27**, 452–67 (1941); *Chem. Zentr.*, **11**, 245 (1943).]

The effects of various procedures in the blocking of motor nerve-ends caused by substances similar to curare were examined in the isolated nerve-muscle preparations of *Rana temporaria* and *R. esculenta*. The following preparations were used: (1) Quaternary ammonium bases (tetramethyl-tetraethyl- and phenyltrimethylammonium salts, choline, acetylcholine, doryl, medoryl and mecholyl); (2) pyrimidine derivatives (piperidine, conine, arecoline, F933, sparteine, etc.); (3) tropine derivatives (atropine, hyoscyamine, duboisine, scopolamine, cocaine); (4) strychnine and brucine; (5) substances with undetermined structure (cobra venom, curare); (6) surplus of Ca ions in isotonic solution. The nerve blocking can be eliminated by (*a*) partial washing with a less concentrated solution of the curarizing substance; (*b*) summation of the stimuli affecting the motor nerve ends; (*c*) veratrine; and (*d*) in case of blocking with curare, by the use of eserine. Hilda H. Wheeler (Chem. Abstr.).

*Consumption of O by the Cerebrum in Toxic Shock.* *Charnyi, A. M., and Syrkina, P. E.* [*Byull. Eksptl. Biol. Med.*, **13**, *No.* 5/6, 13–16 (1942).]

In healthy dogs the O absorption in the brain is lower than in the muscle tissues. In early period of shock (histamine, peptone, insulin or metrazole) the brain O consumption rapidly rises, with increased formation of $CO_2$. G. M. Kosolapoff (Chem. Abstr.).

*Carbohydrate Metabolism of the Brain at the Early Shock Stage.* *Charnyi, A. M., Syrkina, P. E., and Krasovitskaya, S. E.* [*Byull. Eksptl. Biol. Med.*, **13**, *No.* 5/6, 17–21 (1942).]

In normal dogs glycogen and sugar are retained by brain tissue and utilized by it. Metrazole shock does not essentially change this activity. Both insulin and metrazole shocks are associated with definite decrease of glycogen content in the brain; apparently this is due to increased utilization rate of the stored carbohydrates. G. M. Kosolapoff (Chem. Abstr.).

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*Intracranial Tumors in Rats after 3,4-Benzopyrene.* *Baglioni, Tommaso.* [*Tumori*, **16**, 402–22 ; (1942) ; *Chem. Zentr.*, **1**, 2406 (1943).]

If a solution of benzopyrene in petrolatum is introduced into the cranial cavity of rats, intracranial tumors develop in a few of the animals. However, many animals are lost with this treatment. R. BEUTNER (Chem. Abstr.).

*K and Ca Content of Muscle and Nerve in the Different Stages of Neuromuscular Transmission.* *Lissák, K., and Kovács, T.* [*Arch. ges. Physiol. (Pflügers)*, **245**, 790–3 (1942) ; *Chem. Zentr.*, **11**, 336 (1943).]

In previous work the acetylcholine content and cholinesterase activity of the gastrocnemius muscle and sciatic nerve in stages 4 and 5 of neuromuscular transmission were determined. Under similar conditions the Ca and K contents of muscles and nerves were determined. It was found that there were no significant differences between the Ca and K contents of the stimulated and resting sides. By indirect stimulation the K content of the muscles was reduced, while the Ca was almost always increased. In stage 5 the K content of the muscles increased to the starting level, exceeding it in most cases. Besides the acetylcholine-cholinesterase system, K also plays an important role in the fatigue of the synapse and regeneration following prolonged stimulation. A. EDELMANN (Chem. Abstr.).

*Vitamin B Factors in Toxic Psychosis of Pregnancy and the Puerperium.* *Hart, B. F., and McConnell, W. T.* [*Am. J. Obstet. Gynecol.*, **46**, 304 (1943).]

Two patients who developed psychosis after toxic disturbances in pregnancy responded to therapy with vitamin B components. The niacin was probably the most important factor. This indicates that psychosis in pregnancy and the puerperium may in some cases be associated with disturbances in the vitamin B metabolism. RUTH BERGGREN (Chem. Abstr.).

*Colorimetric Determination of Nicotinic Acid.* *Teeri, Arthur E., and Shimer, Stanley R.* [*J. Biol. Chem.*, **153**, 307–11 (1944).]

The use of m-phenylenediamine as the chromogenic amine in the colorimetric determination of nicotinic acid has been found highly satisfactory if its addition to the nicotinic acid-cyanogen bromide solution is followed immediately by acidification with 20 per cent. HCl. Colour development is immediate, and the stability and intensity are both greater than is the case with most other amines. Colour or turbidity in extracts is accounted for by use of two blanks, the effects of which are additive. A. E. TEERI (Chem. Abstr.).

*Serological Studies of the Nucleoproteins of the Central Nervous System.* *Valenta, András.* [*Debreceni Tisza István Tudományos Társaság II, Osztályának Munkai*, 480–3 (1942) ; *Chem. Zentr.*, **1**, 747 (1943).]

Nucleoproteins (I) from different parts of the central nervous system of man and cattle were prepared. Serological tests identified the different sources of (I). The proteins are not species-specific, but are organ-specific. W. J. PETERSON (Chem. Abstr.).

*Nerve Conduction as an Effect of Mutual Influence.* *Schmidt, Otto.* [*Naturwissenschaften*, **30**, 644–5 (1942) ; *Chem. Zentr.*, **1**, 1076 (1943).]

Mutual influence exists between valence electrons which are in agreement with simple linkage and those which are not. This leads to regularities in chemical behavior governing many reactions. In the unsaturated molecule an unbound valence electron (present in the radical, formed by stimulation from a multiple bond) represents a place of disturbance. The simple bond next to this disturbance is reinforced, the one following is weakened. This alternation continues through the molecule with decreasing intensity. An interpretation of nerve conduction is given, based on this theoretical reasoning, explaining why a stimulus runs always in one direction only and why no two stimuli may travel in opposite directions simultaneously. In the polypeptides of the nerve fiber CO-CHR-NR groups follow periodically ; this causes the formation of periodically distributed disturbances in the chain able to transmit a stimulus through the whole chain. No places of disturbance exist in the protein chain in the original state, since the double linkages of C : O groups are not stimulated. If a ketonic group is transformed into an enolic group, the C : O group next to the new C : N or C : C linkage is in $\gamma$-position. The stimulation may jump over to this one and thus follow the whole chain. The direction from the first disturbance depends on whether the enol formation is effected from the first C : O group by migration of the methine H or the amine H.

A. E. MEYER (Chem. Abstr.).

*Role of Aneurine in the Cholinergic Neuro-effector Systems.* *Lissák, Kálmán.* [*Debreceni Tisza István Tudományos Társaság II, Osztályának Munkai*, 113–34 (1942) ; *Chem. Zentr.*, **1**, 856–7 (1943).]

The sensitivity to acetylcholine of the isolated heart of the guinea-pig, rat and frog was not increased by aneurine nor was that of hearts of animals deficient in vitamin $B_1$ or treated with vitamin $B_1$. The acetylcholine content of heart, transversely striated and small intestine musculature of $B_1$-avitaminotic animals was near normal, that of the central nervous system decreased and that of the sciatic nerve unchanged. The cholin-esterase content of the organs

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studied was decreased. The neuromuscular transmission of impulses and the appearance of fatigue and excitability of the muscles of animals deficient in vitamin $B_1$ remained unaltered.
MAURICE M. RATH (Chem. Abstr.).

*Myasthenia Gravis: A Consideration of its Causation in Fourteen Cases. Wilson, Andrew, and Stoner, H. Berrington.* [*Quart. J. Med.*, **13**, 1–18 (1944).]
Patients with myasthenia gravis are able to synthesize acetylcholine in sufficient amounts to produce the normal "muscarine" effects on glands and involuntary muscle. These effects are increased by administration of pilocarpine. The serum of patients not under prostigmine treatment, when tested on the isolated nerve-muscle preparations of the frog, produced a block in neuromuscular transmission caused by a substance partially soluble in alcohol. The serum cholinesterase activity of patients with myasthenia gravis was not increased; hence the muscular weakness is not due to rapid destruction of acetylcholine.
JOHN T. MYERS (Chem. Abstr.).

