Part III.—Bibliography and Epitome.*

AN attempt is being made to provide as far as possible a complete bibliography compiled from journals dealing not 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.

2 *Abh. Neur. Psychiat.*
Abh. Psychother.
2 *Acta Española Neur. y Psiquiat.*
Acta Neurol., Naples.
3, 4 *Acta Psychiat. et Neurol.*
3 *Acta Psychol., Hague.*
Acta Psychol., Keijo.
Alg. Neder. Tijdschr. Wijsbegeert. Psychol.
Aliéniste Français.
2 *Allg. Ztschr. f. Psychiat.*
Altersprobleme.
Am. Imago.
2 *Am. J. ment. Def.*
2, 3 *Am. J. Orthopsychiat.*
1, 2, 3, 4 *Am. J. Psychiat.*
2, 3 *Am. J. Psychol.*
Am. Psychol.
An. Bras. Hig. Ment.
An. Istit. Psicol. Univ. B. Aires.
An. Psicotec., Rosario.
Anal. Inst. Neurol., Montevideo.
Analele Psihol. (Rumania).
Anales de psicol., Buenos A.
2, 3 *Année Psychol.*
2, 3 *Ann. Méd. Psychol.*
2 *Ann. Osp. psichiatri., Perugia.*
Appl. Psychol. Monogr.
Arb. Psychiat. Inst., Sendai.
Arch. Anthrop. crim.
2 *Arch. Argent. de Neurol.*
Arch. Argent. Psicol. norm. pat.
2 *Arch. Bras. de Neur. e Psiquiat.*

* 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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Arch. Brasil Hig. Ment.
Arch. Chilenos de Crim.
Arch. Ital. di Studi Neuropsich.
1, 2 *Arch. Neurobiol.*
4 *Arch. Neurol., Paris.*
2 *Arch. de Neurol. de Bucarest.*
1 *Arch. de Neurol.*
2 *Arch. di Antropol. Crim.*
Arch. di Crim. Neuropsiquiat. y Disc. Con., Quito.
Arch. di Sci. Cereb. Psich., Salerno.
2, 4 *Arch. f. Psychiat.*
2, 3 *Arch. ges. Psychol.*
Arch. Internat. 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.*
2, 3 *Arch. Psychol., N.Y.*
Arch. Relig. psychol.
Arch. Speech.
Arq. da Assist. a Psicop. de Pernambuco.
Arq. de Neuro-psiquiat., Brasil.
3 *Austr. J. Psychol. Phil.*

Beih. Z. angew. Psychol.
Beih. Zbl. Psychother.
Bol. Inst. Psiquiat., Rosario.
2, 3, 4 *Brain.*
3 *Brit. J. Educ. Psychol.*
2, 4 *Brit. J. Inebriety.*
1, 2, 3, 4 *Brit. J. Med. Psychol.*
2, 3, 4 *Brit. J. Psychol.*
2, 3, 4 *Brit. J. Psychol. Monogr. Suppl.*
Bull. Canad. Psychol. Ass.
2, 4 *Bull. de la Soc. de Psychiat. de Bucarest.*
2 *Bull. de la Soc. Roumaine Neur. Psychiat. Psychol. Endocrin.*
Bull. du Group. Franç. d'étud. de neuro-psychopath. infant.
Bull. industr. Psychol., Melbourne.
2 *Bull. Los Angeles Neur. Soc.*
2 *Bull. Menninger Clin.*
Bull. Milit. Clin. Psychol.
Bull. Soc. Psihol. med., Sibiu.

Canad. Journ. Occup. Ther.
2, 3 *Cath. Univ. of Amer. Studies in Psychol. and Psychiat.*
2 *Cervello.*
2, 3 *Character and Per.* (now *J. Personal*).
2 *Child Develpm.*
2 *Child Develpm. Abstr.*
Child Develpm. Monogr.
Child Study.
Chin. J. Educ. Psychol.
Chin. J. Psychol.
3 *Comp. Psychol. Monogr.*
2 *Confinia Neurol.*
Contr. del Lab. di Psichol.
Contr. Lab. Psicol., Milan.
Contr. psychol. Theor.
Criança Portuguesa.

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

3 *Educ. psychol. Measmt.*
Egypt. J. Psychol.
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. Jap.*
2 *Fortsch. Neur. Psychiat.*

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

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

Illinois Psychiat. J.
2 *Index Neurol. y Psiquiat., Buenos Aires.*
3 *Indian J. Psychol.*
Indiv. Psychol. Bull.
3 *Industr. Psychol.*
3 *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, 2, 3 *J. Comp. Psychol.*
J. Consult. Psychol.
J. Crim. Law and Criminol.
2 *J. Crim. Psychopathol.*
2 *J. de Psychiat. Infant.*
3 *J. Educ. Psychol.*
J. Except. Child.
3, 4 *J. Exp. Psychol.*
2 *J. f. Psychol. u. Neurol.*
3 *J. Gen. Psychol.*
2, 3 *J. Genet. Psychol.*
J. Geront.
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, 2 *J. Neuropath. Ex. Path.*
2, 4 *J. Neurophysiol.*
J. Neuropsychiat. du Pacifique.
2 *J. Neurosurg.*
3 *J. Parapsychol.*
J. of Psychic. Res.
J. Psihoteh.
2 *J. Psycho-Asthen.*

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2, 3 *J. Psychol.*
J. Psychol., Moscou.
2, 3 *J. Psychol. Neurol., Leipzig.*
2 *J. Psychol. Norm. Path.*
J. Soc. for Psychic. Res.
3 *J. Soc. Psychol.*
J. Speech Dis.

Kriminal.
3 *Kwart. Psychol.*

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

Ned. Tijdschr. Psychol.
2 *Neopsichiat.*
2 *Nervenarzt.*
3 *Neue psychol. Stud.*
Neurbiol., Pernambuco.
Neur. a Psychiat., Česká.
Nevrasse.
2 *Nevropath. i. Psikhiat.*
2 *Note e Riv. di Psichiat.*
Now. Psychjar.
Nuova Riv. di Clin. ed Assist. Psichiat.

Obshch. Klin. Nevropat.
2, 3 *Occup. Psychol.*
2 *Occup. Ther. and Rehabil.*
2 *Onder. Psychiat-Neur. Klin., Utrecht.*
2 *Osped. Psichiat.*

3 *Person. J.*
Pisani.
Polsk. Arch. Psychol.
Prace Psychol.
2 *Proc. Amer. Assoc. Stud. Ment. Def.*
2 *Proc. A. Res. Nerv. and Ment. Dis.*
3 *Proc. Soc. Psych. Res. London.*
3 *Psichotec.*
Psicoanalisi, Rome.
Psicoter. Cordoba.
Psyche, Cambs., Mass.
Psychiat. Monogr.
2 *Psychiat. en Neurol. Bl., Utrecht.*
2 *Psychiat. et Neurol. Jap.*
2 *Psychiat. Neurol. Wchnschr.*
2 *Psychiat.*
1, 2 *Psychiat. Quart.*
2, 3 *Psychoanal. Quart.*
1, 2, 3 *Psychoanal. Rev.*
2, 3 *Psychol. Abstr.*
2, 3 *Psychol. Bull.*
Psychol. Clin.

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Psychol. Exch.
3 *Psychol. Forsch.*
Psychol. Index.
2 *Psychol. Monogr.*
Psychol. Rec.
3, 4 *Psychol. Rev.*
3 *Psychol. Rev. Monogr.*
Psychol. Stud. Kbh.
Psychol. Stud. Univ. Bp.
Psychol. wychow.
3 *Psychomet.*
Psychometr. Monogr.
2 *Psychosom. Med.*
2 *Psychosom. Med. Monogr.*

Quart. J. Speech.
2 *Quart. J. Stud. Alc.*
Quart. Rev. Psychiat. Neur., Washington.

Rass. Neurol. Veget.
Rass. Studi Psichiat.
Rev. Argent. Neurol. Psiquiat.
Rev. de Psicoanal., Argentina.
Rev. di Neur. e Psychiat., S. Paolo.
Rev. di Psiquiat., Chile.
Rev. di Psiquiat. y Crim.
Rev. Espan. de Oto-neuro-oftam. y Neurocir.
3 *Rev. Franç. Psychoanal.*
Rev. Ibero-Amer. de Anal. Biblio. de Neurol. y Psiquiat.
Rev. Mex. Neurol. Psiquiat. y Med. Legal.
1, 2, 4 *Rev. Neurol.*
2 *Rev. Neurol. de Buenos-Aires.*
Rev. Neurol. Psychiat., Praha.
2 *Rev. Neuro-psiquiatr., Lima.*
2 *Rev. Oto-Neur.-Oftal. Cir. Neur. Sud-Am.*
Rev. Neuropsiquiatr.
Rev. Psicol. Pädag.
Rev. Psicoandl., B. Aires.
3 *Rev. Psihol.*
Rev. Psihol. Icor. Aplic., Sibiu.
2 *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.
Ric. Psich.
2, 3, 4 *Riv. di Neurol.*
Riv. di Neuro-psiquiat., Peru.
3 *Riv. di Psichol.*
Riv. Ital. di Endocrin. e Neurochir.
2 *Riv. Patol. nerv. ment.*
Riv. Psichol. Norm. Pat.
2, 4 *Riv. Sper. Freniat.*
1 *Rocz. Psychjat.*
2 *Rorschach Res. Exch.*

1 *Schizofrenie.*
2 *Schweiz. Arch. Neurol. Psychiat.*
Schweiz. Z. Psychol.
Schweiz. Z. Psychol. Anwend.
Skand. Arch. Psychol.
Sovet. nervropatol., psichiatri, psichoguoguia.

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Sovet. Psichoneurol.
Sovet. Psikhotekh.
Speech Monogr.

Tohoku Psychol. Folia.
2 *Tr. Am. Neurol. A.*
Tr. Beritov Inst., Tiflis.
Tr. Kostchenko Ment. Hosp., Moscow.
2 *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. Psychol.
Z. Berufs. des Pflegepers.
Z. Individ. Psychol.
2, 4 *Z. ges. Neurol. Psychiat.*
3 *Z. Pädag. Psychol.*
Z. Parapsychol.
2 *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 CHIR. SCAND.

VOL. XC. 1944.

Surgical Care for Gunshot Wounds of Brain. *Tönnis, W.* 275

ACTA PSYCHIAT. ET NEUROL.

VOL. XXI. 1946.

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On the Psychopathology of Disseminated Sclerosis.

A brief review is given of the psychopathology of disseminated sclerosis, and the specificity and differential-diagnostic importance of the euphoria is emphasized.

A relatively well-examined, modern collection of cases is analysed.

47·3 per cent. of 330 patients developed mental symptoms. The neurologic symptomatology is stated and appears to have been comparatively equal for the psychonormal and the psychopathological groups, only the severe motor symptoms and the sphincter disturbances being most frequent in the psychopathological group (39·5 per cent. against 21·4 per cent., and 60·5 per cent. against 42·8 per cent. respectively). Psychiatric predisposition and premorbid mental abnormality were likewise a little more frequent in the psychopathological group.

Three patients died. The pathologico-anatomical findings were those usually made in the case of disseminated sclerosis.

The different mental symptoms and syndromes registered are stated and those

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occurring most frequently pointed out. Euphoria was found in 33·0 per cent. of the total number of cases and in 69·2 per cent. of the psychopathological cases, dementia was found in 14·0 per cent. and 29·5 per cent. respectively, and euphoria combined with dementia in 8·5 per cent. and 17·9 per cent. respectively.

A parallel is demonstrated between the degree of the euphoria and that of the paralyses, as well as the frequencies of the sphincter disturbances and the spinal fluid changes. Further it is shown that the mental changes are early symptoms (41·6 per cent. at least developed within the first 3 years).

19·2 per cent. of the psychopathological patients were themselves aware of their mental symptoms.

A number of special mental pictures are mentioned. Three patients presented psycho-reactions of the exogenous type during their stay in this Department, two had previously experienced exogenous psycho-reactions, and one a schizophreniform psychosis of several years' duration, which is characteristic of disseminated sclerosis. This psychosis was cured completely.

(Authors' abstr.)

A Family with Alzheimer's Disease.

The author describes a family in which the father, the daughter and one son died from Alzheimer's disease and the remaining son suffers from mental deficiency.

(Author's abstr.)

On Delirium Acutum.

By means of a number of biochemical examinations in 5 patients with delirium acutum the writers have been able both to confirm certain observations previously made (increased urobilinuria, uremia) and to demonstrate characteristic changes of the concentration in the blood of two intermediary carbohydrate metabolites (pyruvic acid and citric acid) parallel with the clinical course of the acute delirium. The importance of the changes demonstrated is discussed; they are interpreted as disturbances of the aerobic carbohydrate metabolism, being especially compared to the knowledge of the intermediary carbohydrate metabolism acquired through enzyme research. It is emphasized that the metabolic disturbances demonstrated are not specific of delirium acutum, but that it is reasonable to suppose that they are of essential importance to the course of the disorder. (Authors' abstr.)

Psychopathic Personality Traits. Terminological Considerations.

A short resumé is given of the patient material at the Asylum for Psychopathic Criminals in Herstedvester, illustrated by a summary of the diagnoses on admission during 1943 and 1944. The definition of the term psychopathy is discussed. The general tendency is thought to be in the direction of considering psychopathy as mainly a hereditary disease, and that Danish psychiatry has definitely decided to accept psychopathy as hereditary as opposed to the environmentally caused disturbances that should be called nervosism. It is emphasized that a boundary line such as this cannot be maintained, since the hereditary and environmental factors both enter, inextricably, into all personality defects. This opinion is illustrated by a review of case-histories. It is furthermore asserted that somatic factors, especially the consequences of organic brain diseases, frequently enter into the picture also, and that in many cases one must be content with deciding that organic disturbances are one of the factors that have caused the demonstrable personality defect.

A warning is given against using the expression psychopathy in the sense that excludes in advance the consideration of any but aetiological hereditary factors. The more nearly neutral expression " personality defect " is proposed instead. This opens a possibility of a fruitful discussion of the aetiology of individual cases and, thereby, also of suitable therapeutic measures. (Author's abstr.)

SUPPLEMENTUM. 1945–46.

Pain in the Distribution-Area of the 4th Lumbar Root. *von Reis, G.* . No. 36

*A Comparison of 1,459 Shock-Treated and 969 Non Shock-Treated Psychoses in Norwegian Hospitals. *Dedichen, H. H.* . . . . . No. 37

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A Comparison of 1,459 *Shock-Treated and* 969 *Non Shock-Treated Psychoses in Norwegian Hospitals.*

A study has been undertaken of 1,087 patients given convulsion treatment (cardiazol or pentrozol), of 243 patients given insulin treatment, of 129 patients given summation treatment, and of 969 patients who served as controls. An account is given of the principles governing the selection and composition of this material with reference to the duration of the disease, the age and sex of the patients, and other factors.

The material is classed in three groups, as it was considered of importance to keep the most pure schizophrenic and manic-melancholic forms of disease separate from the more mixed forms.

After it is pointed out that the treated material and the controls are so alike that they can fairly be compared with each other, statistical calculations are undertaken.

As the duration of the disease before the institution of treatment plays so important a part that it is necessary always to class the material in syndrome groups and durational groups, the numbers in each column often become so small that they afford little opportunity for drawing conclusions by direct calculation. Standard calculations have therefore been undertaken, several durational groups being added to each other so as to provide bigger figures.