*The Production of Antibrain Antibodies in the Monkey. Koepfoff, Lenore M., and Kopeloff, Nicholas.* [*J. Immunol.*, **48**, 297–304 (1944).]
Homologous brain antibodies were produced in the rhesus monkey.
C. A. ZITTLE (Chem. Abstr.).

*Sudden Destruction of Motor End Plates by Lactic Acid. Carey, Eben J., and Massopust, Leo.* [*Proc. Soc. Exptl. Biol. Med.*, **55**, 194–7 (1944).]
Lactic acid in concentrations of 0·05–0·3 per cent. injected into the exposed zone of innervation of the sternomastoid muscle in white rats produced destruction of the motor nerve end plates in 0·5–30 minutes, depending on the concentration of the acid. Histological details are given.
L. E. GILSON (Chem. Abstr.).

*Experimental Brain Tumors. III: Tumors Produced with Dibenzanthracene. Arnold, Hildegarde, and Zimmerman, H. M.* [*Cancer Research*, **3**, 682–5 (1943); *cf. C.A.*, **36**, 1081⁹.]
Tumors were produced in 13 of 21 female C3H mice after implantation of pellets of dibenzanthracene in the right cerebral hemisphere. The tumors included 2 gliomas, 2 meningeal sarcomas, 8 extracranial fibrosarcomas and a squamous cell carcinoma of the scalp.

*IV: The Incidence in Different Strains of Mice. Zimmerman, H. M., and Arnold, Hildegarde,* [*Ibid.*, **4**, 98–101 (1944).]
The incidence of methylcholanthrene-induced tumors was not related to the tendencies of different mouse strains to develop spontaneous mammary tumors.
E. R. MAIN (Chem. Abstr.).

*An Apparatus for Grinding Small Samples of Brain Tissue. Terrill, H. M., and McKinstry, D. W.* [*J. Franklin Inst.*, **237**, 315–16 (1944).]
The apparatus consists of a stainless steel pestle fitting a pyrex test-tube. The head (diagram given) has shallow, rounded grooves cut spirally in such direction that the ground material is forced downward in the pyrex tube fitting the pestle. The pestle is used in the chuck of a small drill press, the drill press being ideal for the grinding, since it provides sufficient power for thorough grinding at low speeds, thereby avoiding local heating. The pyrex test-tube (chilled with its contents and pestle beforehand, if desired) is held by hand and occasionally raised and lowered to ensure grinding of all the brain tissue, to which a little sterile distilled $H_2O$ is usually added before starting. The instrument has proved safe and sufficient for homogenizing small quantities of brain tissue.
EDWARD B. SANIGAR (Chem. Abstr.).

*Phosphatase Activity in Chromatolytic Nerve Cells. Bodian, David, and Mellors, Robert C.* [*Proc. Soc. Exptl. Biol. Med.*, **55**, 243–5 (1944).]
Markedly increased acid phosphatase activity was found, by histochemical methods, in regions of the cytoplasm of nerve cells in which chromatolysis had been produced by axone section. The increase in activity was proportional to the degree of chromatolysis, and is thus probably associated with increased nucleoprotein synthesis or degradation.
L. E. GILSON (Chem. Abstr.).

*Oxidation of Fructose by Brain in vitro. Klein, J. Raymond.* [*J. Biol. Chem.*, **153**, 295–300 (1944).]
In the aerobic oxidation of fructose by cat brain in vitro, $O_2$ and inorganic phosphate disappeared. Resistance to acid hydrolysis of the major fraction of the organic phosphate suggested that phosphoglycerate had been formed. Lactate was formed in some, but not all of the experiments. Direct oxidation of triose phosphate is suggested as a step in the formation of lactate. Previous attempts to demonstrate lactate formation from fructose in brain probably did not succeed owing to the use of concentrated preparations. The reactions that account in part for the oxidation of glucose and fructose are the same.
HOWARD C. HOPPS (Chem. Abstr.).

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*The K and Ca Contents of Muscles and Nerves in the Various Stages of Neuromuscular Transmission of Stimulation. Kovács, Tibor.* [*Debreceni Tisza István Tudományos Társaság II. Osztályának Munkai*, 414–19 (1942); *Chem. Zentr.*, **1**, 858–9 (1943).]

Supermaximal stimulation of an ischiadic nerve for 30 minutes causes no change in the Ca and K contents in cats and rabbits as compared with the non-stimulated nerve. After indirect stimulation of the gastrocnemius the K content is lowered and the Ca increased. After restitution of the contractibility of the muscle the K content increases beyond the initial value. Next to the cholinesterase, the K content has a fundamental influence on fatigue and recovery in muscular stimulation. A. E. MEYER (Ch m. Abstr.).

*Neuromuscular and Ganglionar Transmission of Excitation. Lissák, Kálman.* [*Debreceni Tisza István Tudományos Társaság II, Osztályának Munkai*, 135–52 (1942); *Chem. Abstr.*, **1**, 859 (1943).]

After intra-arterial injections of acetylcholine (I) supplying the muscle or after injection into the ganglion the depressor effect in the second stage is prolonged; in the first and third stages the nerve endings are paralyzed, in the fourth a delay of fatigue is observed. In this latter stage the content of (I) is described in the nerve and increased in the muscle, while the cholinesterase activity (II) is reduced. In the fifth stage the (I) content of the muscle is decreased, the (II) enhanced. The K plays an important role also. Experiments on transmission of excitation in Waller's degeneration of the ischiadic nerve in the cat showed that during completely abolished neuromuscular transmission the current of nerve action retains almost its normal strength. The intensity of the currents of impulse remains unchanged even after prolonged stimulation. After prostigmine the muscles react to indirect single stimulation with rhythmic, to direct stimulation with single, contractions. A. E. MEYER (Chem. Abstr.).

*Brain Metabolism and Mental Deficiency. Himwich, Harold E., and Fazekas, Joseph F.* [*Am. J. Mental Deficiency*, **48**, 137–41 (1943); *cf. C.A.*, **36**, 3855[5].]

The difference in $O_2$ content of arterial blood entering and venous blood leaving the brain (A–V difference) was detected in 101 mentally deficient patients over 20 years old and in 123 under 20 years. In adult undifferentiated mental defectives with no anatomical, physiological or biochemical stigmata the A–V difference was normal, but in adult mongoloids, cretins and phenylpyruvic oligophrenics it was significantly below normal. In children with undifferentiated mongoloid mental deficiency the A–V difference was considerably below the normal adult level. With increasing age the A–V difference increased to the normal adult level in the undifferentiated defectives, but the increase was arrested before it reached the normal in mongoloids. WARREN M. SPERRY (Chem. Abstr.).

*The Inheritance of Certain Varieties of Mental Defect. Allen, W., and Halperin, S. L.* [*N. C. Med. J.*, **3**, 363 (1942).]

This article summarizes some special studies, including a few by the authors, which attempt to break down the "conglomerate mass of conditions, known to-day as mental deficiency, into component clinical entities with definite pathology in which process genetics may play an important role." Mongolism is significantly familial with an as yet unknown genetic factor and is not a result of defective thyroid functioning. Phenylketonuria and gargoylism are both conditions in which it has been found that mental deficiency is associated with abnormalities of bodily metabolism. It has been verified that the former is a result of the action of a single recessive gene, as is Tay-Sachs disease or amaurotic idiocy. Statistical studies suggest that gargoylism is likewise due to the action of a pathological gene, recessively inherited. R. W. BEEBE (Psychol. Abstr.).

*Thiamine Inactivation by the Chastek-paralysis Factor. Inhibition of Thiamine Inactivation. Sealock, Robert R., and Goodland Ruth L.* [*J. Am. Chem. Soc.*, **66**, 507–10 (1944).]

The destruction of thiamine (I) by the Chastek-paralysis factor of fish tissues (*cf. C.A.*, **37**, 4112[3]) is markedly decreased by the addition of Cu++, Zn and Fe+++; the percentage inhibition (concentration $1 \times 10^{-3}$ mol./l, unless otherwise given) is: $CuSO_4$ 50, $ZnCl_2$ 41·3 $FeCl_3$ 45·6, KCN 23·8, NaF 13·1, $ICH_2CO_2H$ 4·3, cysteine (0·1) 56·7, (0·01) 9·9, $Na_2SO_3$ (0·01) 9·5. Other common enzyme inhibitors could not be tested because they interfered with the determination of (I) by the diazotization method. Compounds resembling the (I) molecule in one or more features were also tested for inhibitory action. Among these, o-aminobenzyl-(II) and β-aminoethyl-4-methylthiazolium chloride were found to be strongly inhibitory. Substitution of the $NH_2$ group of these compounds by the $NO_2$ and phthalimido-groups respectively abolished the inhibition in the former and greatly reduced it in the latter. Other thiazoles and various 4-aminopyrimidines were either without effect or exhibited considerably lessened inhibition. With (II), the extent of the inhibition proved to be dependent upon the ratio of its concentration to that of (I). Use of the Lineweaver-Burk method (*C.A.*, **28**, 3092[1]) showed that (II) specifically inhibited (I) destruction by competing with the vitamin for the fish principle. The same method yielded a Michaelis constant of $0{\cdot}831 \times 10^{-4}$ mol./l for (I) and $0{\cdot}0197 \times 10^{-4}$ for (II). The data presented show that the fish principle behaves with inhibitors as do many enzymes; this may be interpreted as additional evidence of the enzyme nature of the fish principle. C. J. WEST (Chem. Abstr.).

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*The Cerebral Circulation in Altitude Collapse. Schneider, M.* [*Luftfahrtmed.*, **6**, 323 (1942); *Klin. Wochschr.*, **21**, 1088 (1942).]

The cerebral circulation in O deficiency was studied by measuring the arterial inflow, the venous outflow and the arteriovenous O difference of the cerebral blood; pressure changes in the cerebrospinal fluid were registered. The cerebral circulation increased in O deficiency, partly by increase in blood pressure, partly by dilatation of the cerebral vessels, which, however, did not begin until an equilibrium had been established toward the hypoxemia for 3–6 minutes. With increasing hypoxemia the dilatation of the vessels increased progressively according to the hyperbolic course of a concentration-activity curve, until at 4–5 per cent. O content of the inspired air the maximum of cerebral circulation generally possible was attained. The registration of the pressure of the cerebrospinal fluid showed changes corresponding to the increase in circulation, so that in precollapse pressure values up to 450 cm. water were obtained experimentally. For the complete suppression of the circulation of the cerebrum it was established that the reassumption of the function here could still be attained after 5 minutes, in the basal ganglia after 10–15 minutes and in the medulla oblongata after 20 minutes. In respiration of the O-poor air mixture the utilization first decreased corresponding to the increase in circulation; the O uptake of the encephalon was not measurably diminished. Upon lowering of the O tension in the venous blood of the brain a measurable decrease in the O uptake of the brain suddenly occurred conforming to a collapse which rapidly appeared. In the treatment of altitude collapse, in addition to giving O every means should be taken to raise the lowered arterial pressure. RUTH BERGGREN (Chem. Abstr.).

*Effect of Mental Activity on Metabolism. Jongbloed, J.* [*Nederland. Tijdschr. Geneeskunde*, **86**, 2012–17 (1942); *Chem. Zentr.*, **11**, 2050 (1942).]

Studies on four students showed that the metabolism as measured by $CO_2$ production rises about 4½ per cent. during reading a foreign language or during mental calculation.
MAURICE M. RATH (Chem. Abstr.).

*Experimental Bases of the Serological Demonstration of the Brain Lipoid in the Blood. Roeder, Fritz.* [*Klin. Wochschr.*, **21**, 1093–6 (1942).]

After the brain lipoids have entered the blood stream a fraction of the organ-specific brain antigen, which can be determined serologically, disappears from the serum but can be demonstrated in the whole blood, probably as a result of binding to the formed elements of the blood.
RUTH BERGGREN (Chem. Abstr.).

*Changes of the Brain Tissue During CO Poisoning. Nakamura, Hirosi, and Takami, Tosikatu.* [*Trans. Soc. Path. Japon.*, **29**, 456–61 (1939); *Chem. Zentr.*, **11**, 2172 (1942).]

CO poisoning inhibits the respiration of the brain of rabbits. This inhibition parallels morphological changes of the tissue. The permeability of the capillaries is increased, but can be reversed if the rabbits are made to breathe O. GERTRUDE E. PERLMANN (Chem. Abstr.).

*Behavior of Vitamin C in a Series of Nervous and Metabolic Ailments. I: Estimation of Ascorbic Acid in Blood with 2, 6-dichlorophenolindophenol. Nielsen, Otto Jul, and Hjarde, W.* [*Nord. Med.*, **10**, 1122–7 (1941).]

Sources of error in the determination are discussed.

*II: Is Ascorbic Acid the Provitamin of Vitamin C? Hjarde, W., and Nielsen, Otto Jul.* [*Ibid.*, 1127–8.]

Experiments show that ascorbic acid and vitamin C are identical. (Through *Chem. Zentr.*, **1**, 3532 (1941).) L. E. GILSON (Chem. Abstr.).

*Effects of Different Intakes of B-complex Vitamins on Neuromuscular Regeneration. Hines, H. M., Lasere, B., and Thomson, J. D.* [*Proc. Soc. Exptl. Biol. Med.*, **55**, 97–8 (1944); *cf. C.A.*, **37**, 1715.]

In rats neuromuscular regeneration was not appreciably effected by acute vitamin B-complex withdrawal, and was not enhanced by excessive intake of these vitamins. The neuromuscular weakness of regenerating and non-denervated muscles of rats on B-complex deficient diets was of the same order as that found in animals subjected to acute inanition or to food withdrawal while continuing B supplements. L. E. GILSON (Chem. Abstr.).

*Neuromuscular Disorders in Rats after Adrenalectomy and Their Origin. Chauchard, Paul, Masoué, H., and Chapolin, H.* [*Compt. rend. soc. biol.*, **137**, 207–8 (1943).]

In young rats adrenalectomy is followed by increases in central and peripheral motor nerve chronaxia. Muscle chronaxia is not increased. The intraperitoneal injection of 1 mgm. of desoxycorticosterone acetate restores the nerve chronaxia to normal and may decrease it to below normal. These effects are very similar to those observed in avitaminosis C.
L. E. GILSON (Chem. Abstr.).

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*Relation of the Hypothalamus to Respiratory Metabolism. Bloch, Walter.* [*Helv. Physiol. Pharmacol. Acta*, **1**, 53–78 (1943) (*in German*).]

A respiratory chamber for cats is described. The normal cat at rest utilized 510–770 c.c. of O per kg. per hour. Removal of portions of the middle part of the hypothalamus caused a decrease in metabolic rate, with body temperature sometimes remaining below normal for days at a time. In some cats the effect was permanent; in others a return to normal occurred after several weeks. Susceptibility to infection of the nasal passages was increased.

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

*Liberation of Thiamine by Stimulation of Peripheral Nerves. Liechti, A., Muralt, A. v., and Reinert, M.* [*Helv. Physiol. Pharmacol. Acta*, **1**, 79–88 (1943) (*in German*); *cf. C.A.*, **35**, 2957.[6]]

An elaborate electrical apparatus for recording the pulse rate of rats is described. With its aid preparations can be assayed for thiamine by its slowing effect on the rat heart. It was shown that electric stimulation of the isolated sciatic nerve of *Rana esculenta* appreciably increases its content of free thiamine (or thiamine phosphate). Acetylcholine is formed at the same time, but in much smaller amounts. Thiamine appears to play an inportant part in the metabolism of peripheral nerves.