The investigations show that in Group I with hebephrenic, paranoid or katatonic syndromes without manic-depressive or confusional trends the percentage of remissions is considerably higher among the pentrozol-treated patients than among the controls. When the disease has lasted less than a year, the difference is 6·6 times the standard error, whereas it has fallen to less than once the standard error when the disease has lasted from 1 to 5 years before the institution of treatment.

In Group II (hebephrenic, paranoid or katatonic syndromes with manic-depressive or confusional trends) there is still a marked difference with regard to the percentage of remissions for the convulsion-treated patients and the controls when the duration of the disease was comparatively short. This difference is 5·7 times the standard error when the disease had lasted less than 1 year, and $\pm$·0·5 times the standard error when the disease had lasted from 1 to 5 years before the institution of treatment.

On the other hand, in Group III (manic-melancholic syndromes) there is no difference in the tendency to remission between the pentrozol-treated patients and the controls.

It would thus appear that, from the statistical point of view, the treatment achieves the best results in forms of the disease in which the prognosis is otherwise worst. For the manic-melancholic syndromes the prospect of a spontaneous remission is so good that the task of convulsion treatment is only to shorten the course of the disease. The observation period for the shock-treated patients was at least 1 year, and for the controls, fully 10 years. Social remissions were accepted as such only when they had lasted at least half a year. Some of the treated patients underwent a follow-up examination after 4 years. In Group I the remissions had been maintained in 76 per cent., 12 per cent. of the patients had relapsed and 12 per cent. could not be traced. In Group II the corresponding percentages were 69, 12 and 19. In all these cases the disease had lasted less than a year before the institution of treatment. When the disease had lasted from 1 to 5 years before the institution of treatment, 61 per cent. of the patients in Group I had maintained their remissions, and 24 per cent. had relapsed. The corresponding percentages for Group II were 78 and 7 respectively.

The follow-up examinations also showed that some of the patients not achieving a remission in response to the treatment did so later on.

This study also showed that in Group I the reaction to the treatment was somewhat better as the patients were older. The sex of the patient does not seem to matter in this respect. It is possible that the treatment is more effective when the outbreak of the disease can be referred to a precipitating factor than when this is not the case. This is so both in Group I and in Group II. In Group I, when the disease has lasted less than a year, it would seem that the prospect of the treatment being successful is better when the onset of the disease has been acute than when it has been insidious. Earlier attacks of the disease do not seem to influence the

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effects of the treatment. The present material does not warrant the drawing of conclusions with regard to the significance in this field of body structure, a prepsychotic mentality and a hereditary taint. There follows a detailed account of each group. In Group I it would seem that the prognosis is more or léss the same for the paranoid and katatonic syndromes, whereas the fate of hebephrenics seems to be worst both among the patients given convulsion treatment and among those serving as controls. Influence phenomena, whether they occur by themselves or in association with a paranoid syndrome, do not seem to make the prognosis appreciably worse.

Certain syndromes not fitting into any of the three main groups are discussed by themselves. It would seem that diseases of the basal ganglia constitute a special contra-indication to convulsion treatment. The special problems arising in connection with women approaching the menopause, with its proneness to anxiety and an oscillating blood pressure, are discussed in detail, and modifications in the technique of the treatment of such patients are described. The complications of convulsion treatment, notably fractures, pulmonary ailments and amnestic syndromes following the treatment are discussed in detail, and the preventive measures calculated to prove effective are mentioned. The number of the patients given insulin and summation treatment being very small, the account given of them is quite brief. Considerable attention is, however, paid to the complications arising from these treatments. (Author's abstr.)

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The Electroencephalogram and Psychophysiological Regulation in the Brain.

The paradoxical association of relatively large amplitude alpha 10 ± per second activity with a relatively low level of waking cerebral function is attributed to the inhibitory effects of subcortical 10 ± per second pacing of activity in the cortex and to associated autonomic effects causing cerebral constriction. On the other hand, the association of fast activity with cortical excitation, metabolite (CO_2) production, and fast activity in cholinergic nerves accounts for associated vasodilatation. Thus, by the opposed effects of fast vs. 10 ± per second slower activity, cortical and subcortical mechanisms may regulate one another. This may provide a homeostatic mechanism by which excitation in the cortex is normally prevented from producing excessive discharge or from self-perpetuation. Underactivity, overactivity, or imbalance of these functions would account for the EEG and autonomic patterns seen in many abnormal mental conditions.

(Author's abstr.)

Effects of Sedative Drugs on the Electroencephalogram.

A study is presented of the electroencephalographic effects of small oral doses of the usual sedative drugs (phenobarbital, sodium amytal, nembutal, seconal, paraldehyde, chloral) in 10 normal subjects with normal EEGs.

Data on 45 psychiatric patients who had received oral sedation within 24 hours of the electroencephalographic recording are compared with a control group of 45 psychiatric patients who had received no sedation. Patients with a diagnosis of epilepsy or of organic central nervous system disease were excluded from this study.

The results of the two methods of study are in essential agreement and lead to the following conclusions :

1. The commonly used barbiturates given orally in small doses may change a normal to an abnormal EEG. Paraldehyde and chloral do not cause prolonged alterations in the normal EEG under the conditions of this study.

2. The change occurs in an estimated 35 per cent. of individuals with normal electroencephalographic tracings. This change is of a nature and degree to require re-evaluation of studies on patients receiving sedatives.

3. The change usually consists of an increase in fast activity, but it may consist of an increase in slow activity. In either case, the direction of the change is more constant for the individual than for the drug employed.

4. The factors which influence the appearance of an abnormal EEG after sedation are individual susceptibility, drug employed, time interval and dosage. The highest percentage of abnormal records occurs in the patients who received 0·3 grm. or more of barbiturate 12 to 14 hours before the EEG.

(Author's abstr.)

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Electroencephalographic Studies of Psychopathic Personalities.

Records showing frequencies lower than 8 a second or an abundance of waves slower than 8 a second in the frontal leads, regardless of amplitude, were considered abnormal. This is essentially in accordance with the criterion of Gibbs, Gibbs and Lennox. The record of one patient was considered adnormal during overbreathing only.

All the abnormal records fell under the type of electroencephalograms usually called "pathologic record of undetermined type." 53 per cent. of the patients had abnormal records. It was possible to divide these pathologic records into two types, namely, those of average amplitude, 5 to 7 a second activity, and those of low voltage, slower than 5 to 7 a second activity. A third pattern—low voltage, fast activity—was frequently encountered, but it was not considered definitely abnormal.

The majority of the abnormal records were those containing sufficient 5 to 7 a second activity of low average amplitude in the frontal and parietal leads to be considered beyond the limits of normal. Some 5 to 7 a second activity is commonly found in the frontal records of normal subjects, but it is unusual in the parietal leads. Any considerable amount of it in the frontal and parietal leads must certainly be considered abnormal. (Authors' abstr.)

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Penicillin Therapy for Spina Bifida. *Walker, E. A., and Johnson, H. C.* . 122

Effects of l (+) Glutamic Acid and Other Agents on Experimental Seizures.

In an attempt to measure in the laboratory the anti-convulsant value of glutamic acid, this substance was tested by a number of technics for investigating the potency of anti-convulsant agents. In a wide range of single large and repeated small doses, and in a variety of species, the amino acid was found ineffective in the prevention or modification of electrically or chemically induced convulsions. Glutamic acid failed to elevate the normal seizure threshold, did not modify the character of the convulsion produced by supramaximal currents, and had no effect on the electric shock threshold lowered by cellular hydration. Glutamic acid was also without effect on metrazol-induced electroencephalographic dysrhythmias of the *petit mal* type. In addition, it did not alter the resting electroencephalogram or the electroencephalographic response to single cortical shocks.

Inasmuch as diphenylhydantoin, phenobarbital, Tridione, dimethyl-N-methyl barbituric acid and benzimidazole give laboratory evidence of anti-convulsant potency with one or more of the technics employed, it is suggested that if further clinical work substantiates the efficacy of glutamic acid in treatment of *petit mal* and psychomotor epilepsy, the mechanism of its action is likely to prove considerably different from that of the known anti-convulsant drugs in clinical use. Indeed, one would not be able to predict from present laboratory screening methods that glutamic acid has anti-convulsant potency or value. (Authors' abstr.)

Sensation of Electric Shock Following Head Injury.

A series of 30 cases of head injury is presented in which the phenomenon of sensation of electric shock was perceived in various parts of the body on flexion of the neck. Multiple etiologic factors may be operative; the hypothesis of concomitant concussion of the cord and of a special cerebral origin are discussed. The pathologic process is unknown, demyelination of spinal tracts and meningeal scars being considered as likely factors; the process, however, is self-limited and reversible so far as the phenomenon is concerned. (Author's abstr.)

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Electric Shock Therapy of Elderly Patients.

The results of electric shock therapy in a group of 53 patients aged 65 or more are reported. The difficulties in diagnosis, the contra-indications to treatment and the complications and effectiveness of therapy are discussed. Three cardinal problems have emerged from this study:

1. The differentiation of true intellectual decline and other types of psychoses of the senium, especially depression, must be made. Suggestions for making this distinction are offered.

2. The significance of an impared cardiovascular system in the patient with serious mental illness must be evaluated in terms of what the future holds for the patient with and without treatment.

3. When a diagnosis other than senile dementia can be made in a patient over 65, especially that of depressive, manic or paranoid psychosis, electric shock therapy should be considered. The only contraindications should be extreme defects in the physical state, particularly in the cardiovascular system.

(Authors' abstr.)

Pattern of Metabolic Depression Induced with Pentothal Sodium.

In this investigation on the pattern of the action of pentothal sodium on the brain, it was possible to show that the cerebral hemispheres are the areas first involved in the depressant action of this drug, because of the peculiarities of the anatomic venous cerebral return, which in the majority of patients directs most of the blood from the cerebral hemispheres either to the right or to the left internal jugular vein, and a major portion of the blood from the basal ganglia to the opposite vein. A total of 36 observations were made on 12 subjects under pentothal anesthesia. Of 22 observations, made at the lighter levels of anesthesia, the results may be divided into two groups. In 9 of the subjects the arteriovenous difference on one side was more depressed than that on the opposite side, and in the 3 remaining patients the arteriovenous differences were similar on the two sides. Further evidence supporting the differences between the values for the right and the left internal jugular vein is obtained from the data for glucose and lactate, for in the same 3 patients in which the arteriovenous oxygen differences were always within the experimental error the arteriovenous glucose and arteriovenous lactate differences exhibited a similar agreement, while in the other 9 patients the paired results did not show a similar precise concordance. With deeper anesthesia the subcortical parts became more involved, and the paired arteriovenous oxygen differences for the two sides were greatly depressed and within the experimental error in all but 2 of 14 observations. These results indicate that oxidation is not decreased to the same extent in all parts of the brain at the lighter levels of barbiturate anesthesia, but that the cerebral hemispheres are the areas of the brain preponderantly involved in the depressant action of the drug. The other parts of the brain gradually suffer an increasing inhibition of oxidation as the deep levels of pentothal anesthesia are produced. (Authors' abstr.)

Effects of Antibiotic Substances on the Central Nervous System.

Clinical and experimental studies indicate that penicillin may produce convulsive manifestations. During systemic administration for conditions other than primary ones of the central nervous system, the electroencephalogram was found to be abnormal in more than 60 per cent. of a series of 51 patients. Control records taken before and after penicillin therapy usually showed normal tracings. Large doses of penicillin injected intrathecally in man or monkey may give rise to convulsions, followed in some cases by coma and death. The application of as little as 500 international units to the cerebral cortex of the macaque monkey may induce epileptic attacks.

Streptomycin in cats and monkeys applied to the cerebral cortex in doses of 1,250 units induced convulsive manifestations in 30 per cent. of the animals. Electroencephalographic records at such times showed slow waves and spikes, with subsequent decrease of cortical activity lasting for 1½ to 3 hours. Cisternal injection in the monkey of 2,500 units of streptomycin induced signs of severe cerebellar dysfunction.

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Streptothricin applied to the parietal cerebral cortex in doses of 5,000 to 10,000 units produced clinical and electroencephalographic convulsive manifestations. Although these phenomena usually disappeared spontaneously in 2 to 3 hours, in two monkeys they persisted for 2 weeks. At necropsy the brains of these animals showed extensive softenings with perivascular petechial hæmorrhages.

Actinomycin injected into the cerebral cortex or the cisterna magna in a dose of 1 mg. after a latent period of 9 hours produced severe prostration, fasciculations and convulsions, with death in 1 to 7 days. At the site of injection into the cerebral cortex a severe necrobiotic reaction with edema and petechial hæmorrhages was found.

Clavacin when injected into the cerebral cortex in doses of 5 to 10 mg. induced clinical and electroencephalographic manifestations of convulsive phenomena with a marked decrease in spontaneous cerebral activity.

There appears to be a wide margin of safety between the antibiotic concentration of penicillin and streptomycin and the convulsive threshold for those drugs. Such does not appear to be the case for streptothricin, clavacin or actinomycin. Although penicillin has few toxic reactions, if it is given in excessive amounts in the cerebrospinal fluid severe neural sequelæ in the form of radiculitis or convulsions may develop. (Authors' abstr.)

Dominant Brain Wave Frequencies as Measures of Physico-chemical Processes in Cerebral Cortex.

Engel and associates note that their method "may give a slightly false impression of the distribution of wavelengths greater and less than the dominant one," since they do not measure wavelength but take as the frequency the total number of waves per second. The authors' main criticism is that the method is misleading in its misuse of the concept of the dominant frequency itself if one considers waves of similar length arranged in sequences as reflecting physico-chemical processes in the cells. Engel and his collaborators are concerned with the effects of physiologic agents on the electroencephalogram, and, while empiric changes are of course brought out by their method, it seems unfortunate to employ a tool which by its very nature, through its definition of "frequency," masks the possibility of a rational interpretation of rates of events going on in the brain.

The dominant frequency as studied with the Brazier method is the frequency which the authors have used in their earlier studies of the effects of physico-chemical agents on the electroencephalogram. It would, for example, be quite impossible on theoretic grounds to calculate meaningful energies of activation of enzyme steps in the carbohydrate cycle with the method of Engel and associates, since their frequencies, including their dominant frequencies, are composed of waves of different lengths and the independent variable, the per cent. time alpha, contributes to the determination of their actual frequencies *per se*.

This may be illustrated by a hypothetic example. Suppose one has a record composed of continuous and uniform sine waves of 10 per second produced by a piece of electrical apparatus. There would thus be only one frequency, the dominant one determined by the properties of the apparatus making the signals. One may now consider an equal length of record consisting of 5-second strips of the same 10 per second waves, alternating with equal intervals with no waves at all. Such a record could be made artificially by turning the apparatus that makes the waves on and off at 5-second intervals. The dominant rhythm in both these records, according to the authors' studies and to the Brazier method, is clearly 10 cycles per second; the dominant rhythm according to the Engel method is 10 per second in the first record and 5 per second in the second record, and from this one would be led to the erroneous conclusion that the apparatus producing the waves with a frequency of 5 per second was operating at half its former rate.

For the reasons the authors have discussed, the measurements of Engel and associates cannot reflect the rates of changes of kinetic processes in the brain, and in effect they mask these processes. The Engel spectrum is a graphic representation of gross qualitative changes observed in the records, and as an analytic instrument it has all the limitations of the delta index, which the authors have described and have used as a purely empiric expression of the numerical representation of the magnitude of slow-wave activity in the electroencephalogram.

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Quantitative changes with physiologic variables of bands of waves of equal length (e.g., the alpha waves) have led to significant insight into the chemical kinetics of the brain, and further studies along these lines hold promise. Much of the value of this type of investigation may be lost unless the limitations of the methods used are borne in mind. (Authors' abstr.)

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*Cerebral Concussion: Histochemical Demonstration of Nucleases in the C.S.F. *Spiegel, E. A., et al.* 175

Cerebral Concussion: Histochemical Demonstration of Nucleases in the Cerebrospinal Fluid.

Incubation of buffered specimens of cerebrospinal fluid from patients who had undergone cerebral concussion with sections of cats' cords produced tigrolysis in the anterior horn cells, while similar treatment of the sections with buffered specimens of cerebrospinal fluid from normal persons or with buffered Ringer's solution failed to produce such an effect.

The findings point to the importance of enzymatic substances in the genesis of chromatolysis following cerebral concussion.

The nucleoproteins of the nerve cells go into solution at the hydrogen ion concentration prevailing in the normal central nervous system *in vivo*. When sections of spinal cords are incubated with various fluids, these fluids must be acidified for demonstration of Nissl bodies. (Authors' abstr.)

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Electroencephalography: Nature and Psychophysiological Significance of the Electric Phenomena of the Brain.

After a discussion of the basic principles underlying electroencephalographic techniques, the author describes the characteristics of the normal EEG in men and animals, individual differences, the effects of sensory stimuli, electric activity of the brain during sleep, the relationship between electric activity and mental activity, the effects of drugs, and relation of the EEG to temperature, circulation, and metabolism. A critical analysis of the results in the literature suggests the following conclusions: the alpha waves are the expression of synchronic "pulsations" of a great number of cortical neurons; the states of excitement (sensorial stimuli, psychical activity) disturb this synchronism and transform the

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alpha rhythm into beta rhythm. Delta waves, the origin of which is still obscure, appear with a diminished cerebral activity. The frequency of the rhythms is consequently related to major or minor cerebral activity. The modifications of the electroencephalograms as an effect of psychical activity are due to sensorial or affective components. (Psychol. abstr.)

New Contributions to Electroencephalography.

After a general consideration of the topic, the author describes the apparatus used in the psychology laboratory of the Catholic University of Sacro Cuore in Milan. Mention is made of an electromagnetic oscillograph with ink-writing pen, also constructed in the same laboratory. Sample records are reproduced. There is a discussion of the principal theories of the nature and origin of alpha waves. The factors of frequency, form and amplitude are examined, and this examination is completed by a systematic study on the lack of synchronism of the waves which originate from different parts of the brain. The author offers the hypothesis of a unit centre for the origin of the alpha waves, and gives an interpretation of the function and the nature of this centre. (Psychol. abstr.)

On Postencephalitic Dementia. The Method of Rorschach Applied to the Study of Postencephalitic Parkinsonism.

The author examined with this test 30 adult subjects affected by postencephalitic parkinsonism. The conclusion is that effectivity, some functions of the intellectual process (resistance to mental work, capacity of concentration and attention), perception and the higher logical processes are involved. This is shown by such typical signs as the following: small number of answers, high percentage of badly perceived forms (F per cent. often with negative values), reduction of kinesthesia and chromesthesia, and tendency to stereotypy. These results do not entirely agree with those of Veit, the only author who has applied the Rorschach test to the study of postencephalitic parkinsonism. (Psychol. Abstr.).

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*Problems of the Frontal Lobe. *Porta, V.* 5

Problems of the Frontal Lobe.

The author describes the case of a wounded patient with a frontal lesion whose symptoms became apparent 10 days after the accident and were of short duration. The syndrome was characterized by severe astasia-dysbasia, dysmetric and asynergic symptoms in the trunk and limbs, loss of motor initiative and emotional contact, spatial disorientation, amnesia and enuresis. Various interpretations of frontal lobe function are discussed, opposite trends represented by Goldstein's holistic approach and Kleist's localizations are evaluated, and the neuropsycho-biological basis of prefrontal lobotomy is analysed. (Psychol. abstr.)

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Narcoanalysis and Sub-shock Insulin Treatment of Anorexia Nervosa. *Gottesfeld, B. H., and Novais, A. C.* 448

Trauma and Neurosyphilis.

The authors report 13 cases in which cranial or spinal injuries probably played a significant role in the development or aggravation of several forms of neuro-syphilis; from the total number of cases, 3 refer to mesenchymatous and the rest to parenchymatous forms (5 cases of tabes and 5 of general paresis). From their own observations and from references in medical literature, the authors can state the following conclusions:

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1. Trauma can act (*a*) as an aggravating factor of a pre-existing, clinically recognized neurosyphilis; (*b*) as a precipitating factor of a latent, asymptomatic neurosyphilis; (*c*) as a localizing factor of a pre-existing syphilis which had not, till then, affected the central nervous system.

2. Each case must be judged on its own merits. The points which estimate the connection between trauma and neurosyphilis are: clinical conditions before trauma, circumstances involving the trauma, site of the injury, severity of the trauma, chronologic interval between trauma and beginning of the symptoms, bridging symptoms, shortening of the incubation period and fast development of the disease.

3. The aforementioned elements are sufficient to consider the trauma as an aggravating or as a precipitating factor. If the normality of the cerebrospinal fluid could be tested just before or just after the injury and, some months later, the beginning of the clinical symptomatology became apparent and was confirmed by the positiveness, in the spinal fluid, of the reactions for syphilis, the trauma must be considered a localizing factor. The authors' cases fulfil these conditions, and perhaps they are the only ones recorded in medical literature.

4. The mode of action of trauma on the development of parenchymatous neurosyphilis may be explained by localization of the *Treponema pallidum* in inflamed areas; (*b*) connections between trauma and immunity in neurosyphilis; (*c*) traumatic rupture of the hematoencephalic barrier; (*d*) relation between the germ's electric charge and the permeability of the barrier; (*e*) influence of the traumatic shock of the sympathetic nervous system on the development of parenchymatous neurosyphilis.

5. Whatever the mode of action of the trauma, the medico-legal aspect is the same. The effects are imputable to trauma, but syphilis will always be a pre-existing concausal element. In the juridical valuation of the injury, the assessor must fix the compensation for incapacity according to the role the trauma played; it will be greatest if the injury localized the syphilis on the nervous system, and it will be least if the trauma was only an aggravating factor. (Authors' abstr.)

Prefrontal Leucotomy.

The author discussed the different techniques for leucotomy, especially that of Freeman Watts.

He has devised a "lobotome" for a three-fold purpose—to ascertain the reference marks, sever the fibres and leave iodine-oil in the transection tract. The instrument consists of a trocar slightly flattened to take the shape of a regular leucotome and having one of the ends sealed. In one of the cutting edges near the blind end there are 4 small holes, 1 centimetre apart. Through the free end a syringe may easily draw fluid and inject contrast media into the transection surface.

To secure a more accurate placing of the transection, the writer has recently followed the technique of transfixing the falx and both frontal lobes with a stylet inserted through the burr holes. He also recommends having a pneumo-encephalography systematically performed before operation in order that the ventricles be accurately localized. He does not present any worth while results as the interval is too short.

All the patients operated on were chronic cases who had undergone all the accepted treatments, including leucotomy. (Author's abstr.)

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The Development of the Substantia Nigra.

1. The substantia nigra is developed by a ventrolateral extension of cells from the characteristic mid-ventral proliferation in the basal lamina of the mesencephalon.

2. It is first seen in the 22 mm. embryo as a peripheral crescentic strip of cells continuous with the ventrolateral angle of the mid-ventral proliferation.

3. At the same time a narrow band of fibres appears on the superficial aspect of the nigral crescent. These are descending fibres from the cerebrum, and with the nigra they form the peduncle of the mid-brain.

4. From the outset there is a close association between the substantia nigra and the descending cerebral pathway.

5. By the 30 mm. embryo stage, the nigral crescent is separated into groups by the descending fibres. Characteristic medial and lateral groups are soon formed at the medial and lateral extremities.

6. By the eleventh week (6·0 cm.) the nigra is disposed into two strata, corpus and cauda, the former lying deep to the latter with cerebral fibres between them. Medial, lateral and intermediate groups are recognizable in the corpus, and fibres extend between corpus and cauda and penetrate from the nigra into the cerebral pathway.

7. In the 9·0 cm. foetus the pyramidal shape of the substantia nigra is evident. The apex lies in the subthalamus and continues into the intermediate part of the corpus. This is soon flanked by the medial and lateral columns. The cauda lies along the ventral part of the nigra. The base is formed by the columns of the corpus and extends into the pons.

8. At mid-term a new nigral stratum appears. It will be referred to as the caput. It lies on the dorsal aspect of the corpus and is separated from it by cerebral fibres. It links the medial and lateral groups of the corpus and projects aberrant groups into the tegmentum. The caput and cauda are present only in the mid-brain portion of the substantia nigra; the corpus extends to the subthalamus and pons.

9. At the eighth foetal month a rich plexus of fine myelinated fibres pervades the nigra, and fibres extend from it into the crus and tegmentum, and into the subthalamic nucleus.

10. After birth the nigra retains its fœtal architecture, but enlarges and attains its maximum size in childhood.

11. Minute black granules are first seen in some of the nigral neurones at mid-term. Thereafter they increase, but not all the neurones contain them. After birth granular cells are numerous.

12. By dissection the pyramidal shape of the substantia nigra is confirmed.

(Author's abstr.)

The Development of the Human Red Nucleus and Corpus Striatum.

1. The human red nucleus develops as a migration of cells from the mid-ventral proliferation of the mesencephalic basal lamina.

2. These cells migrate towards, and become associated with, cerebello-mesencephalic fibres (brachia conjunctiva) which reach the mid-brain in the embryo of 19 mm. length (6½ weeks).

3. A collection of cells, situated among the cerebello-mesencephalic fibres at the base of the cerebellar outgrowth is seen in the 29 mm. embryo (8 weeks). These cells develop into the dentate nucleus, which appears in series with the red nucleus and is linked to it by tiny groups of cells in the dentato-rubral (cerebello-mesencephalic) fibre pathway. The possibility of a tegmental origin of the dentate nucleus is suggested.

4. Grouping of neurones in the red nucleus begins in the 4·0 cm. foetus (9 weeks).

5. Minute brown granules appear in the rubral neurones at mid-term. Later a diffuse brown coloration distinguishes the cells.

6. The corpus striatum arises as a thickening of the lateral wall of the cerebral vesicle in the 10 mm. embryo (5 weeks).

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7. In the 22 mm. embryo (7 weeks) descending cerebral fibres separate the corpus striatum into medial (caudate) and lateral (lentiform) parts.

8. In the 7·0 cm. fœtus (12 weeks) the lentiform nucleus is subdivided into putamen and globus pallidus. (Author's abstr.)

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*Some Qualitative Observations on Verbal Memory in Cases of Cerebral Lesion. *Zangwill, O. L.* 8
*An Examination of the Varying Effect of Certain Stimuli upon the Alpha Rhythm of a Single Normal Individual. *Harrison, J. M.* 20
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Some Qualitative Observations on Verbal Memory in Cases of Cerebral Lesion.

1. Some qualitative observations on verbal memory in cases of cerebral lesion are described. These have reference to (*a*) the auditory-verbal memory span; (*b*) rote learning, and (*c*) recall of short stories (substance memory). Special attention is given to the results in cases with disorders of language and of memory respectively.

2. An impairment of memory span for verbal material and severe difficulties in rote learning were consistently found in cases with language disabilities associated with focal lesions of the speech areas. It is pointed out that these defects may long outlast the grosser symptoms of an acute aphasic disorder.

3. Cases of pronounced impairment of general memory (amnesic or Korsakow syndrome) were found to have intact verbal memory span but grossly impaired verbal learning. Substance recall was in general very defective in these cases and fabrication was often marked.

4. It is suggested that the defect of verbal learning in dysphasic cases is syntactical in nature and closely associated with the primary disorder of symbolic formulation and expression. In amnesic cases, on the other hand, it is argued that the defect is independent of language, and due to a generalized inability to organize consecutive responses into a unitary habit.

5. It is pointed out that both rote and substance recall are equally affected in the amnesic syndrome, but that the latter is often well preserved in dysphasic conditions. Some implications of this finding for the theory of memory are briefly considered.

6. The available evidence suggests that some impairment of verbal learning is very common in organic cases irrespective of the precise nature and localization of the lesion. A tendency to stereotyped error has been noted especially in cases with lesions of the frontal lobes.

7. The bearing of the results on practical problems of diagnosis, assessment and re-education in cases of brain injury is briefly indicated. Some suggestions for future research are also mentioned. (Author's abstr.)

An Examination of the Varying Effect of Certain Stimuli upon the Alpha Rhythm of a Single Normal Individual.

A single normal subject has been repeatedly examined on the EEG and the effect of certain types of attention and emotional stimuli systematically examined. Inter-correlations of the stimuli are small and the scatter of the data about the respective means is large, with the exception of suppression of the alpha rhythm associated with visual activity. The effect of a mental arithmetic problem upon 27 cases of a small sample of normal individuals has been compared with the results of the present experiment. The following conclusions may be tentatively drawn from the results. It should be emphasized that these conclusions are based on work with this subject only, and cannot be generalized without further individual investigations:

(i) There is a low positive correlation between the degree of suppression associated with mental arithmetic and concentration problems. This appears to indicate the existence of common mental (or neural) activities for these two tasks. The lack of correlation between these two tasks and the immediate memory span test suggests that the attention associated

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with immediate memory processes is to be distinguished from that involved in arithmetical problems and indicates specific types of attention.

(ii) Suppression of the alpha rhythm is associated with focusing of the attention, but is an unreliable guide to the degree of focusing. This indicates lack of direct connection between attention and the amplitude of the alpha rhythm.

(iii) The amplitude of the alpha rhythm is an unreliable indicator of activity of the sympathetic branch of the autonomic nervous system.

(iv) Suppression of the alpha rhythm is not reliably connected with the degree of conscious emotion. The results indicate that a large emotional response is associated with a greater degree of rhythm suppression than either an average or small emotional response, but the difference between the means of these three values is not statistically significant. A small negative correlation exists between associative reaction times and degree of suppression.

(v) The neural processes directly responsible for suppression of the alpha rhythm take place below the level of consciousness since the subject is unable to state the degree of suppression associated with a particular stimulus.

(vi) In solving problems in mental arithmetic the variation in rhythm suppression shown by the single individual, who served as the main subject in this experiment, is almost as wide as that found in 27 different subjects. With the latter both the standard deviation and the mean were slightly greater; but the increases were so small as to be statistically non-significant. (Author's abstr.).

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*Performance of Brain-damaged Cases on a Memory-for-Designs Test. *Graham, F. K., and Kendall, B. S.* 303

Performance of Brain-damaged Cases on a Memory-for-Designs Test.

A memory-for-designs test, requiring less than 10 minutes to administer, and consisting of 15 designs, presented singly, was given to a group of 70 brain-damaged patients and 70 controls. The controls were also a patient population and had the same age distribution and educational and occupational background as the experimental group. An additional group of 60 controls and 10 experimentals, as well as 16 cases above the upper age limit of 60, was used for cross-validation.