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

*The Presence of Thiamine and Cocarboxylase in the Spinal Fluid. Abderhalden, Rudolf, and Elsaesser, Karl-Heinz.* [*Arch. ges. Physiol.* (*Pflügers*), **247**, 24–33 (1943).]

Normal and pathological cerebrospinal fluids contain from 0·2 to 1·5 per cent. of thiamine and from 0 to 0·5 per cent. of cocarboxylase. No correlations are found between various pathological processes and the contents of these substances. After the intravenous or intramuscular injection of thiamine, the latter appears in the cerebrospinal fluid. After the injection of 50 mgm. of thiamine, the total thiamine content of the spinal fluid increases about 25 per cent. after 3 to 5 hours, but returns to its normal level within 24 to 26 hours. When thiamine is injected into the cisterna magna it rapidly disappears from the cerebrospinal fluid, and hence there is no advantage in introducing it in this manner clinically.

ARTHUR GROLLMAN (Chem. Abstr.).

*Partition of Phosphorus in Mammalian Brains. Lahiry, N. L., Venkataraman, P. R., Anantakrishnan, C. P., and De, N. N.* [*Indian J. Med. Research*, **30**, 619–22 (1942).]

Total P in brains of goat, dog, rat, ox and rabbit ranged from 305 to 360 mgm. per 100 gm. moist tissue. The distribution of acid-soluble, inorganic and ester fractions labile and resistant to hydrolysis was roughly similar in the five brains.

M. H. POWER (Chem. Abstr.).

*A Parallelism Between the Quantitative Incidence of Carbonic Anhydrase and Functional Levels of the Central Nervous System. Ashby, Winifred, and Chan, Dorothea V.* [*J. Biol. Chem.*, **152**, 235–40 (1944); *cf. C.A.*, **38**, 1252[9].]

In the steer, dog, cat, hog, guinea-pig, chicken and man, the content of carbonic anhydrase in the cerebral tissue is approximately twice that in the cord. The larger amount present in the cerebrum is not due to the larger proportion of grey matter present, but may be due to differences in the amount of enzyme contained in the neurons entering into the composition of the respiratory areas. If this be so, it is probable that the carbonic anhydrase content might bear a relationship to the factors which cause the functional dominance of the cerebrum over the cord.

A. E. TEERI (Chem. Abstr.).

*Acetylation in Patients with Myasthenia Gravis. The Elimination of p-Aminobenzoic Acid. Torda, Clara, and Wolff, Harold G.* [*Proc. Soc. Exptl. Biol. Med.*, **55**, 86–90 (1944).]

After large or small oral doses, both healthy subjects and patients with myasthenia gravis excreted p-aminobenzoic acid in essentially the same manner with respect to time of elimination and amounts of free and acetylated forms. It is inferred that there is no serious insufficiency of acetyl radical in such patients.

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

*Similar Nervous Disturbances Due to CO Poisoning in Two Subjects of the Same Family. Silberschmidt, W.* [*Schweiz. med. Wochschr.*, **71**, 441–2 (1941); *Chem. Zentr.*, **11**, 231 (1941).]

Two patients from the same family fell to the left when walking, showed unsteady gait and disturbances in the left leg and foot in CO poisoning established by blood analysis. No vomiting occurred.

MAURICE M. RATH (Chem. Abstr.).

*Aspects of the Effects of Intravenous Injections of Acetylcholine on the Central Nervous System. Stavraky, Geo. W.* [*Trans. Roy. Soc. Can.*, **37**, *Sect. V*, 127–39 (1943).]

A study of the effects of intravenous injections of acetylcholine in unanesthetized cats having one or both frontal lobes aseptically removed. The injection of 0·05–0·2 mgm. into cats with one lobe removed 5–6 weeks previously produces characteristic tonic muscular seizures and unilateral sympathetic stimulation on the opposite side of the body. Larger doses produce generalized convulsions. Bilateral removal of the frontal lobes produces an animal that shows



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all signs of sympathetic excitation when acetylcholine is injected. Semidecerebrate cats are very sensitive to acetylcholine, and may be distinguished from frontal-lobectomized animals.
C. J. CARR (Chem. Abstr.).

*Acetylcholine Content of the Myenteric Plexus and Resistance to Anoxia. Welsh, John H., and Hyde, Jane E.* [*Proc. Soc. Exptl. Biol. Med.*, **55**, 256–7 (1944).]
It is shown that the estimated acetylcholine content of the myenteric plexus of rabbits and guinea-pigs (0·4–0·8 mgm./gm.) is higher than that of any other mammalian nervous tissue. The cells of this plexus are the most resistant of all nerve cells to anoxia. Thus further evidence is provided that indicates a relationship between resistance to anoxia and acetylcholine content of different parts of the nervous system. L. E. GILSON (Chem. Abstr.).

*Acetylcholine Content and Cholinesterase Activity of Muscles and Nerves in Different States of Neuromuscular Excitation Transfer. Nagy, E. Klara.* [*Debreceni Tisza István Tudományos Társaság II, Osztályának Munkai*, 443–51 (1942); *Chem. Zentr.*, **1**, 747 (1943).]
The acetylcholine (I) content of a nerve (frog or cat) irritated to complete fatigue is appreciably lower than the un-irritated nerve. (I) is increased in the irritated muscle and the cholinesterase activity (II) is decreased. If irritation is continued further, the nerve function and contraction capacity of the muscle are restored after 2–3 hours. The content of (I) in the nerve and muscle is then greater than in the resting state, while (II) is lower. (I) plays an important role in irritation transfer and cannot be considered a by-product of nerve metabolism.
W. J. PETERSON (Chem. Abstr.).

*The Amphomimetism of Acetylcholine. Research on the Nictitating Membrane of the Cat. Danielopolu, Daniel, and Marcu, Ion.* [*Compt. rend.*, **215**, 427–8 (1942).]
Additional evidence is submitted to support the authors' claim of the amphomimetic nature of acetylcholine (I). In an adrenalectomized, anesthetized cat with its cervical sympathetics sectioned, 1 mgm. of (I) produces a hypertension and contraction of the nictitating membrane if preceded by 1 mgm. eserine and atropine. In an adrenalectomized, cervical sympathectomized, anesthetized cat treated with cocaine and atropine, adrenaline and (I) produce a greater hypertension and contraction of the nictitating membrane than adrenaline alone. In an adrenalectomized cat (I) produces a contraction of the nictitating membrane after atropine. This action is sensitized by cocainization. A summary of earlier work on this subject is included.
A. EDELMANN (Chem. Abstr.).

*Acetylcholine Content and Cholinesterase Activity of Muscle and Nerve in Different Stages of Neuromuscular Transmission. Lissak, K., Nagy, E. K., and Pasztor, J.* [*Arch. ges. Physiol.* (*Pflügers*), **245**, 783–9 (1942); *Chem. Zentr.*, **11**, 335–6 (1943).]
Additional support for the chemical theory of transmission is furnished. Stimulation of muscles at a fixed frequency (400/second) by indirect current at regular intervals showed the following stages: (1) A prompt rise in potential; (2) subsidence of the same; (3) a new rise followed by a slow fall; (4) complete fatigue; (5) continued ascent of potential and recovery of contractibility. Acetylcholine (I) was determined in normal and stimulated motor nerves during stages 4 and 5. There was a continual decrease in (I) content from the start of excitation until the beginning of fatigue, but there was a subsequent rise, and at stage 5 it exceeded that of normal resting nerve. In the present work the relationship between (I) content and cholinesterase (II) activity in the phases of excitation was investigated. The (I) content and (II) activity of the gastrocnemius muscles and sciatic nerves of frogs and cats were determined at stages 4 and 5. After stimulation of the sciatic nerve with maximum condenser charge (at 60/second) the neuromuscular synapse went into full fatigue (stage 4). In this state the (I) content of the nerve was significantly lower than that of the unstimulated nerve from the opposite side. The (I) content of the correspondingly stimulated muscle was increased, the activity of (II) reduced. By continuing the stimulation in stage 4, the potential and contractibility were restored (stage 5) in 2 to 3 hours. In this phase the (I) content was equal to or somewhat higher than that of resting nerve. With prolonged stimulation, therefore, the relationships between (I) content and (II) activity are reversed. In stimulated muscle the (I) content is less, the (II) activity higher than in resting muscle of the opposite side. Control experiments show that the (I) content and the (II) activity of resting muscles and nerves from both sides of the animal are similar. There exists a relationship between the (I)–(II) system and the stage of neuromuscular excitation. (I) occupies an important place in the transmission of impulses, and in no case can it be regarded as a by-product of nervous metabolism. A. EDELMANN (Chem. Abstr.).

*Acetylcholine Content in Dog Blood in the Hunger Activity of the Gastro-intestinal Tract. Prokopenko, V. G.* [*Byull. Ekspti. Biol. Med.*, **13**, *No.* 3/4, 91–2 (1942).]
Both during digestive and hunger activity, there appear in dog blood substances which are capable of causing contraction of the dorsal muscle of the leech, which is the classical test for acetylcholine. G. M. KOSOLAPOFF (Chem. Abstr.).

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*Epileptoid and Shock Phases of the Syncope Syndrome and the Possible Role of Acetylcholine in their Genesis.* *Barnard, Robert D.* [*Ohio State Med. J.*, **39**, 907–12 (1943).]

The syncope syndrome in both its splanchnic and somatic manifestations can be explained on the basis of acetylcholine intoxication. A hypothesis relating psychogenic syncope to the excessive systematic liberation of acetylcholine is presented.

RUTH BERGGREN (Chem. Abstr.).

*Potentiation of the Action of Acetylcholine with Donaxine.* *Mashkovskii, M. D.* [*Byull. Eksptl. Biol. Med.*, **13**, *No.* 3/4, 87–90 (1942).]

Donaxine action cannot be considered to be similar to that of eserine, as shown by experiments *in vivo*. Donaxine does not inactivate cholinesterase; hence it should not be classed with eserine-type substances. G. M. KOSOLAPOFF (Chem. Abstr.).

*The Distribution of Cholinesterase in the Bovine Retina.* *Anfinsen, Christian B.* [*J. Biol. Chem.*, **152**, 267–78 (1944).]

The histological structure of the retina permits the isolation and study of cytologically homogeneous sections of this issue. Thus the retina is useful as a test organ for the study of the general physiology of otherwise inaccessible components of the central nervous system. By use of single horizontal sections of bovine retina both for quantitative histological analysis and for chemical study, it was determined that the enzyme cholinesterase has a predominantly synaptic localization. This provides direct support for the theory of chemical mediation of the nervous impulse. A. E. TEERI (Chem. Abstr.).

*The Cholinesterase of the Blood Serum.* *Werle, E., and Stuttgen, G.* [*Klin. Wochschr.*, **21**, 821–2 (1942).]

Pure callicrein preparations added to serum *in vitro* did not change the cholinesterase activity. Impure preparations contain a low-molecule, highly stable substance which activates the enzyme. Intravenous injection of callicrein in human beings had no effect on the esterase activity of the serum. Histamine can inhibit the cholinesterase in human serum to a very marked degree. The cholinesterase in the serum was inhibited by tetanus toxin, but such large doses were required that a specific action cannot be assumed. The esterase in 0·05 c.c. human serum was not affected by 3,300 toxin units; 13,000 units effected a 70 per cent. inhibition. The cholinesterase values in chronic constipation were distinctly above the normal. The blood cholinesterase was lowered in a 4-year-old boy born without thyroid glands.

RUTH BERGGREN (Chem. Abstr.).

*Inhibition of Cholinesterase Activity of Nervous Tissue by Eserine in vivo.* *Cantoni, G. L., and Loewi, O.* [*J. Pharmacol.*, **81**, 67–71 (1944).]

The acetylcholine present in the central nervous system and peripheral nerves of the frog (*Rana pipiens*) is completely or almost completely hydrolyzed during grinding and subsequent incubation of the tissues. Subcutaneous injection of eserine, even in large doses, does not affect this process in the case of the central nervous system, but almost completely protects the acetylcholine of the peripheral nerves. The eserine does not inhibit propagation of impulses in the peripheral nerves. L. E. GILSON (Chem. Abstr.).

*Changes of Cholinesterase Activity in the Nerve Fibers on the Poles of Direct Current.* *Minaev, P. F.* [*Byull. Eksptl. Biol. Med.*, **13**, *No.* 3/4, 77–8 (1942).]

Application to nerve fibers of the anode of d.-c. circuit causes an enhancement of cholinesterase activity; the cathode causes a decrease in the activity.

G. M. KOSOLAPOFF (Chem. Abstr.).

*Plasma Cholinesterase in Male and Female Rats.* *Mundell, Dorothy B.* [*Nature*, **153**, 557–8 (1944).]

The greater activity of the plasma of mature female rats towards acetylcholine, as compared with that of mature males and immature females, is due mainly to the activity of the pseudocholinesterase. E. D. WALTER (Chem. Abstr.).

*Determination of Protein in Cerebrospinal Fluid.* *Abelin, I.* [*Schweiz. med. Wochschr.*, **73**, 332–4 (1943).]

Trichloroacetic acid is added to 4 to 6 c.c. of cerebrospinal fluid, the washed precipitate separated centrifugally and the N content estimated by the micro-Kjeldahl method. Albumin N is estimated as the difference between the values for total N and globulin N, the latter being determined in the precipitate formed in strong $Na_2SO_4$ solution at 38°. The new method was compared with Kufka's method, in which the protein content is determined after precipitation with picric acid and was found to be more reliable and sensitive.

MAURICE M. RATH (Chem. Abstr.).

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*Vitamin C Content of the Cerebrospinal Fluid in Nervous and Mental Diseases. Horányi, B., and Láng, S.* [*Wiener med. Wochschr.*, **93**, 464–5 (1942).]

The cerebrospinal fluid of the cases studied contained 0·5–2·0 mgm. per cent. of ascorbic acid. The content varied with the age of the patient, the diet and the season of the year, but there was no definite correlation between it and the severity of the nervous disorder.

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

*Relation between the Ascorbic Acid Content of Serum and Cerebrospinal Fluid. Westerguard, Frode.* [*Nord. Med.*, **16**, 3536–8 (1942); *Chem. Zentr.*, **1**, 971 (1943).]

A linear relation was found between the content of reduced ascorbic acid of the serum and that of the cerebrospinal fluid, wherein the concentration in the latter was four times as great as in the former. In two persons with low ascorbic acid values in the cerebrospinal fluid (<0·4 mgm. per cent.) the attacks of vertigo were stopped by treatment with vitamin C.

MAURICE M. RATH (Chem. Abstr.).

*The System Ca-protein in Serum and Spinal Fluid. Lockhart, Carlos E.* [*Rev. facultad cienc. quim. (Univ. nacl. La Plata)*, **17**, 265–77 (1942).]

The content of Ca in the spinal fluid is higher than the content of ionized Ca in the serum. The differences are larger than can be attributed to the presence of protein in the spinal fluid.

A. E. MEYER (Chem. Abstr.).

*Thiocyanate Compounds in Dog Saliva and Cerebrospinal Fluid. Gubenko, V. K.* [*Byull. Eksptl. Biol. Med.*, **13**, *No.* 3/4, 102–3 (1942).]

Up to 3 mgm. per cent. of CNS compounds are found in dog saliva from the submaxillary glands; the gland excretion of CNS compounds weakens (or exhausts itself) rapidly. Artificial feeding of KCNS increases the content of CNS compounds in saliva. In the cerebrospinal fluid up to 2 mgm. per cent. of CNS compounds are found; these are not increased by feeding KCNS.

G. M. KOSOLAPOFF (Chem. Abstr.).

*Nicotinic Acid in the Cerebrospinal Fluid in Infancy. Verrotti, Irene.* [*Pediatria*, **51**, 1–9 (1943); *Chem. Zentr.*, **1**, 2312 (1943).]