1. Experimental and control groups showed significant mean differences in score on the test.

2. Impairment on the test, indicated by a score above a critical value, was rare in the controls and analysis of the individual cases indicated that it occurred only with feeble-mindedness, where concomitant brain damage is likely, or severe psychiatric illness. Psychiatric cases could generally be differentiated by qualitative indicators not included in the scoring system.

3. Impairment occurred in only 50 per cent. of the experimentals. Although additional factors such as tremor, number of reversals, etc., differentiated some of those scoring low, a considerable number still remained whose performance was in every observed respect like that of the controls.

4. Unusually low correlations were obtained between test score and age and test score and intelligence, indirectly measured by school grade completed and vocabulary. This should make the test an especially valuable addition to a battery for detecting brain damage. (Author's abstr.)

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Use of the Shipley-Hartford Test in Evaluating Intellectual Functioning of Neuropsychiatric Patients.

The Shipley-Hartford Retreat Test " was given to 977 randomly selected neuropsychiatric patients at a service hospital. Of this group, 134 were also given the Wechsler-Bellevue Intelligence test. The following conclusions seem warranted: (1) The Shipley-Hartford test can be used as a rough estimate of functional intelligence. (2) The distribution curve of ' original ' intelligence of neuropsychiatric patients is very similar to that obtained by Wechsler in a sampling of the adult population. (3) A large proportion (62 per cent.) of neuropsychiatric patients tend to show a lowering of efficiency in their intellectual functioning."
(Psychol. Abstr.).

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J. COMP. NEUR.

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Connections of the Cerebral Cortex. I. The Albino Rat. B. Structure of the Cortical Areas.

The identification and arrangement of the cortical areas of the albino rat have been determined on the basis of cell- and myelin-stained material sectioned in various planes. The picture presented by each area has been characterized and illustrated by means of sample strips taken perpendicular to the surface. Some 40 cortical areas are distinguished. Individuality and boundaries of the areas were determined on the basis of appearances alone. The identification, expressed here in Arabic numerals as proposed by Brodmann, has been made with regard to accumulated evidence on connections and probable evolutionary patterns of the cerebrum. It is believed that the parietal cortex has been subdivided in a manner that will enable areas to be equated with those of higher forms. Thus, 3, 1 and 2 occupy a prominent place, while 7 and 39 are, as expected, smaller. Motor, premotor and frontal areas have been distinguished and the frontal region has been divided into polar (10) and basal (8, 11) areas. The auditory cortex (41) is identified, changing radically the pattern formerly claimed for the temporal region of lower animals. The primary sensory receptive cortices are seen to dominate the cerebrum. Sensory associative cortices form marginal strips or fill in chinks. The frontal area is poorly developed. On the other hand, the limbic lobe is markedly developed and presents numerous subdivisions.

The study of the myelin pattern of the cortex has been instructive. Attempting to explain significance of the various categories of fibers, instead of regarding them merely as a means of distinguishing areas, has resulted in the establishment of a number of generalities. The fact that in the rat a large bulk of the interareal associational fibers are intracortical is an important one for cortical analysis.

The plan here established has been compared with the studies of others on related forms.

A plan has been devised to explain the differentiation of cortical areas, the large number that appear in the rat and the conservatism of this arrangement.

The place which this survey has in the project of cortical study under way is discussed. (Author's abstr.)

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*Experimentally Induced Convulsive Reactions of Laboratory Rats. I. *Stainbrook, E.* 243
Sensory Pre-conditioning and the Convulsions Associated with Magnesium Deficiency in the Rat. *Patton, R. A., and Lazovik, A. D.* 265

Experimentally Induced Convulsive Reactions of Laboratory Rats. I. A Comparative Study of the Immediate Reactions.

The conclusions derivable from the experimental observations described in the present study are summarized as follows:

1. The essential motor pattern of abnormal behavior evoked in some rats by noise-fright stimulation can also be evoked in the same animals by direct electrical stimulation of the brain.

2. The essential motor features of the noise-induced abnormal behavior pattern can be produced by electrical stimulation in rats which do not easily react to the noise-fright situation with the NF-3 or NF-4 type of response.

3. However, the electrically induced noise-fright pattern is suggestively more frequently produced in rats which do react with the NF response to the noise-fright situation.

4. Vigorous running is not a necessary precursor of the convulsive phase of the electrically induced NF pattern and of the ensuing passive, hypokinetic state of the reaction.

5. Since the electrical stimulus lasts only ·2 second and since the convulsive phase of the ES-NF-4 pattern is immediately precipitated in some cases, the motor aspects of the abnormal behavior pattern may occur in the absence of any psychologically significant state of fear.

6. Inasmuch as the ES-NF response is always evoked by an electrical voltage less than that necessary to produce a maximal generalized convulsion, the ES-NF pattern represents a reaction to an abnormal but not maximal neurophysiological stimulation. In view of the motor similarity of the ES-NF and NF patterns, it is strongly suggested, therefore, that the NF response is also a reaction to an abnormal but not maximal neurophysiological stimulation.

7. The pattern of the motor expression of the NF reaction in the rat is similar to the motor symptoms of experimental catatonia described as occurring in many other animals following doses of bulbocapnine as well as following other physical and chemical alterations of the internal environment of the animals.

8. The symptoms of catatonia observed following the noise-induced acute behavior reaction last for a significantly longer time than the catatonic symptoms demonstrable after an electrically induced acute NF-4 response. This relatively longer duration of the NF catatonic symptoms is not, therefore, produced simply by the neuromuscular expression of the motor pattern of the response, since the catatonia of the ES-NF-4 reaction in which the same motor pattern may be seen is of shorter duration.

9. The catatonic symptoms following a generalized electro-shock convulsion have a significantly shorter duration than the same symptoms manifest after either

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ES-NF-4 or NF-4 reactions. Therefore, the longer-lasting catatonia of the noise-induced behavior is not entirely attributable to any convulsive exhaustion of the animal, since the catatonic symptoms following a generalized convulsion with maximal electrophysiologic discharge of the nervous system are of significantly shorter duration.

10. The longer duration of a catatonic symptom in rats following noise-induced abnormal behavior as compared to such behavior electrically induced is not attributable to any significantly variable duration of the pre-convulsive running phase of the reaction.

11. The absence of a gross startle reaction and the immediate presence of reflexes in the post-convulsive period of the ES-NF and NF responses contrasted with the complete loss of reflexes and the appearance of uncontrolled startle reactions in the recovery period following generalized electrically-induced convulsions indicates that the ES-NF and NF reactions do not involve complete central nervous system dissolution or a discontinuity of consciousness.

12. The demonstration of rage reactions, squeaking, biting, running away from the situation, reaction to pin-prick, and a general more excitatory response to stimuli in the ES-NF reactions as compared to the NF response indicate that while the motor expression of the two differently evoked reactions may be the same, the NF reaction is characterized not only by neuromuscular dysfunction attributable to the motor expression of the reaction but by behavior which is probably best described as reflecting a generalized inhibitory state of the animal.

(Author's abstr.)

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A Method for the Maintenance of Chronic Spinal Animals. *Kellogg, W. W.* . 366
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Level of Autonomic Activity and Electroencephalogram.

In a study of 120 persons in the resting-waking state a correlation of $r = \cdot 28 \pm \cdot 06$ between parietal-occipital EEG potential and palmar skin conductance and a correlation of $\cdot 33 \pm \cdot 06$ with heart rate were demonstrated. Combination of skin conductance with heart rate gave a correlation of $\cdot 48 \pm \cdot 05$, thereby suggesting synergistic autonomic action on the EEG. That these correlations obtained in the resting-waking state are of opposite sign to those typical of excitatory effects after stimulation, suggests that the low level of cortical activity and metabolite production in the resting state permits dominance of the cortex by the subcortically determined 10 $\pm$ per sec. rhythm and by vasoconstrictor regulatory effects from the autonomic system. It is pointed out that in certain conditions of anticipation where cortical activity precedes the stimulus and in certain emotional reactions where there may be a suppression of central ideational activity and an exaggeration of subcortical and autonomic effects, such a relationship may explain instances of "facilitation" of alpha and slower EEG potentials. It is suggested that levels of autonomic activity giving correlations with EEG in the resting state opposite in sign to correlations produced in conditions of cortical excitation provide evidence of opposing cortical *v.* subcortical influences on cerebral function and offer a means for studying phenomena of mutual cortical-subcortical regulation.

(Authors' abstr.)

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Disseminated, Diffuse and Transitional Demyelination of the Central Nervous System.

The relationship between disseminated and diffuse sclerosis has been under consideration since Schilder's well-known papers on encephalitis periaxialis diffusa. A table is presented in support of the opinion that a very close relationship exists between disseminated and diffuse demyelination as far as heredity traits, sex, age, duration of disease, clinical diagnosis and gross pathologic findings are concerned.

Cases of diffuse or patchy demyelination of sporadic and heredo-familial type have been reported to occur at all ages from the early age of 1 to 2 months up to the age of 70. The duration of the disease has varied from a few days to 18 to 20 and 26 years and the pathology varied from one of patchy, transitional and diffuse demyelination, irrespective of the original clinical diagnosis.

There are no special mental disturbances which are typical of disseminated sclerosis as opposed to diffuse sclerosis. Bouman thinks that they are not very prominent and Guillain considers them of secondary importance. However, in Gans', Redlich's, Rochon-Duvignaud and the authors' cases they were predominant. Generally, the mental symptoms are more severe in diffuse than in disseminated types. The resulting dementia, if observed, in patchy processes of demyelination is in most cases not so marked as in the diffuse type and the mental symptoms develop sooner in the latter.

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Changes of the eyeground (choked disc, optic neuritis, bilateral atrophy or bitemporal atrophy) is relatively more common in the diffuse than in the patchy type. Persistent blindness as opposed to transitory amaurosis, with or without changes of the fundus, is seen very seldom in patchy demyelination, but occurs more frequently in the diffuse type in adults as well as in children. On the other hand, many cases of diffuse sclerosis are reported without alterations of the fundi (Gozzano and Vizioli, Rochon-Duvignaud, the authors' cases). Transitory deafness now and then is found in the diffuse type, more persistent in children. Epileptiform manifestations are more frequent in children, though not uncommon in adults (D'Antona, Bouman, Curschmann). With respect to articulatory speech disturbances, pupillary reactions, abdominal reflexes, Babinski and deep reflexes, paresthesia, nystagmus, disturbances of swallowing, tottering gait, cerebrospinal fluid findings and presence of fever, there is no marked difference between their type and occurrence in either patchy or diffuse demyelination. The tremor of hands, head and trunk occurring frequently in disseminated sclerosis is only occasionally found in the diffuse (Shelden *et al.*). The paresis of the abducens is often mentioned in the diffuse type but its typical alternate appearance and disappearance is exceptionally seen (Austregesilo *et al.*). Facial paralysis was found at times in both disseminated and diffuse demyelination but usually of central type (Bouman, Wilson).

Remissions, which are rare and often of a very short duration in diffuse demyelination (Austregesilo *et al.*, Shelden *et al.*), but longer in Ottonello's cases are more frequently observed in disseminated cases.

Taking into consideration the many clinical analogies between diffuse and disseminated types apart from the cases where a so-called combination was present (Gans, Kufs, Benoit, etc.), it is easy to understand why the diagnosis of disseminated sclerosis is often made in cases of diffuse sclerosis. Schilder himself has asked whether diffuse sclerosis is not a form of acute disseminated sclerosis, especially incident in childhood. Marburg, too, accepts the opinion that a great many of the cases of diffuse sclerosis are really instances of disseminated sclerosis. Bouman, however, thinks that from a " practical point of view it is preferable to separate the two forms though they are very similar."

Histopathologically the fundamental and most striking structural changes in multiple and diffuse sclerosis is the demyelination accompanied or not by involvement of the axis cylinders and followed by a more or less marked gliosis.

The histopathologic picture appears variable mostly in relation to the distribution, extension, intensity and duration of the process responsible for myelinolisis.

From a morphologico-topographic point of view the demyelinated areas may be differentiated in (*a*) multiple or patchy demyelinations, variable in form and size. The areas may occasionally be large, scattered here and there in white matter. At times interspersed among the larger ones are isolated smaller patches. At times neighboring small areas coalesce, forming larger ones which gradually may involve very large areas of the white matter, assuming the aspect of so-called " transitional type. " (*b*) Diffuse demyelination which may involve the whole subcortical white matter leaving generally intact the " U " fibers. In this group are included Schilder's encephalitis periaxalis diffusa (sporadic and familial cases), encephaloleukopathia scleroticans progressiva, degenerative subcortical encephalopathy, symmetrical cerebral centro-lobar sclerosis, etc. (*c*) Diffuse demyelination, interspersed by islands of more or less preserved myelin sheaths generally surrounding blood vessels (Pelizaeus-Merzbacher variety). (*d*) Concentric demyelination formed by bands of more or less normal myelin alternating with demyelinated stripes. This type of " concentric demyelination " (Balo) or " map-like demyelination " (Marburg) is a rare incidence found by the authors and by other authors in cases of disseminated or transitional type in both acute and chronic cases.

After Schilder's detailed description of " encephalitis periaxialis diffusa," many authors accepted his original description of the main histopathologic characters of diffuse sclerosis, namely : (1) large extension of demyelination ; (2) limitation of the pathological process only to the white matter of the cerebral hemispheres, with integrity of the cortex and " U " fibers ; (3) relative integrity of the axis cylinders ; (4) presence of large glia cells and increase of the fibrillary type of neuroglia ;

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(5) infiltration of the vascular walls with compound granular corpuscles and lymphocytes.

However, in the multiple or patchy and transitional type of demyelination, similar fundamental histologic changes are encountered. Although some authors have attempted to establish differences between disseminated and diffuse sclerosis on the basis of histopathologic features, it seems to us that such a differentiation does not hold true in the light of comparative studies. The review of some of the most important histopathologic features described as typical of diffuse sclerosis will establish their similarities and identities with the histopathologic features of disseminated sclerosis.

1. *Extension and Distribution of the Demyelinated Areas.*—As mentioned above, the histopathologic diagnosis of Schilder's disease is based on the fact that demyelination is intense, diffuse, and is limited to the subcortical white matter. But many cases of diffuse sclerosis have been reported in which, in addition to diffuse demyelination, presence of disseminated isolated patches identical with those of multiple sclerosis were present (Bouman, Gagel, Kufs, Wertham, Schaltenbrand, etc.). The isolated patches were distributed not only in the subcortical white matter but also in the brain stem (Bouman), optic nerves, internal capsule (Gagel), brain stem and spinal cord (Wertham), cortex, brain stem and spinal cord (Gozzano and Vizioli), internal capsule and cerebellum. The presence of these small isolated and disseminated patches of demyelination has been considered by Wertham, Kufs and by the authors as indicative of the mixed or transitional type of demyelination. Such an occurrence has been illustrated by the authors in which the fusion of small isolated patches resulted in large demyelinated areas with a tendency to assume the aspect of a diffuse demyelination. It is presumed that these "transitional forms" complete a passing stage in the development of a diffuse sclerosis.

2. *Involvement of the Ventricular Walls.*—Hallervorden suggested "the integrity of the ventricular walls in diffuse sclerosis" as a differential character between diffuse and multiple sclerosis. However, marked involvement of this region in diffuse sclerosis has been reported by various authors (Stewart *et al.*, Ferraro, Davison and Schick, D'Antona, etc.), and in the authors' case.

3. *Diffusion of the Demyelinating Processes.*—It was felt that generally the areas of demyelination in Schilder's disease have the tendency to diffuse by expansion, whereas large patches in disseminated sclerosis are constituted by coalescence of smaller ones. But in Schilder's disease D'Antona reported the presence of at least three originally isolated foci which fused into a large one involving both cerebral hemispheres. In other cases of diffuse sclerosis, the appearance of large bizarre and irregular patches seems to point to the same mechanism. In Gozzano and Vizioli's case it was noticed that foci of limited dimensions were reunited with the larger central areas through a narrow bridge of demyelination. The authors had also the impression that the peripheral foci were not the expression of a diffusion of the central areas but represented foci in the process of coalescing with the main one.

Another important character in Schilder's disease (Siemerling and Creutzfeld, Walter, Foix and Marie, Scholz, etc.) is the tendency of the demyelinating process to expand along nervous pathways, such as the internal and external capsule. On the other hand, the inferior longitudinal bundle is considered almost constantly preserved. But again this "feature" is really not characteristic because in the case of Gozzano and Vizioli the internal capsule was intact. In case 4 it was only slightly involved, whereas both inferior longitudinal bundles were affected, though in lesser degree than the surrounding areas.

4. *Involvement of the Axis Cylinders.*—Schilder reported in his cases of encephalitis periaxialis diffusa that the axis cylinders were more severely damaged than in cases of multiple sclerosis. Other authors (Gozzano and Vizioli, Cardona, Scholz, Gagel, etc.) have the impression that the involvement of the axis cylinders is in relation to the extension of the process of demyelination, i.e., where the demyelination involves the fibers for a short course the axis cylinders are less involved and better resisting, in contrast with more severe damage when they are deprived of myelin sheaths for a longer course. In the authors' cases the findings were similar to those reported by Weiman, Bielschowsky and Henneberg, Ferraro and Jervis, etc.), namely, partial integrity of the axis cylinders in the initial phase

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of the demyelination followed by more or less pronounced destruction in proportion to the myelinolytic process. Sometimes at the periphery of the patch the axis cylinders are much better preserved than the myelin sheaths, while in the center they disclose marked swelling, fragmentation, a granular appearance or " boules terminales " (Weiman). At times they completely disappear. Now and then, however, the axis cylinders appear undoubtedly comparatively better preserved than the myelin sheaths. The authors felt that the severity of the alteration of the axis cylinders is in relation to the intensity and duration of the myelinolytic process.

5. *Demarcation of the Demyelination.*—It is generally considered that in the diffuse type of sclerosis the demarcation of the demyelination is smoother and less distinct than in the patchy type where it appears neat and sharp. Here again many cases of diffuse sclerosis have been reported with neat and sharp demarcation and in the authors' cases of multiple sclerosis the demarcation was hazy and indistinct.

6. *Glia Reaction.*—For some authors the gliosis is considered to be more intense in multiple sclerosis than in diffuse. But it has been noted over and over again that this is not always the case. In the authors' cases they encountered also very intense gliosis in the diffuse type. Although generally there is no appreciable and definite differences between the gliosis of disseminated and diffuse types, distinction must be made between the central parts of the patch and its periphery and the age of the plaques. Generally, the central portion of the diseased areas is occupied mostly by proliferating glia fibrils. The most recent areas or the peripheral zone of the more or less active areas contain numerous astrocytes but not dense fibrils, whereas the older areas are poor in nuclei and contain compact bands of more delicate fibrils.

As far as the presence of " globoid cells " is concerned, the authors mention only that these elements which are considered to be a characteristic of diffuse demyelination (Collier and Greenfield, Coenen and Mir, Barre *et al.*, Cardona, etc.) were noticed also in acute disseminated encephalomyelitis (Ferraro and Jervis, Gill and Richter) and multiple sclerosis (Marburg, Austregesilo *et al.*, etc.). Collier and Greenfield reported that these " globoid " elements were encountered particularly where there was active myelin destruction or where the demyelination appeared to be of more recent date. Austregesilo and co-workers believed that they are identical to the " gemastete Gliazellen " of Nissl and " giant gliocytes." Barre *et al.* insisted upon the straight analogy of these giant glia cells with Alzheimer's cells described in the pseudosclerosis of Westphal-Strumpel and with those encountered in tuberous sclerosis of Bourneville and Brissaud. In the authors' cases of diffuse demyelination as in the cases of Coenen and Mir and of Collier and Greenfield, the authors found astrocytes and " globoid cells " intermingled with compound granular corpuscles and interfascicular oligodendrogliacytes which in certain instances appeared also swollen.

The question of the role of the glia elements in the pathogenesis of the demyelinating processes and whether an abiotrophic deficiency (Collier and Greenfield, Greenfield, Levaditi, Bielschowsky, Jervis) or a metabolic disfunction or insufficiency (Scholz, Coenen and Mir) of the oligodendroglia cells (particularly) is responsible for the development of diffuse demyelination is still open for discussion.

7. *Blood Vessel Changes and Perivascular Infiltrations.*—In one of the authors' cases of multiple and transitional demyelination the blood vessels were apparently increased in number and size as in a case of diffuse demyelination reported by Bouman. In all of the authors' cases of multiple, transitional and diffuse demyelination, the adventitial sheath of the blood vessels was variably infiltrated by lymphocytes, plasma cells, a few " mast " cells and scavenger cells which were scarce in some places and more abundant in others. In addition, in the sheaths of the blood vessels oval nuclei similar to swollen endothelial nuclei were found. Similar findings were reported also by many other authors in both multiple and diffuse demyelination (Dawson, Marburg, Schilder, Jakob, Siemerling and Creuzfeld, Ferraro).

Now and then the character of the perivascular elements varied; at times predominance of compound granular corpuscles or lymphocytes was found, at others these infiltrative cells were mixed with other kinds of cells as mentioned above. This occurred in both patchy and diffuse demyelination.

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Without entering into detail the authors will mention only that in some cases of multiple or diffuse demyelination the perivascular infiltrations were considered as expression of inflammation (Schilder, Bouman, Rhodes, Jacob, Stauffenberg, Gozzano-Vizioli, etc.), of degeneration (Haberfeld and Spieler, Krabbe, Scholz, Globus, Bielschowsky and Henneberg, Hassin, etc.) or of symptomatic inflammation (Spielmeyer and Ferraro). In certain cases it was, however, impossible to give a decisive opinion (Weil), and Spielmeyer himself was often in doubt as to whether the exudative infiltration was related to the reaction of " breakdown " products or expression of independent inflammation.

It is very curious that in some instances the opinions of different authors concerning the same cases were completely at variance. For instance, the case of Stauffenberg was regarded by Weimann as of a degenerative nature, whereas Steiner considered it as an inflammatory one. Weimann referred Bouman's first case of diffuse demyelination to the degenerative group, while Bouman himself considered it as belonging to the inflammatory one.

To date there has been no complete agreement among the neuropathologists concerning the concept of inflammation, degeneration or symptomatic inflammation of the nervous tissue. Ferraro has suggested the term of " hyperergic inflammation " of allergic nature to reconcile the two extreme viewpoints. Also, Marburg, by introducing the new term of " Parencephalomyelitis " intended to indicate that in this condition there is an " accompanying but not a genuine inflammation " of the nervous system. He believed that this reaction might be similar to that elicited by Schwartzmann's phenomenon with sterile toxins.

8. *Relationship of the Demyelinating Areas with Vascular Pattern and their Histogenesis.*—The patches of demyelination in multiple or patchy sclerosis have been considered to be vascular in distribution, whereas in the diffuse type the demyelination is supposed to be independent of the vascular pattern.

But this statement is not supported by many histopathologic reports. Kufs in his first case of diffuse sclerosis observed that one small thalamic focus was in relation to a vein. Similarly in one of Bouman's cases, in Stewart *et al.* and in Perdrau's monkey, isolated patches were in relation to blood vessels. On the other hand, while this perivascular distribution, in the diffuse type has been observed mostly in relation to isolated foci, many disseminated patches of multiple sclerosis have been reported as independent of vascular pattern.

This latter problem has been much discussed since the time of Rindfleisch and Ribert who claimed that the demyelinated areas were related to a primary vascular lesion or thrombosis. But Dawson, Symonds and Wilson, although recognizing a frequent relationship between the lesions and small blood vessels, expressed the belief that the changes were secondary to inflammation produced either by the noxious agent or by the breakdown of the damaged tissue.

Recently, Putnam and co-workers found that demyelination, with or without destruction of the axons, followed experimental obstruction of the small vessels and especially venules. Also, in multiple sclerosis and disseminated encephalomyelitis Putnam, Putnam and Alexander and Scheinker emphasized the occurrence of thrombi in the smaller veins and considered them the primary cause of demyelination. In support of this hypothesis, Simon and Solomon and Dattner found in disseminated sclerosis increased coagulation of the blood.

Marburg considered phlebothrombosis as only one of the etiologic factors of the demyelination in disseminated sclerosis. Hassin instead maintained that " blood vessels play no role in the genesis of the changes in multiple sclerosis, in which the nerve fibers are attacked primarily and directly, without the intermediation of the blood vessels."

In 1942, Dow and Berglund studied the vascular pattern of lesions of multiple sclerosis in serial sections. " Although the lesions were not always oriented about the veins, they found that two-thirds of them were." These authors were inclined to attribute the thrombosis to a " local absorption of thromboplastic substances revealed by the breakdown of myelin." Ferraro, in his attempt at considering the pathology of demyelinating diseases (both patchy and diffuse) as expression of allergic reaction, is in agreement with Abel and Schenck's as well as Putnam's views that the thrombi might be one aspect of allergy. In the authors' cases they found that a large number of demyelinated areas were in relation to blood vessels (arteries or veins) but only occasionally did they observe presence of thrombi.

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Many authors have also failed to detect thrombi in cases of disseminated sclerosis. This failure Putnam believed to be due to technical difficulty in demonstrating certain thrombi and to their rapid disappearance from the lesions. It seems necessary to mention that while the writers who disagree with Putnam's views speak in general terms of " blood vessels or arteries," the latter insists more specifically on the involvement of the " venous drainage."

More recently, Hurst and Ternnent Cooke studied " capillary fat embolism in the brains of sheep, pigs and monkeys with special reference to demyelination." In their experiments " the lesions ranged from slight demyelination to full necrosis." In addition, they observed that " obstructive plugs (not thrombi) present in the early stage of the reaction, disappeared within a few days from lesions to the formation of which they have obviously contributed."

However, the theory of venous obstruction would still have to explain : (1) why the white matter is almost exclusively involved in many cases ; (2) the mechanism of formation of Balo's concentric type of demyelination or of Marburg's map-like (" Landkartenherde ") formations, and (3) the occasional preserved perivascular myelin islands encountered in the Pelizaeus-Merzbacher type of demyelination.

9. *Cortical, Extracortical and Spinal Cord Lesions.*—In the diffuse type of demyelination many authors reported that the pathologic process was limited only to the white subcortical areas, whereas the involvement of " extracortical " areas or of the spinal cord was considered secondary and of degenerative character. For this reason they called it also progressive degenerative subcortical encephalopathy, encephalopathia subcorticalis diffusa, etc.

In this connection the authors will mention only that involvement of the gray matter in both diffuse and patchy demyelination was observed in the cases of Gagel, Coenen and Mir, Klarfeld, Weimann, Bouman, Neuburger, Walter, Bielschowsky and Maas, Schelden *et al.*, Kraus and Weil, Wertham, etc., and in the authors' cases.

In addition, several cases of diffuse demyelination were reported in which the demyelinating process also involved the spinal cord. The lesions were not exclusively of a secondary degenerative character but were identical with primary patches of demyelination (Stewart *et al.*, Popi, Jacob, Wertham, Gozzano and Vizioli).

The supracortical pia-arachnoid has generally been reported as normal, but quite often various changes were noticed also in both patchy and diffuse demyelination. Such changes consisted generally of simple thickening of the meninges, lymphocytic infiltration or association of both (Austregesilo *et al.*, Neuburger, Kaltenbach, Bouman, Schilder, Braun, Schaltenbrand), presence of fat granular cells or of fat droplets with slight edema of the meninges (Kraus and Weil) and patches of dense infiltration by macrophages as well as by lymphocytes and polyblasts. In the authors' case the pia-arachnoid in many regions was intact, but over the forebrain there were patches of more or less perivascular and free infiltration of lymphocytes, macrophages, a few plasma cells and some polyblasts.

10. *Products of Degenerative or Disintegrative Metamorphosis*, such as different types of fat-staining material, or prelipoids or " Markballen " (Greenfield and King), mucinophile, argentophile and siderophile products, corpora amylacea (" protagonoide Abbaukugeln " Kaltenbach) and metachromatic substances were found by the authors in both disseminated and diffuse demyelination as well as in the transitional type. Their distribution and histochemical character is generally identical in both types of demyelination. In the authors' estimation, they cannot be used as " typical " differential elements for one or another type of demyelination as Cardona does concerning the intensity of the " metachromatic substance " which he considered to be specific for " Schilder's disease."

11. *Cystic Formations.*—They were described in both patchy and diffuse demyelinating processes (Schilder's first case, Rossolimo, Bouman's case 1, Bodechtel and Guttman, Braun, Borst, etc.). Sometimes these were surrounded by several layers of cylindrical cells giving the impression of small gliomas (Barre *et al.*). A cystic formation was found in the authors' cases of diffuse sclerosis (case 3).

12. *Hemorrhages, Necrosis and Edema.*—Hemorrhages were observed by various authors in cases of diffuse, transitional and patchy demyelination. However, more often congestion of blood vessels is found either in the parenchyma or in the meninges. Perivascular hemorrhages were noticed by Putnam, Zimmerman

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and Janet, Davison and Friedfeld in cases of encephalomyelitis which Putnam and Ferraro include in the group of the " acute multiple sclerosis."

Necrotic areas may be present, especially in the very acute cases or in the stages of reacutization of multiple sclerosis (Marburg, Ferraro). Similar findings were also described in cases of diffuse demyelination (Schilder, Barre *et al.*, Bodechtel and Guttman, Bielschowsky and Hanneberg, Wohlwill, Gerstman and Straussler).

Edema is also found especially in acute cases either free in the parenchyma or in the perivascular spaces (Ferraro, Roizin, Moriarty and Weil, the authors' case).

From these data it is obvious that not only clinically but also anatomically there is a very close relationship between diffuse and disseminated demyelinating processes. It is to be noticed also that the given clinical differential diagnostic points between the diffuse and disseminated types appear to be not of decisive value because they are found in both conditions. Histopathologically close relationship between diffuse and disseminated types must be admitted because disseminated patches and transitional stages are found in some cases of diffuse demyelination and because of the presence in cases of typical disseminated type of demyelination of transitional and diffuse demyelinated areas.

It is not the authors' aim to discuss here in detail the great complication of nomenclature of diffuse and multiple demyelinations which Ferraro attempted to unify and classify some years ago. However, they wish to state that in the absence of more specific and definite data concerning the etiopathogenesis and histogensis of this insidious disease of the nervous tissue it seems more justifiable, particularly from a histopathologic point of view, to use the term " primary demyelinating diseases " (as Ferraro does) in place of other various arbitrary names, because demyelination is the most striking alteration, and, irrespective of whether it is patchy or diffuse, acute, subacute or chronic in character and sporadic or familial in type, the process is fundamentally the same.