In normal children nicotinic acid content of the cerebrospinal fluid is 15–35 $\gamma$ per cent., average 23·9 $\gamma$ per cent. In tetanus, meningitis and two cases of diencephalohypophyseal syndrome the content was decidedly higher.

R. P. E. HOPF (Chem. Abstr.).

## 2. Pharmacology and Treatment.

*Bromide Intoxication. Kay, Frank A., Smith, Donald, and Johnson, Georgia.* [*J. Med. Assoc. State Alabama*, **13**, 284–8 (1944).]

The ease and rapidity with which bromide becomes concentrated in the blood serum is demonstrated. The effectiveness of NaCl in hastening the elimination of bromide from the blood and tissues is shown graphically. The signs and symptoms of bromide intoxication are given.

RUTH BERGGREN (Chem. Abstr.).

*Influence of Some Narcotics on Activity of Blood Cholinesterase. Heim, Fritz, and Rohde, W.* [*Arch. exptl. Path. Pharmakol.*, **202**, 216–18 (1943).]

The activity of serum cholinesterase *in vitro* was inhibited 40 per cent. by addition of 0·05 per cent. of chloral hydrate to the serum, 25 per cent. by 0·1 per cent. of barbital, 20 per cent. by 0·1 per cent. of evipan-Na and slightly by 0·125 per cent. of phenobarbital-Na, $CHCl_3$, EtOH or tribromoethanol.

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

*Hypnotics and Their Action on the Thalamic Centers. IV: The Action of $MgSO_4$ on the Pancreas. La Barre, Jean, and Vesselovsky, Olga.* [*Arch. intern. pharmaco-dynamine*, **67**, 267–75 (1942); *cf. C.A.*, **36**, 2917$^7$.]

The intravenous injection of $MgSO_4$ causes a diminution of the response of the pancreas in dogs, and the lipase content of the collected juice is low. In vagotomized dogs the inhibition does not take place after section of the medulla.

*V: Effect of Na Barbital on Blood-sugar Regulation.* [*Ibid.*, 334–42.]

The intravenous injection of Na barbital into normal dogs causes a fleeting rise of the blood-sugar level, and in dogs anesthetized with chloralose the effect is similar but much less. Transfusion of blood from the pancreatic vein of a dog under chloralose into the jugular vein of a depancreatized dog causes a 30 per cent. fall in the blood-sugar level of the latter. If the donor dog is under chloralose and barbital (250 mgm./kg.) the fall is only 9 per cent. Thus the barbital reduces the secretion of insulin.

M. L. C. BERNHEIM (Chem. Abstr.).

*Effects of Narcotics and Benzedrine on Metabolic Processes in the Central Nervous System. Quastel, J. H.* [*Trans. Faraday Soc.*, **39**, 348–59 (1943).]

The action of a drug in interfering with the respiratory activity of the nervous system is to

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disturb functional activity, the disturbance being proportional to the degree to which the respiratory activity is affected. Narcotics at low concentrations have the power of inhibiting the respiration of brain tissue *in vitro*. By use of the brain slice technique with brain cortex, definite inhibitions of respiration take place in the presence of narcotics at concentrations which produce narcosis in animals. These effects are not irreversible with narcotics such as the barbiturates or chloretone, or hyoscine. The steady state of the diminished respiration of brain slices brought about by small concentrations of narcotics is found to be greatly dependent on the concentration of $K^+$ in the medium. If a narcotic enters into a mass action equilibrium with an enzyme with which the substrate oxidized by the brain is also combined, an increase in substrate concentration does not lead to a diminution in the inhibitive power of the narcotic. Narcotics at low concentrations do not affect the known dehydrogenases. The addition of benzedrine at low concentrations to brain cortex respiring in a glucose medium neither increases nor decreases the respiration, nor will it neutralize the diminution of respiration due to the presence of a narcotic. VICTOR R. DEITZ (Chem. Abstr.).

*The Effect of Narcotics on the Excretion of Free and Combined Ascorbic Acid. Ghosh, Baidya Nath.* [*Ann. Biochem. Exptl. Med.*, **2**, 221–8 (1942).]

Administration of chloretone, paraldehyde, phenobarbital, amidopyrine and bromobenzene caused increased elimination of free ascorbic acid in the urine of rats. The excretion of combined ascorbic acid was markedly reduced, especially in cases where chloretone, amidopyrine and bromobenzene were fed. Administration of paraldehyde, amidopyrine and bromobenzene and, to a certain extent, of chloretone influenced similarly the elimination of free ascorbic in the urine of guinea-pigs. Urethan caused increased excretion of both free and combined ascorbic acid. Phenobarbital caused decreased elimination of both free and combined ascorbic acid in the urine. A general depletion of tissue ascorbic acid was observed in guinea-pigs, even when they were able to excrete increased amounts of free ascorbic acid. Rats showed increases both in concentration of tissue ascorbic acid and of urinary free ascorbic acid.

BENJAMIN PRESCOTT (Chem. Abstr.).

*An Injectable Barbituric Acid Narcotic. Marri, Rodolfo.* [*Arch. famacol. sper.*, **70**, 199–21 (1940); *Chem. Zentr.*, **11**, 2584 (1941).]

The action of "Narkogen" (formula not given) is described.

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

*The Action of Some Barbiturate Derivatives (Barbital, Evipal, Narcogene, Eunarcon, Rectidon) on Smooth Muscle in vitro. Marri, Rodolfo.* [*Sperimentale*, **94**, 620–5 (1940); *Chem. Zentr.*, **11**, 3093 (1941).]

Surviving muscles of intestine and bladder of the rabbit are inhibited in movement and tonus by the barbiturates. Applied in advance they prevent the increased tonus produced by acetylcholine and $BaCl_2$. The effect of barbital is less pronounced than that of the other compounds. If applied following acetylcholine or $BaCl_2$ they abolish the increased tonus and lead to hypotonicity. The effects of the barbiturates are reversible. A. E. MEYER (Chem. Abstr.).

*Barbital Poisoning Treated with Amphetamine (Mecodrin) and Warm Baths. Frandsen, Sv.* [*Ugeskrift Laeger*, **106**, 33–5 (1944).]

A case of poisoning with 5 gm. of barbital leading to unconsciousness of four days' duration was treated with large doses of amphetamine (mecodrin). Hyperemia, which occurred as a consequence of the amphetamine treatment, was treated with warm baths (26° for 10 to 20 minutes). Frandsen discusses the use of sulfonamides in preventing pneumonia in such poisonings. Ten gm. of sulfathiazole, given in one case, produced a long-lasting polyneuritis.

GERTRUDE E. PERLMANN (Chem. Abstr.).

*Decolorizing Test Solutions for the Determination of Nicotinic Acid and Nicotinamide. Taufel, K., and Dahle, F.* [*Z. Untersuch. Lebensm*, **85**, 414–23 (1943).]

Decolorization procedures in the determination of nicotinic acid (I) by the CNBr and $PhNH_2$ colorimetric method were investigated. Tests were made on uncolored solutions and solutions colored with caramel. Various common bleaching carbons adsorbed 38·2–90·2 per cent. of (I) amide with the coloring matter, depending on the C sample and amount used; 20–100 mgm. per 20 c.c. of solution was used. A wide range of adsorption was also obtained with common bleaching earths, (I) amide being more highly adsorbed than (I). However, a method for determination of total (I) and (I) amide by adsorption on Clarite standard A and elution with $Ba(OH)_2$ yielded good results, and this method is presented in detail. Precipitating $Zn(OH)_2$ in the test solution removed 10·9–17·7 per cent. of the (I) with the color. The production of $BaSO_4$ precipitate in the solution was too low in decolorizing power to be practical. The Ritsert decolorizing method (*C.A.*, **33**, 8659[3]), by which the (I) extract is dried and extracted with $C_6H_6$: iso-PrOH or $C_6H_6$: $CHCl_3$ mixtures yielded very satisfactory results.