From a descriptive point of view the naming of cases, whether " patchy " or " diffuse," should be based according to the predominance of symptoms or lesions in the case. The term transitional will of necessity have to be used, particularly from the pathologic standpoint, because the clinical symptoms fail at times to furnish precise indication of this occurrence. The term acute, subacute or chronic will be determined mostly by the clinical course. The classification of the familial cases into infantile, juvenile or adult type will be considered as respective varieties of the same clinico-pathologic syndrome and not as individual clinical or pathologic entities. (Authors' abstr.)

AUGUST.

The Treatment of Psychiatric Disorders due to Combat by Means of a Group Program and Insulin in Sub-Shock Doses. *Teitelbaum, T. A., et al.* . 123
*Use of the Bellevue-Wechsler Scale in Clinical Psychiatry. *Goldman, R., and Coon, G. P.* 144
Psychiatric Nomenclature 180
Syphilitic Meningitis with Negative Blood and Spinal Fluid. *Kaplan, L. I., and Becker, T. F.* 200

Use of the Bellevue-Wechsler Scale in Clinical Psychiatry.

Experience with the Bellevue-Wechsler test used over a period of 8 years at the Boston Psychopathic Hospital, leads the authors to believe that it is a superior test because it can be administered with relative ease, it has been standardized on adults over a wide age range, it has the flexibility of a point scale with possibility of direct comparison of performance on various test items; and also because it taps a wide range of functions and elicits data of considerable value and interest to the clinician. In the group of cases characterized by the term " brain damage," this test would seem to be of special value for both diagnosis and investigation.

Both quantitative and qualitative signs are found in patients with " brain damage." The quantitative signs consist essentially in marked discrepancy in the weighted scores between various sub-test items. As a rule, performance on non-verbal items (picture arrangement, picture completion and block designs) is most markedly affected, but there is always at least *one* verbal sub-test on which there is also loss in ability to function. The qualitative signs are best displayed on the

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non-verbal test items where the patients show rigidity, inability to shift attention or change the mode of responding, inability to ignore superficial or extraneous stimuli and difficulty in organizing material into either a required pattern (block designs) or into a meaningful logical sequence (picture arrangement). The same signs do appear in the verbal material but are more difficult to recognize and may often be missed.

Where both quantitative and qualitative irregularity of performance are found, clinical analysis reveals a very high incidence of brain damage.

Within the broader framework of the "organic" pattern as elicited by the Bellevue-Wechsler test, the authors have noted 7 variant configurations or profiles which are more or less mutually exclusive. The full significance of these profiles is not clear as yet; certainly they are not specific for any given disease entity. As a rule, the greater the loss of function as displayed on the psychological examination, the more serious is the brain damage. There are suggestive relationships between the particular psychological configurations and certain groups of symptoms, but it is felt that the meaning of these trends must await further study.

(Authors' abstr.)

SEPTEMBER.

*Paroxysmal Slow Waves in the EEG of Patients with Epilepsy and with Subcortical Lesions. *Lennox, M., and Brody, B. S.* 237
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Electroshock and Personality Structure. *Mosse, E. P.* 296
The Concept of Induced Insanity in Family Units. *Kallman, F. J., and Mickey, J. S.* 303

Paroxysmal Slow Waves in the Electroencephalograms of Patients with Epilepsy and with Subcortical Lesions.

A study has been made of psychomotor waves encountered in 3,000 EEGs over a 2-year period. The characteristics of "psychomotor" activity in 135 epileptics are summarized, and the tendency of psychomotor waves to form shifting foci is stressed. Similar activity has been encountered in addition in 17 proven and 14 probable cases of subcortical pathology. The slower (2–3 per second) psychomotor waves tend to be associated with lesions in the region of the basal ganglia, thalamus and cerebellar hemispheres, whereas 4–6 per second psychomotor waves tend to be associated with lesions in the region of the hypothalamus, pons and (in one case) the cerebellar vermis. It is suggested that psychomotor activity in epileptics probably arises in deep structures. It is emphasized that a focus consisting of or associated with psychomotor activity is usually inconstant and does not indicate focal cortical pathology in the majority of instances.

(Authors' abstr.)

Preliminary Report on the Effect of Glutamic Acid Administration in Mentally Retarded Subjects.

It might be expected that a substance affecting brain metabolism would be more effective in patients whose intellectual potentialities were basically normal, but had been damaged by brain disease or injury, than in patients with a primary mental deficiency. Furthermore, we have found that in functional brain damage, artificially produced by electric shock, glutamic acid administration may exert a favorable influence on confusions, memory and other mental performances. For these reasons we attempted to select for our preliminary study patients with secondary mental deficiency, but in some cases it is very difficult to differentiate between these two types of deficiency and in some of our patients the diagnosis was not certain.

In 7 of the 8 patients studied an unmistakable rise of varying degree in the level of mental function followed the oral administration of glutamic acid in amounts

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of 8 to 10 gm. a day. The effect depended on the continuous administration of the amino-acid; when the patients were put on placebos they eventually slumped back to their previous level of performance. This tendency to relapse was not always discernible immediately after discontinuation of the glutamic acid.

On an empirical basis psychometric tests are accepted to-day as giving a good insight into mental capacity even though they do not test the whole performance ability of the patient. To safeguard against errors which are not uncommon in work on mental deficiency, our subjects were repeatedly tested with a battery of different psychometric tests which agreed closely in evaluating the mental capacity. It should be emphasized that most of our patients had been repeatedly tested by different psychologists over a period of years before the present study was undertaken. The IQ was found to be practically static or the gain was very small. The patients received adequate educational attention, and one of them underwent psychotherapy prior to the experiment. With this background the rapid rise in IQ following the administration of glutamic acid can hardly be ascribed to other factors, such as maturation or environmental influences.

It may be argued that the gain in IQ was not valid but resulted from an increased familiarity of the patient with the tests. We believe that this possibility is excluded for the following reasons: (1) The patients were tested several times prior to glutamic acid administration and none of them showed a significant variation in IQ. These pre-treatment tests were spaced at the same time intervals as those carried out later during the glutamic acid periods. (2) Each time the patient was retested, not only the original Binet but other tests with which the patient was not familiar were used. (3) The best proof, however, rests in the fact that the patients dropped in IQ during the placebo period and regained the loss again when glutamic acid was administered.

In analyzing the intellectual gain resulting from the administration of glutamic acid, the question must be asked whether this gain represents an acquisition of new intellectual tools, i.e., an increase of intelligence, or the better utilization of existing intellectual abilities. It was noted that patients who, before administration of glutamic acid, appeared to be dull, inattentive and easily fatigued became more alert, attentive and persistent in trying to accomplish the tasks put before them. It was also observed that in the pre-treatment phase some of these patients appeared at times to be irritable, restless and emotionally labile. During administration of glutamic acid they behaved in a manner which suggested an improved emotional regulation. It seemed to us that the performance of the total personality, rather than of some circumscribed intellectual functions, showed improvement.

In recent investigations on the structure of memory in patients who were exposed to electric shock treatment, similar observations were made by ourselves and Dr. Zubin. Memory impairment and more or less severe confusions after electric shock treatment are known to occur in a large number of patients. 10 to 33 c.c. of a 10 per cent. solution of glutamic acid were administered intravenously to some of these patients at the height of their confusional state, which usually occurs when they have already had 6 to 12 electric shock treatments. The impression was gained that the administration of glutamic acid shortened the period of confusions and memory impairment, and in some patients psychological tests showed an immediate and definite improvement. The memory-defective and confused patients often showed an emotional imbalance such as increased irritability, rapid mood swings, depressed or impulsive behavior, distractibility and inattention. Usually these emotional symptoms improved first and the change in behavior for the better preceded the improvement in the intellectual performance.

It is known to-day that the ability to perform intellectually depends upon many factors, among which the emotional status of the individual plays a significant role. Some patients suffering from organic brain damage show, owing to emotional inhibitory factors, a much more pronounced organic incapacitation than can be explained by the tissue destruction. In hypnosis or under the influence of sodium amytal patients suffering from aphasia, apraxia and many other neurological disturbances may perform much better than otherwise. Memory lapses can also be reduced by these methods. In rehabilitating such patients this factor is now taken into consideration.

From the foregoing considerations it appears possible that glutamic acid does

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not so much influence or increase the intellectual ability as such, but only enables the patient to regain his intellectual function which was inhibited by other mechanisms. But inhibitory mechanisms similar to those observed in diaschisis may play a part in addition to the emotional factors. The nervous system often responds to damage with increased inhibitory and excitatory manifestations until a new neural equilibrium is reached. The removal of inhibitory mechanisms in acute cases of brain damage is not surprising, but it is rather impressive that it can occur to some extent in patients with a long-standing brain damage, as in the boy who had an encephalitis 25 years before.

It could be argued that the patients in whom the glutamic acid proved to be beneficial were suffering from traumatic epilepsy. Organic brain damage even without epileptic manifestations sometimes causes intellectual and emotional changes which are similar to those seen in some patients who have epileptic attacks. It could, therefore, be claimed that the improvement with glutamic acid is a result of removing the epileptoid mechanism. There is some evidence that this is not the case. Price, Waelsch and Putnam have pointed out that increase in intellectual efficiency is not always accompanied by a decrease in *petit mal* seizures; and furthermore, our patients did not show a characteristic epileptic pattern in the electroencephalogram.

For practical and for theoretical reasons it is of interest to consider the toxic symptoms observed after the administration of glutamic acid. The oral administration in mentally defective children did not cause any toxic effects. However, in the psychiatric patients, after electric shock treatment, we have occasionally seen gastro-intestinal disturbances, complaints of fullness in the stomach, vomiting and diarrhœa, which, however, subsided rather quickly on reduction of the dose. The intravenous application of glutamic acid caused in some individuals toxic effects of the same type. The individual sensitivity was very variable. Patients with hypersensitivity to the amino-acid complained of flushes, vomiting, feelings of dizziness and of general malaise. These symptoms could be avoided in some cases if the glutamic acid was given in more dilute solution than 10 per cent. and if the injection was given slowly, at a rate of about 1 c.c. per minute. But there remained sporadic cases in whom the toxic reactions occurred even with this technique. These observations on the toxicity of glutamic acid by intravenous injection were made during the last two years, during which time similar observations were reported by others in experiments carried out with animals.

It is impossible at this stage of the investigations to point to any biochemical mechanism as an explanation of the effect in epileptic patients of the administration of glutamic acid, and the same is true for the results obtained with mentally defective subjects. It may well be that other body constituents which may or may not be metabolically related to glutamic acid have the same effect. Up to the present we do not know whether the beneficial results are due to the fact that glutamic acid is administered as free amino-acid and whether they would be also obtained if the amino acid were offered in its linkage in proteins. For these and other reasons it cannot be decided whether we are dealing with a deficiency of glutamic acid which is overcome by its administration. Such a possibility is of major interest from the nutritional point of view since glutamic acid is not only supplied in large amount with the food but is also synthesized in the body.

There are few biochemical studies which may point to a specific role of glutamic acid in brain metabolism. It has been claimed that glutamic acid is the only amino-acid oxidized by brain slices, whereas chopped brain appears to be able to oxidize a variety of amino-acids. A more specific function of glutamic acid in brain metabolism may be deduced from experiments showing that the enzyme system which synthesizes acetylcholine in brain extracts, if inactivated by dialysis, is partially reactivated by the addition of glutamic acid. This finding, if transferable to the intact organism, may offer an explanation of the toxic symptoms, resulting from the intravenous or oral administration of glutamic acid, encountered in psychiatric patients after electroshock.

In this report the authors do not want to raise any extravagant hope that cases of secondary mental deficiency and, by implication, other forms of feeblemindedness, may be cured with glutamic acid; but there is some indication that a limited influence can be demonstrated on some of these patients. Mental defects have been considered unalterable, and research and treatment have been concentrated

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on the social and educational aspects, which are of great importance. Nevertheless, the authors felt that attempts should be made to test the possibility of improving brain function by biochemical means. The results obtained with glutamic acid are considered to be a starting point for further investigations. Long and tedious research will be necessary to solve the question of the specificity of glutamic acid and to test other substances which may benefit some patients and may also elucidate basic neural mechanisms in relation to mental activity. (Authors' abstr.)

OCTOBER.

*The Influence of Emotion on the Human EEG. *Cohn, R.* . . . 351
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*Porphyria. *Schneck, J. M.* 432

The Influence of Emotion on the Human Electroencephalogram.

1. Hyperemotional states give rise to two characteristic types of electroencephalographic pattern. Type I is characterized by a " spontaneous " low voltage random high frequency pattern. When the attention of the patient is removed from the test and his environment, a normal brain wave pattern ensues. The Type II pattern is characterized by rhythmic, approximately 20 per second activity predominantly in the frontal and parietal regions. This pattern is stable and uninfluenced by afferent stimuli of ordinary intensity.

2. The recognition of these electroencephalographic patterns is important from neuropsychiatric considerations.

3. The rhythmic high frequency potentials observed in hyperaffective disorders is a product primarily of frontal lobe activity.

4. Physiologically increased frontal lobe activity, as evidenced by the superimposition of somatic complaints on a basic emotional disorder, results in an enhanced frontal electric potential output. Hence, the rhythmic high frequency oscillations of the frontal regions may be considered as " true " action potentials. (Author's abstr.)

Porphyria : Neuropsychiatric Aspects in the Case of a Negro.

The diagnosis of porphyria rests on the sum total of signs and symptoms which may be recorded briefly as follows : a hysterical personality with remissions and exacerbations of marked muscular weakness accompanied at times with pronounced but temporary visual impairment and excretion of red-colored urine ; an unfavorable response to barbiturates, variability in reflex responses with the recently emphasized absence of knee jerks in the presence of ankle jerks and a summation effect reinstating the normal response of the former ; thenar and hypothenar atrophy indicating early anterior horn cell involvement. In addition to these, the other signs and symptoms mentioned in the report add further weight to the diagnosis. Regarding bone changes in porphyria, Nesbitt says : " The bone marrow may appear hyperplastic, and occasional large cells laden with brown pigment may be seen."

An especially interesting fact in this patient is the sequential relationship of the anxiety episodes to the onset of the physical manifestations of the illness. (Author's abstr.)

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The Relation Between the Electroencephalogram and Muscle Action Potentials in Certain Convulsive States.

1. The EEG's of 2 subjects with severe myoclonic seizures and 1 subject with mild myoclonic jerkings accompanying wave and spike discharge in the EEG are described.

2. Repeated simultaneous recordings of the EEGs and muscle action potentials have been made in these subjects during myoclonic episodes.

3. All subjects show some form of spike discharge, with or without a slow wave, in the inter-seizure periods. In the seizures the patients with the severe myoclonic jerkings show discharges of spikes in their EEGs with a period of 30 to 75 msec. at a rate of 8 to 13 per second.

4. There is an accurate correlation between the occurrence of the spike-like discharges in the EEG and the action potentials of the muscular jerkings in the severe cases and a less accurate correspondence in the mild case.

5. The muscle action potentials show recruitment and facilitation having a different time course of decay for flexor and extensor muscles.

6. In the periods between the spontaneous jerkings changes in spinal reflex excitability may be demonstrated.

7. Important differences in the characters of the muscular jerkings in the severe myoclonic states and the milder state accompanying wave and spike discharge are described.

8. These findings are discussed and their bearing on the origin and mediation of the abnormal discharges in these conditions are considered.

(Author's abstr.)

The Disorganization of Behaviour in Fatigue.

The skilled activity required by an exercise in instrument flying was observed to become disorganized in an experimental situation which was assumed to be fatiguing. The disorganization had two phases, in each of which it was associated with characteristic changes of feeling. The two phases of the disorganization, with the associated effects, were considered to constitute two distinct reactions to the test conditions.

In the first phase, all parts of the work were similarly affected. Although there was little or no increase in their duration, errors became more frequent and were overcorrected. Activity became excessive; movements of the controls tended to be too extensive, and there was a large increase in restless and inappropriate movements. Attention was distributed irregularly over the work and was held by aspects which had acquired a temporary urgency.

Subjects felt under strain, dissatisfied and irritable, and they were preoccupied with the test after it was finished. Signs of emotion, such as agitation and restlessness, were observed, and there were indications that muscular tonus was increased. Flushing of the face was common, and visible sweating also occurred.

In the second phase, some parts of the work were affected more than others, those regarded by the subjects as of greater importance being the better preserved. Errors became less frequent, but their correction was tardy and they were larger. Movements of the controls were often as extensive as in the first phase, but were fewer. Concentration failed and interest flagged. Some subjects were satisfied with a lowered standard of accuracy, whereas others were dissatisfied with their attainment, but made an insufficient response to remedy it, either emotionally or in organized activity, and they were apathetic and discouraged. Postural changes were variable, but flushing, or sweating was rare. (Author's abstr.)

APRIL.

A Familial Disease Characterized by a Diffuse Angiomatous Non-calcifying Cortico-meningitis and Progressive Demyelinization of the White Matter. *Divry, P., and van Bogaert, L.* 41
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*Re-education in Aphasia. *Butfield, E., and Zangwill, O. L.* 75

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Aetiology and Pathogenesis of Acute Sporadic Disseminated Encephalomyelitis and Multiple Sclerosis.

As a result of our experimental, anatomical and clinical findings we have been able to draw the following conclusions:

1. This work is the first in which experimental investigation of acute encephalomyelitis and multiple sclerosis has been successfully carried out and the infectious origin of these diseases experimentally and clinically confirmed.

2. Virus investigation of patients suffering from acute disseminated encephalomyelitis and multiple sclerosis has enabled us to isolate two strains of filterable virus—from the blood of one case, and from the cerebral tissue of another case of acute disseminated encephalomyelitis. These strains have proved to be identical, and differ from other known human neurotropic viruses.

3. The syndrome of acute disseminated encephalomyelitis has been produced in animals by means of inoculation with this virus. The morphological picture obtained has proved identical, particularly in rabbits and puppies, to that found in man suffering from acute disseminated encephalomyelitis. These experiments are the first in which experimental acute encephalomyelitis has resulted from the inoculation of human material.

4. On the basis of the above data an attempt has been made to neutralize the virus thus isolated with the sera of patients suffering from acute disseminated encephalomyelitis and multiple sclerosis. The virus has been neutralized by sera from 50 per cent. of cases of multiple sclerosis and from 70 per cent. of cases of disseminated encephalomyelitis.

5. These positive neutralization tests suggest that both of these diseases result from the same agent. On the other hand, a negative test does not necessarily indicate a different causative agent. Hence, acute disseminated encephalomyelitis and multiple sclerosis have the same aetiology in some of the cases, while in others the same anatomical and clinical syndromes may be elicited by other agents, or possibly by the same agent but with a different antigenic structure.

6. A small number of cases of acute disseminated encephalomyelitis and multiple sclerosis, subjected to specific vaccine therapy, has yielded so far satisfactory results. Clinical improvement has been registered, together with a considerable increase of antibodies in the blood.

7. It is our belief that this work is far from being complete. The results put forward by us may form a basis for further investigation. (Authors' abstr.)

Re-education in Aphasia.

1. The problem of re-education in aphasia is briefly considered and some practical methods of retraining described.

2. The outcome of re-education in a group of 70 dysphasic cases is reviewed and evaluated. Treatment in 14 of these cases was begun 6 months or more after the onset of the disorder, when relatively little further spontaneous improvement was to be expected.

3. The results are considered in relation to the various aspects of language affected (speech, reading, writing and calculation), and to the type, severity and aetiology of the dysphasic condition. Although the number of cases is too small to permit a determination of the statistical significance of the results, it is suggested that the most favourable outcome is obtained in traumatic cases and in those whose symptoms are relatively mild and predominantly on the executive side.

4. Two-thirds of the patients given re-educational treatment were resettled in economic employment. (Authors' abstr.)

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*Mechanism of Pupillary Dilatation Elicited by Cortical Stimulation. *Ward, A. A., jun., and Reed, H. L.* 329
*Inhibition at the Nerve Muscle Junction in Crustacea. *Kuffler, S. W., and Katz, B.* 337

Electromyographic Studies of Muscular Co-ordination on Stimulation of the Motor Cortex.

Experiments were performed on the effect of stimulation of the motor cortex of cats and monkeys on muscular co-ordination by means of electromyograms of flexor and extensor muscles. It was found that the effects depend on duration and intensity of stimulation, as follows :

1. Very weak stimulation of the motor cortex causes relaxation of flexor and/or extensor muscles if a pre-stimulatory tonic activity is demonstrable in the electromyograms. Such cortically induced inhibition is frequently followed by a post-stimulatory rebound.

2. With slightly increased intensity which calls forth a movement (flexion or extension), reciprocal innervation of agonist and antagonist may be observed, whereas somewhat stronger stimuli cause simultaneous activity in flexor and extensory muscles, i.e., co-innervation, and consequently co-contraction, during flexion or extension.

3. Variations in the duration of the period of stimulation exert effects similar to those seen in experiments involving different intensities. Whereas reciprocal innervation appears during the early phase of stimulation it is later followed by co-innervation while the stimulation continues.

4. Even the agonist may show a transition from inhibition to excitation during stimulation at constant frequency and intensity.

5. It is shown that an inhibitory period frequently occurs on cessation of stimulation.

6. The phenomena described under 1–5 are interpreted as indicating that with increasing number of discharging neurons the peripheral effect on individual muscles changes from inhibition to excitation, and in the case of an antagonistic pair of muscles, from inhibition to reciprocal innervation, and finally to co-innervation. Under conditions permitting facilitation the predominant type of co-ordination of fore and hindleg muscles in flexor and extensor movements of moderate intensity is that of co-innervation. (Authors' abstr.)

Centrifugal Deterioration of Asphyxiated Peripheral Nerve.

Electrical thresholds of points on a circulated nerve preparation in the cat were determined as the least electrical stimulus required to evoke either a contraction of muscle or the A-wave led from the distal end of the nerve. Then the trachea was clamped and threshold observations were continued.

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The most proximal point was always the first to undergo an increase in threshold culminating in complete inexcitability. Successively more distal points underwent similar change with passage of time. Points 6 per cent. and 85 per cent. of the distance between proximal and distal ends of the nerve underwent a five-fold threshold increase in 13 and 96 minutes respectively.

Roots were inexcitable before onset of threshold elevation at the mid-point along the nerve length. In a partly deteriorated nerve, deterioration was further advanced in the central than in the peripheral half of a segment, but of the fibers blocked in the segment, some were blocked only in the peripheral half.

It is thought most likely that in these experiments a given block along a given fiber involved but a very short segment of the fiber, blocking along a greater length resulting from fusion of these short blocked segments. The field of blocking originated at the central end of the nerve and moved peripheralward. Within the field of blocking, blocks appeared most rapidly at the central end, least rapidly at the peripheral end.

This proximo-distal gradient of deterioration must be a manifestation of a gradient along the intact nerve in the living animal. (Authors' abstr.)

The Hypothalamic Regulation of Sleep in Rats: An Experimental Study.

1. In the rat complete bilateral transection of the hypothalamus, irrespective of location, interferes with the normal regulation of sleep.

2. The location of lesions which cause disturbances of the function of waking indicates the existence of a structure in the caudal hypothalamic region and in the adjacent part of the midbrain tegmentum, which is of specific importance for the capacity of maintaining the waking state during the absence of external stimuli (" waking centre ").

3. There is reason to accept a structure in the preoptic region, which is of specific importance for the capacity of sleeping (" sleep centre ").

4. Evidence is offered that sleep is caused by an inhibitory action of the sleep centre on the waking centre.

5. The lateral hypothalamic area seems to be of more importance for the regulation of sleep and waking than the inner areas. It seems probable that the medial forebrain bundle, which occupies this area, is implicated in the transmission of impulses determining the sleep-waking rhythm.

6. The hypothalamic centres involved in the regulation of sleep are topographically identical with those determining the autonomic balance.

(Author's abstr.)

Frequency of Centripetal Stimulation in Inhibition and Facilitation of the Knee-jerk.

An analysis has been made of the relations between the frequency of centripetal stimulation of a nerve-trunk and the resulting reflex effects upon the anterior horn cells innervating the quadriceps muscle in the acute spinal cat. These effects can be classified as (i) ipsilateral extensor activity; (ii) ipsilateral inhibition of the knee-jerk; (iii) contralateral extensor activity; (iv) contralateral facilitation of the knee-jerk; (v) inhibitory after-effects, and (vi) excitatory rebound.

The intensity of the excitatory process increases with frequency from 1 per sec. up to 30 per sec., without further increase up to 120 per sec. The rate of increase is slow at frequencies lower than 8 per sec. The intensity of the inhibitory process increases with frequency from 1 per sec. up to at least 120 per sec., the highest frequency studied. (Author's abstr.)

Mechanism of Pupillary Dilatation Elicited by Cortical Stimulation.

Pupillary dilatation elicited by electrical stimulation of area 8 in the cerebral cortex of the monkey is a localized sympathetic response having all the properties of reciprocal innervation. This response is abolished by section of the cervical sympathetic chain, minimal pupillary dilatation due to inhibition of the oculomotor nucleus being obtainable only under special conditions of anesthesia. The active dilator component travels over pathways involving the hypothalamus. This cortico-hypothalamic pathway is not a direct one. The reciprocal inhibitory pathway from area 8 to the oculomotor nucleus has been shown to be a direct projection. (Authors' abstr.)

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Inhibition at the Nerve Muscle Junction in Crustacea.

The effect of inhibitory nerve impulses on the potential changes and on the subsequent contractions of crustacean muscle was investigated.

1. The action potentials of inhibitory axons do not differ from those of other nerve fibres. Inhibitory impulses alone have no detectable electrical effect on the muscle. These findings confirm those of other investigators.

2. There are two separate actions of inhibitory impulses on the motor response:

(*a*) An electrical effect, the α-action, reduces the junctional potential (e.p.p.) to an extent depending upon the relative times of arrival of inhibitory and motor impulses at the junction. Due to the reduction of the e.p.p. (i) the setting up of propagated muscle impulses is prevented, and (ii) local contractions at the junctional region are diminished or abolished. At 17° C. that α-action lasts about 20 to 25 msec. and declines to half in about 5 msec. The time course of the e.p.p. is not appreciably altered by inhibition.

(*b*) The inhibitory impulse has direct action on the contractile process at the junction, the β-action. At low frequencies complete mechanical inhibition can be obtained without reduction of the e.p.p. This confirms observations by Marmont and Wiersma (9). The β-effect can sum at intervals of 0·1 sec.

3. Facilitation responsible for the growth of successive e.p.p.s is not affected by inhibition at 50 per sec. The first or second e.p.p., after cessation of inhibitory impulses, reaches the amplitude to which it would have grown in the absence of inhibition.

4. It is suggested that the α-effect is due to action of the inhibitor on the junctional receptors of the muscle membrane, and that the release of the transmitter is unaffected. (Authors' abstr.)

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Observations on Encephalographic Findings in Cerebral Trauma.

1. The encephalographic findings in a series of 261 patients, 206 of whom had had cranial trauma, have been reviewed. Of these, 177 had head injuries with skull defects, while 29 had closed head injuries.

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2. Abnormal encephalograms were found in 74 per cent. of the cases of head trauma.

3. Unilateral dilatation of the ventricles was found in 29 per cent. of the cases of head trauma. Symmetrical bilateral dilatation of the ventricles was found in 16 per cent. of these cases; asymmetrical bilateral dilatation in 29 per cent.

4. In a group of 60 cases in which second-day study of the ventricles was made, an increase in the ventricular size on the second day was found in over one-third.

5. Many of the encephalographic abnormalities were found within a short time after injury. (Authors' abstr.)

Cortical Localization by Electroencephalography. The Value of a Quantitative and Statistical Analysis of Homologous Recordings Obtained Simultaneously.

1. A modified electroencephalographic technique based upon the quantitative and statistical analysis of homologous recordings obtained simultaneously has been investigated for its cortical localizing value. The principle involved is identical with that employed in the neurological examination in which focal abnormal findings on one side are checked against the normal side as a control. The technique methods of analysis and principles of evaluation are presented in detail.

2. This technique has been employed in 1,200 examinations on patients who have received an adequate clinical work-up. Definite cortical localizations were obtained in approximately one-third of this abnormal group, which gave a high degree of correlation with the clinical findings. 90 per cent. of the remainder had convulsive states or other conditions in which focal signs and symptoms or neurological findings of localizing value were not obtained.

3. In 248 patients of this group subjected to pneumoencephalography, an over-all correlation of 81 per cent. was observed between the roentgenographic findings and those of the electroencephalogram obtained by means of the technique of homologous recordings.

4. In 85 proved cortical lesions an electroencephalographic correlation of 95 per cent. was obtained.

5. It is estimated that electroencephalographic localization of lesions of a less gross character was made in approximately 20 per cent. of the entire group, which correlated well with the clinical and pneumoencephalographic findings and which would not have been detected by other techniques.

6. The extra time and effort required by the technique are not excessive and appear to be well justified when used for purposes of localization.

(Authors' abstr.)

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Psychotic Casualties in New Guinea, with Special Reference to the Use of Convulsive Therapy in Forward Areas.

1. The problems of accommodation and treatment of psychotic casualties at an Australian general hospital in a forward area are presented.

2. The dangers attendant upon air transport and the methods successfully adopted to combat them are discussed.

3. A description and classification of the types of psychoses encountered in service personnel in New Guinea during the period from June, 1944, to May, 1945, inclusive, reveal that the same forms of illness occurred in service personnel as are met with in civil life. An interesting feature was the extreme degree of excitement and agitation manifested immediately prior to admission to hospital.

4. The role of " Atebrin " in the production of psychotic illnesses is discussed, and the type of illness attributed to such intoxication is described.

5. Certain impressions resulting from the observation of 333 cases have been given. A comparison is made of the results obtained before and after the introduction of convulsive therapy, and the value of this form of treatment in the management of psychotic patients who are violent and resistive and in the overcoming of the necessity for physical restraint is shown. (Author's abstr.)

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*Motor Chronaxia in Aged Patients with Generalized Arteriosclerosis. *Krayukhin, B. V., and Scherbakov, M. M.* 263

Motor Chronaxia in Aged Patients with Generalized Arteriosclerosis.

An analysis of the chronaxia and rheobase values obtained in generalized arteriosclerosis patients varying in age from 57 to 96 enables us to note peculiarities in the excitation of the neuro-muscular system in aged persons as compared with the chronaxia values in healthy people of middle age (men and women).