M. M. PISKUR (Chem. Abstr.).

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*Interrelation of Methionine, Choline, Betaine and Arsenocholine in the Chick. Almquist, H. J., and Grau, C. R.* [*J. Nutrition*, **27**, 263–9 (1944).]

The effects of betaine or methionine and arsenocholine are additive, and together, are practically a complete substitute for choline. G. P. (Chem. Abstr.).

*Inhibition of Cholinesterase by Novocaine and Larocaine. Ammon, R.* [*Klin. Wochschr.*, **20**, 696–7 (1941); *Chem. Zentr.*, **11**, 3083 (1941); *cf. C.A.*, **35**, 2162$^5$.]

Cholinesterase is markedly inhibited by novocaine and larocaine. This is probably connected with the favorable therapeutic experience with the combination of acetylcholine with one of these anesthetic agents in disturbances of peripheral circulation (Ratschow).

RUTH BERGGREN (Chem. Abstr.).

*The Effect of Atabrine on Choline Deficiency in the Young Rat. Hegsted, D. M., McKibbin, J. M., and Stare, F. J.* [*J. Nutrition*, **27**, 149–53 (1944).]

The inclusion of atabrine in low-choline diets at a level of 65 mgm. per cent. almost completely prevents hemorrhagic kidneys in the rat. A level of 40 mgm. per cent. prevents death, but allows some kidney damage to develop. No lipotropic action was observed at these levels.

GERTRUDE PARKINSON (Chem. Abstr.).

*Addiction to Benzedrine and Pervitin. Hansen, P. From.* [*Ugeskrift Laeger*, **106**, 95–9 (1944).]

Hansen reports four cases of addiction to benzedrine. He reviews the pathological picture of chronic addiction to benzedrine and pervitin, and points out that the tolerance to these drugs becomes considerably greater with use, with the result that larger doses are needed. Excessive use results in chronic intoxication and often leads to psychosis. Results are desiccation and loss of weight. Abstinence symptoms are not pronounced, but the risk of recidivation is great. Not infrequently the use of these drugs is combined with the use of hypnotics or sedatives, so that a vicious cycle is set up. Hansen also discusses the indications for utilization of the drugs of the benzedrine group. GERTRUDE E. PERLMANN (Chem. Abstr.).

*Action of Glyceryl Trinitrate on Chronaxia of the Snail Foot. Hazard, Rene, and Jequier, Robert.* [*Compt. rend. soc. biol.*, **135**, 1086–9 (1941); *Chem. Zentr.*, **11**, 2925 (1942); *cf. C.A.*, **37**, 5139$^8$.].

In concentrations of 1 : 150,000–1 : 10,000 glyceryl trinitrate has a stronger action than $NaNO_2$ and increases the chronaxia of the snail foot. Paradoxically concentrations of 1 : 5,000–1 : 2,000 have less effect. A 1 : 1,000 solution renders the muscle non-irritable. $NaNO_2$, 1 : 1,000 has a diphasic effect. L. E. GILSON (Chem. Abstr.).

*The Action of Theophylline and Other Drugs on the Blood Circulation in Brain. Noell, W.* [*Z. ges. exptl. Med.*, **110**, 589–600 (1942); *Chem. Zentr.*, **11**, 2495–6 (1942).]

Theophylline, which causes a dilatation of the cerebral vessels, has a stronger action than caffeine but is less active than nitrite. The influence of deriphylline, euphylline, theophylline-isopropanolamine and theocin Na aceticum was insignificant. If equimolecular doses of isopropanolamine, diethanolamine and ethylenediamine were administered, only the latter was active. It is concluded that in the preparations euphylline and deriphylline, theophylline acts in a favorable way, whereas in theophylline-isopropanolamine the action can be attributed to the theophylline entirely. GERTRUDE E. PERLMANN (Chem. Abstr.).

5, 5-*Diphenylhydantoin and Its Na Salt, Antiepileptics but not Hypnotics. Cheymol, J.* [*J. pharm. chim.* (9), **2**, 305–9 (1942); *Chem. Zentr.*, **11**, 2496 (1942).]

Hydantoin derivatives (Na diphenylhydantoin) are good antiepileptics without the hypnotic action of barbituric acid compounds. The excitability of the motor cortex is not influenced; the rheobase is not changed; the chronaxia is decreased. If the drug is administered intravenously, the circulation is depressed. Na diphenylhydantoin is insoluble in ether and benzene, slightly soluble in EtOH, soluble in water (pH 11·7), dissociated by acids and $CO_2$. Diphenylhydantoin is water-insoluble, stable. The hydantoins are less well borne, but not as poisonous as barbiturates. GERTRUDE E. PERLMANN (Chem. Abstr.).

*Action of Atabrine on the Electro-cortical Potentials. Pick, E. P., and Hunter, J.* [*J. Pharmacol.*, **80**, 354–61 (1944).]

Atabrine in doses of 5–20 mgm./kgm. given intraperitoneally or intravenously can alter the electrocorticogram of the cat under nembutal anesthesia. Tracings are shown. The effect is central depression, and seems to be independent of the concentration of atabrine in the blood but related to the atabrine content of the brain. A depressant action was also observed in pithed frogs after injection of 0·5–2·0 mgm. L. E. GILSON (Chem. Abstr.).

*Relation of Drug Addiction to the Autonomic Nervous System: Results of Tests of Peripheral Blood Flow. Himmelsbach, C. K.* [*J. Pharmacol.*, **80**, 343–53 (1944).]

The resting blood flow to the hands of morphine addicts and post-addicts is subnormal, while that of habitual marihuana users is normal. Morphine significantly increases the rate of

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blood flow to the hand and forearm. The total increase in the first hour after injection is greater with 10–20 mgm. of morphine than with 40–60 mgm. The effect on the hand seems to require intact sympathetic nerve control. Demerol also increases blood flow to the hand.

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

*The Blood-brain Barrier and Cerebrospinal Fluid in Relation to the Efficacy of Sleeping-sickness Drugs. Lourie, E. M.* [*Trans. Faraday Soc.*, **39**, 340–7 (1943).]

It is pointed out that in the first stage of sleeping sickness the infection is practically limited to the blood and the lymphatic system. In the second stage some of the trypanosomes enter the substance of the brain, and in this case an administered drug must find its way, or exercise its influence, beyond the blood-barrier brain. Some facts and opinions are reviewed under the following topics: (1) Localization of the blood-brain barrier and its relationship to the cerebrospinal fluid; (2) the need of a technique for determining the ability of trypanocides to appear beyond the blood-brain barrier; (3) examination of trypanocidal power of the cerebrospinal fluid after intravenous treatment; and (4) factors determining the ability of substances (*a*) to penetrate the blood-brain barrier and (*b*) to appear in the cerebrospinal fluid.

VICTOR R. DEITZ (Chem. Abstr.).

*Action of Acrichine on Parenchymatous Organs and the Nervous System (Experimental-morphological Studies). Dubovskaya, R. S., and Balanina, N. V.* [*Therap. Arch.* (*U.S.S.R.*), **19**, 354–9 (1941); *Chem. Zentr.*, **11**, 2817 (1942).]

Intravenous injections of therapeutic doses of acrichine in amounts of 2–2·5 mgm./kgm. over a period of seven days produced morphological changes in the organs; this indicates a stimulation of function of the active connective tissues and the protective reaction of the organism (significant amounts of macrophages in the spleen with a high iron pigment content, aggregation of hystiocytes and satellites in the region of the heart ganglion, proliferation and hypertrophy of the Hortega cells). With the introduction of subtoxic doses of acrichine in amounts of 3, 4, 5 mgm./kgm., especially with repeated injections, the blood-vessel walls (increased permeability) and the elements of the active connective tissue were affected. In toxic doses acrichine exerted a direct toxic action on the vegetative nervous system, especially on the ganglion cells and heart plexus. HELEN LEE GRUEHL (Chem. Abstr.).

*Action of Desoxycorticosterone Acetate on the Nervous System. Chauchard, Paul.* [*Compt. rend. soc. biol.*, **137**, 175–6 (1943).]

In normal rats and guinea-pigs the intraperitoneal injection of 0·04–0·20 mgm./kgm. slightly decreases peripheral and central nerve chronaxia, while 2 mgm./kgm. causes general depression with a large increase in nerve chronaxia. All doses decrease muscle chronaxia. Doses up to 1 mgm./kgm. restored normal chronaxia in nerve centres depressed by avitaminosis C.

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

*Pharmacology of the Nitrate of Choline Perchlorate. Carr, C. Jelleff, Bell, Frederick K., Evans, Wm. E., jun., and Krantz, John C., jun.* [*J. Pharmacol.*, **80**, 171–5 (1944).]

The nitrate ester of choline perchlorate (I) m. 188–9° is stable in air. A saturated aqueous solution at 20° contains 0·8 gm./100 c.c. A 1 per cent. solution dropped in the rabbit's eye produces miosis. (I) is about 10 times as toxic as acetylcholine (II) in rats. Its depressor activity is about half that of (II), and is abolished by atropine, but not by cholinesterase, and remains unchanged upon incubating an hour with dog blood serum. In rats the injection of 1–40 mgm./100 gm. produces chromodacryorrhea (red tears) within 1 minute; this is prevented by intraperitoneal injection of atropine sulfate (10 mgm./100 mg.).

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

*Action of Morphine on the Central Nervous System of the Cat. Wikler, Abraham.* [*J. Pharmacol.*, **80**, 176–87 (1944).]

Experiments were made on intact, acute decorticate, acute hypothalamic, acute decerebrate, acute spinal and chronic spinal cats in an effort to determine the mechanism and loci of action of morphine. L. E. GILSON (Chem. Abstr.).

*Tetany. III: The Action of Metrazole on Blood Glucolysis. Robuschi, Luigi.* [*Biochem. terap. sper.*, **28**, 39–50 (1941); *Chem. Zentr.*, **11**, 1531–2 (1941).]

The addition of metrazole (I) to whole blood or washed erythrocytes *in vitro* causes a decrease of glucolysis, of the formation of lactic acid and of the glucolytic quotient. Low concentrations of (I) cause increase of the glucose metabolism without affecting the glucolytic quotient. Intravenous injections of 20 mgm./kgm. of (I) in rabbits attenuate the glucolytic activity of the blood; it remains uncertain whether this is a direct action of (I) or is secondary to the hyperglucemia and change in blood-cell count. (*Cf. C.A.*, **34**, 5540[6].) A. E. MEYER (Chem. Abstr.).

*Comparative Studies of the Action of Various Analeptics on Convulsions and Blood Pressure. Hahn, F.* [*Arch. exptl. Path. Pharmakol.*, **198**, 491 (1941); *Klin. Wochschr.*, **21**, 867 (1942); *cf. C.A.*, **38**, 2109[9].]

The actions of various intravenously administered analeptics with regard to the production

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of convulsions and effect on the blood pressure were compared with metrazole (I) in cats with vagi cut and carotids clamped during chloralose anesthesia. The action of azoman on the convulsions and blood pressure was 7·5 and 10 times that of (I) respectively. The blood-pressure curve, however, showed the same course as with (I). In the case of hexetone a marked lowering preceded the rise in blood pressure. Convulsant and pressor activity corresponded to 3·5 times that of (I). With the same dosage caffeine produced the same increase in blood pressure as (I). Here, too, a steep, deep but brief lowering of the blood pressure preceded the increase. (I) had a 5-fold greater effect on blood pressure than theophylline. The convulsant and pressor activity of (I) was exceeded 30- to 35-fold by picrotoxin. The effect lasted ten times as long. In the region of non-convulsant doses the action of strychnine on the blood pressure was uncertain. In the case of coramine the course of the blood-pressure curve was variable; the convulsant dose was 5 times greater than that of (I). The convulsant action of neospiran was 5–6 times greater than that of (I); small doses frequently caused a lowering of the blood pressure, while larger doses produced first an increase, then a decrease in blood pressure. The convulsant activity of (I) was three times that of cycliton. Small doses of cycliton caused only a lowering of the blood pressure. The experiments showed that the analeptic agents have very different points of attack; this must have practical significance. RUTH BERGGREN (Chem. Abstr.).

*Detoxification of Metrazole. Fournier, Gabriel.* [*J. Hotel-Dieu Montreal*, **12**, 112–15 (1943).]

Albino rats of 170–195 gm. weight were divided into three groups, one undergoing hepatectomy, another bilateral nephrectomy, and the third acted as control. Injection of 20 mgm. in one dose, 10 mgm. given initially followed by two 5-mgm. injections at hourly intervals, or 0·8 mgm. per gm. of weight, in all three cases showed no significant differences in the mortality of the three groups. The traumatic factor was eliminated by comparison with a group which had undergone laparotomy. It is concluded that the liver and kidneys play no role in the detoxification of metrazole. H. LEVERNE WILLIAMS (Chem. Abstr.).

*Metrazole and the Differential Diagnosis of Comatose Conditions. Heinrich, A.* [*Klin. Wochschr.*, **21**, 1106–7 (1942); *Chem. Zentr.*, **1**, 976 (1943).]

In intoxication with narcotics, EtOH or alkaloids a rapid intravenous injection of 1–3 c.c. of metrazole (10 per cent. solution) produces increase in pulse rate and a temporary opening of the eyes but never convulsions. In many other comatose conditions the same doses cause convulsions, motor restlessness, vomiting, trismus or cyanosis with temporary arrest of respiration. This is the case in diabetic and uremic coma, in hypoglucemic conditions, meningeal and cerebral apoplexy, and after concussion, epileptic attack and Co poisoning. Recommendations for differential diagnosis are: 50 to 100 c.c. of 35 per cent. glucose given intravenously to exclude hypoglucemia, followed by metrazole (1–3 c.c.) if no response was obtained. If no general reactions follow, a narcotic poisoning may be suspected; this is confirmed if a second injection of 3–5 c.c. given 15 minutes later equally fails to elicit general reactions. The method is without danger if precautions are taken against fractures and biting of tongue.

A. E. MEYER (Chem. Abstr.).

*The Chemotherapy of Experimental Virus Infections.* (1) *The Effect of Certain Pyrimidine Derivatives on Experimental Murine Poliomyelitis. McKinstry, Donald W., and Reading, Elizabeth H.* [*J. Franklin Inst.*, **237**, 422–31 (1944); *cf. C.A.*, **38**, 2653[3].]

It was thought that such compounds might inhibit virus growth by competing with the normal virus pyrimidine components for the synthetic enzyme systems involved. Graded doses of the compounds suspended in equal parts of distilled $H_2O$ and mucilage tragacanth were administered by gavage to 15–20 gm. Swiss albino mice (male and female); significant prolongation of life or survival over controls was used as the criterion of therapeutic potency. Brain tissue from mice infected with the S. K. murine strain of poliomyelitis virus was used to prepare the inoculum for experimental infection, 0·1 ml. (usually 100 minute lethal dose) of freshly prepared suspension being used for each infection. The results were essentially negative, although 5 of the 42 pyrimidines investigated appeared to exert a slight favorable influence against experimental murine poliomyelitis. Substitution into uracil, e.g. alkyl, halogen, nitro or amino groups into position 5, sulfur or alkylmercapto groups into position 2, methyl groups into position 3 or into positions 1 and 3 and carboxyl into position 6 did not give compounds having anti-poliomyelitis activity, although the 6-phenyl- and the 6-methyluracil compounds showed definite antiviral action. The most effective compounds examined were the ethyl esters of 2-oxo-4-R-6-methyl-1, 2, 3, 4-tetrahydro-5-pyrimidine-carboxylic acids where R was phenyl, 4-hydroxyphenyl, n-hexyl, styryl or 3-methoxy-4-hydroxyphenyl: a number of related and unrelated substituents for R gave compounds which were either inactive or only slightly effective.

EDWARD B. SANIGAR (Chem. Abstr.).

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