A comparison of the data obtained with the results of chronaxia and rheobase measurements in persons of the same age who had attained normal physiological senescence, shows that no essential differences exist between the values except those for the rheobase. The rheobase figures in aged persons who have attained physiological senescence is somewhat higher than the rheobase value in middle-aged persons; whereas in diseased persons of advanced age it is twice and sometimes three times as high as the normal.

The most pronounced cases of decreased chronaxia are noted in cases of generalized arteriosclerosis and in emphysema of the lungs, while cases of increased chronaxia are encountered in generalized arteriosclerosis attended by manifestations of cardiovascular deficiency. (Authors' abstr.)

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Pavlov's Doctrine of the Protective and Therapeutic Role of Inhibition in Instances of Trauma of the Nervous System.

It is generally considered that Pavlov thought of inhibition as playing a secondary, protective role in the regeneration of injured nerve centers, preventing further damage from pathogenic stimuli while other factors brought about the regeneration of the tissue. Asratian believes it is closer to Pavlov's doctrine to say that inhibition not only plays a protective role but is also an active, independent, therapeutic factor. Having thus stated Pavlov's point of view, he recounts a series of his own investigations whereby he developed techniques for accelerating recovery from injuries to the nervous system by the use of narcotics.

(Psychol. Abstr.)

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A Comparison of the Exercise Response in Anxiety States and Normal Controls.

1. Twenty normal controls, 10 anxiety states (with somatic anxiety symptoms but with no complaint of poor exercise response) and 10 effort syndrome patients are compared regarding their responses to moderate exercise on a bicycle ergometer.

2. O_2 uptake, lactate rise and pulse area were determined after exercise for each group.

3. The anxiety states as well as the effort syndromes had a significantly poor exercise response compared with the normal controls.

4. O_2 consumption after standard work is probably the most satisfactory quantitation of exercise response, but the three tests collectively give the most reliable index. (Authors' abstr.)

The Effect of Exercise on Soldiers with Neurocirculatory Asthenia.

1. A simple classification for different types of effort syndrome is outlined.

2. Twenty-five Group II ("constitutional") effort syndromes showed a mean blood lactate rise of 28·9 mgm. per cent. ± 6·14 s.d. after standard exercise on a bicycle ergometer. The corresponding figure for 35 normal controls was 21·1 mgm. per cent. ± 5·89 s.d., which is significantly different.

3. Ten Group III (psychogenically produced) effort syndromes did not show a significantly different lactate rise compared with the controls.

4. When worked to exhaustion point on a bicycle ergometer 20 normal controls showed a mean blood lactate rise of 78·0 mgm. per cent. ± 8·15 s.d. Twenty E.S. patients gave a mean blood lactate rise of 50·2 mgm. per cent. ± 5·74 s.d.

5. Pulse rise and decrement after standard work is highest in Group II E.S. and lowest in the normal controls, with Group III E.S. occupying an intermediate position. (Authors' abstr.)

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A Comparison of the Exercise Response in Various Groups of Neurotic Patients, and a Method of Rapid Determination of Oxygen in Expired Air, Using a Catharometer.

1. The exercise response to standard work on a bicycle ergometer of five different groups of neurotic patients (E.S., Group II; E.S., Group III; somatic anxiety states; anxiety states without demonstrable somatic anxiety, and patients with conversion hysteria) is compared with 20 normal controls, regarding oxygen uptake, lactate rise and pulse area.

2. All five groups are significantly worse than the controls when oxygen uptake is compared, but this differentiation is not so complete with the lactate rise and pulse area. The oxygen uptake figures indicate that poor exercise response is an attribute of neurotic patients in general.

3. A catharometer is a simple, quick and accurate method for oxygen estimation, and compared with Haldane's method of gas analysis on 22 gas samples gave a product moment correlation of 0·996. (Authors' abstr.)

JULY–AUGUST.

Studies on Headache. *Simons, D. J., and Wolff, H. G.* 227
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Changes in the Electroencephalogram and in the Excretion of 17-Ketosteroids Accompanying Electro-shock Therapy of Agitated Depression.

1. Thirteen women suffering from agitated depression have been studied before, during and after periods of 8 to 15 electro-shock treatments.

2. Daily ratings with the use of a new psychiatric scale are found to correlate well with fast (> 13 per second) frequencies in the electroencephalograms. When the patients improve, the percentage of time these waves are present decreases; when they relapse the fast waves tend to return. However, there is also a tendency for the fast waves to return in some patients showing good remission.

3. The diurnal rhythm of the nightly rate of 17-ketosteroid excretion divided into the morning rate as a measure of adrenal cortical activity was also measured. While this value for normals is 1·60, in the patients studied the mean pre-treatment value was 1·03; during the treatment period it rose to 1·32, and declined in the post-treatment period to 1·25.

4. The possible significance of these findings is discussed in terms of questions they raise for future exploration. (Authors' abstr.)

PUB. HEALTH REP., WASHINGTON.

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https://doi.org/10.1192/bjp.93.390.118 Published online by Cambridge University Press

1. Biochemistry, Physiology, Pathology, &c.

Particularly Intense Acetylcholine-like Activity of a New Series of Polyalcohol Amino Acetals. Fourneau, E., Bovet, D., Bovet, F., and Montezin, G. (Inst. Pasteur, Paris). [*Bull. soc. chim. biol.*, **26**, 516–27 (1944).]

All of the compounds mentioned below have a parasympathomimetic and muscarinic action in experimental animals. F 2249 and F 2301 also have a nicotinic action (produce hypertension in the atropinized dog); the others do not (2,3-dihydroxypropyl) trimethylammonium iodide formal (F 2249), $\overline{O.CH_2.O.CH_2}.CHCH_2NMe_3I$, is 0·5–2·0 times as active as acetylcholine (1). The acetal, $\overline{O.CHMe.O.CH_2}.CHCH_2NMe_3I$ (F 2268), is 10–50 times as active as (1) and is possibly the most potent poison of this type known. The propionaldehyde derivative (F 2292), $\overline{O.CHEt.O.CH_2}.CHCH_2NMe_3I$, and the acetone derivative (F 2269), $\overline{O.CMe_2O.CH_2}CHCH_2NMe_3I$, are 0·1 as active as (1), and the benzaldehyde derivative (F 2262), $\overline{O.CHPh.O.CH_2}CHCH_2NMe_3I$, is about 0·001 as active as (1). $\overline{O.CHMe.O.CH_2}CHCH_2.CH_2CH_2NMe_3I$ (F 2301), and the corresponding triethylammonium derivative (F 2302), have a very weak action, and so do $\overline{O.CHMe.O.CH_2}.CHCH_2NMe_3I$ (F 2276), and $\overline{O.CH_2.CH_2.O.CH}CHCH_2NMe_3I$ (F 2291).

L. E. Gilson (Chem. Abstr.).

Biochemistry of Choline and its Derivatives. XII. Determination of Total Choline and Water-insoluble Choline of Tissues. Kahane, Ernest, Levy, Jeanne, and Libert, Odette (Univ. Paris). [*Bull. soc. chim. biol.*, **27**, 65–72 (1945); *cf. C. A.*, **40**, 1548[2].]

A 1 gm. sample of blood, milk, urine, or fresh animal or plant tissue is mixed with 0·5 c.c. of concentrated H_2SO_4 in a porcelain crucible and left for 24 hours in an oven heated to 100–110°. At this temperature, in contact with air containing a little moisture, the H_2SO_4 becomes concentrated to approximately 65 per cent. by weight and remains at this concentration. Much organic matter is destroyed. Free and combined choline is converted to the salt of the half ester, $(CH_3)_3\overline{NCH_2CH_2SO_4}$. No decomposition of choline occurs under these conditions although conversion to $(CH_3)_3N$ may occur at higher temperatures. The contents of the crucible are washed into a flask with about 25 c.c. of water (the resulting mixture should contain approximately 4 per cent. by weight of H_2SO_4) and refluxed 8 hours to hydrolyze the ester. Then 1 c.c. of 50 per cent. $FeCl_3$ solution and an excess of $CaCO_3$ are added, the mixture is filtered, and choline is determined in the filtrate by known methods. It is sometimes necessary to treat the precipitate on the filter paper with dilute HCl to dissolve the $Fe(OH)_3$ and excess $CaCO_3$ and treat the insoluble residue of organic matter (containing $CaSO_4$) with H_2SO_4, etc., as before, to render soluble some choline not made soluble during the first treatment.

L. E. Gilson (Chem. Abstr.).

Changes in the Contents of Acetylcholine in Nervous Tissue Under the Influence of Constant Current. Babskii, Eug. B. (State Pedagogical Inst., Moscow). [*Nature*, **157**, 730 (1946).]

Non-polarizable electrodes were applied to dog brain and a d.c. of 4–10 ma. was used for 10 minutes, after which pieces of the brain were removed and analyzed. Nerve fibers of dog and cat were similarly treated. In all cases the tissue immediately adjacent to the cathode showed an increase in acetylcholine and that next to the anode a decrease. L. E. Gilson (Chem. Abstr.).

Mechanism of Apnea Provoked by Acetylcholine. Martini, V., and Calcagno, L. (Univ. Geneva). [*Boll. soc. ital. biol. sper.*, **15**, 587–8 (1940).]

The production of apnea in experimental animals by acetylcholine is not due to action through the central nervous system, but appears to be a direct effect on the lungs, probably on the bronchial musculature. L. E. Gilson (Chem. Abstr.).

https://doi.org/10.1192/bjp.93.390.118 Published online by Cambridge University Press

Action of Acetylcholine and of Vitamin B_1 by the Suboccipital Route on Blood Sugar and Calcium. Gomez, F. Idoipe, and Ortiz, A. Valcarreres. [*Farmacoterap. actual* (*Madrid*), **2**, 169–76 (1945).]

Acetylcolhine, 100 mgm. per kgm., injected into the cisterna magna of dogs produces a large rise of blood sugar. This does not occur after previous adrenalectomy. Blood Ca falls probably owing to the resulting overbreathing. Vitamin B_1 (25 mgm.) has a similar action on the blood sugar but does not alter the Ca level.

B. A. (Chem. Abstr.).

Experiments on the Mechanism of Action of Acetylcholine. Effects of Potency and Concentration, and their Recognition in the Isolated Intestine of the Rabbit. Beccari, E. [*Arch. sci. biol.* (*Italy*), **29**, 191–224 (1943).]

The diastolic tonus of decontraction of the tonus in the isolated surviving intestine of the rabbit indicates a duality in the mode of action of acetylcholine upon that organ. The pharmacological action is related to the quantity of the drug fixed upon the effectors which may be termed the " concentration effect," and on the speed of this fixation, which may be called the " potential effect." Experiments reveal that the " potential " effect does not follow the strictly physicochemical laws which apply to non-living membranes.

D. I. MACHT (Chem. Abstr.).

Studies on Choline Acetylase. II. Formation of Acetylcholine in the Nerve Axon. Nachmansohn, David, John, Hedda M., and Berman, M. (*Columbia Univ.*). [*J. Biol. Chem.*, **163**, 475–80 (1946); *cf. C. A.*, **39**, 2519[6].]

A relatively high concentration of choline-acetylating enzyme system (I) was observed in the peripheral axon (II) of rabbit, which is free of cell bodies and nerve endings. The presence of this (I) in (II) supports the assumption that acetylcholine (III) is not only essential for the transmission of the nerve impulse across the synapse but also for its propagation along the axon. In degenerating nerve fiber the enzyme activity decreases slowly, and 72 hours after section, when the condition disappeared, the enzyme activity decreased two-thirds. Six days after section no enzyme activity remained. These results support the assumption that the formation of (III) is associated with condition. L. WEIL (Chem. Abstr.).

Microscopic Lesions in Acetylcholine Shock. Altschul, R., and Laskin, M. M, (*Univ. Saskatchewan, Saskatoon, Saskatchewan, Canada*). [*Arch. Path.*, **41**, 11–16 (1946).]

Sublethal and lethal doses of acetylcholine caused vascular reactions in rats and guinea-pigs comparable to other types of shock. About half the animals showed pulmonary edema. M. L. C. BERNHEIM (Chem. Abstr.).

Stimulation of Phosphorylation in Muscle by Acetylcholine. Wajzer, J. [*Compt. rend. soc. biol.*, **139**, 375–6 (1945).]

In frog muscle poisoned with iodoacetate, acetylcholine 1 : 10,000, accelerates the transfer of H_3PO_4 from creatine phosphate to hexose with formation of hexose diphosphate. The adenosine triphosphate remains unchanged. In this respect acetylcholine acts like ionic K. L. E. GILSON (Chem. Abstr.).

Chemical and Electrical Origin of Nervous Energy. Barnes, T. Cunliffe. (*Hahnemann Med. Coll., Philadelphia*). [*Hahnemannian Monthly*, **81**, 56–60 (1946).]

A discussion of the acetylcholine theory of the action current in nerve.

JOSEPH S. HEPBURN (Chem. Abstr.).

Vagus Substances. v. Muralt, Alexander (*Bern*). [*Experientia*, **1**, 136–46 (1945) (*in German*).]

A lecture discussing the liberation of acetylcholine and thiamine on nerve stimulation and the physiological interrelationships between the two substances.

J. M. LITTLE (Chem. Abstr.).

https://doi.org/10.1192/bjp.93.390.118 Published online by Cambridge University Press

Acetylcholine Synthesis. *Torda, Clara, and Wolff, Harold G.* (*Cornell Univ. Med. Coll., Ithaca, N.Y.*). [*Science*, **103**, 645–6 (1946); *cf. C. A.*, **39**, 550[3], 983[3], 1208[7], 1927[3], 4101[5]; **40**, 2211[7].]

Chiefly review with 33 references.

It is postulated that normally occurring constituents of cells and extracellular fluid modify the amount of acetylcholine synthesized in the living organism. There is a dynamic equilibrium between potentiator substances and inhibitor substances. During physiological activity the original dynamic equilibrium is disturbed and new dynamic equilibrium is established. Certain metabolites of muscle released during prolonged work decrease the synthesis of acetylcholine. The accumulation of such metabolites is important in the production of fatigue resulting from indirect stimulation and secondary to decreased acetylcholine synthesis.

E. D. WALTER (Chem. Abstr.).

Effect of Acetylcholine and Adenosine Triphosphate on Denervated Muscle. *Buchtha, Fritz, and Kahlson, Georg* (*Univ., Lund, Sweden*). [*Acta Physiol. Scand.*, **11**, 284–8 (1946).]

Na adenosine triphosphate initiates contractions in denervated anterior tibial muscles of the cat which last longer than in normal muscles. But whereas normal muscle is not affected by a previous injection of acetylcholine, this abolishes the sensitivity of denervated muscle to the ATP. Denervated muscle is 40–50 times as sensitive as normal muscle to acetylcholine whose curare-like action also is very pronounced in denervated muscle. It is argued that the motor end plate survives even a high degree of muscle atrophy, such as exists after 75–85 days of nerve degeneration because in this condition the muscle still shows increased sensitivity to acetylcholine which can be abolished by curarization.

S. MORGULIS (Chem. Abstr.).

Properties and Distribution of the Enzyme System which Synthesizes Acetylcholine in Nervous Tissue. *Feldberg, W., and Mann, T.* (*Cambridge Univ., Eng.*). [*J. Physiol.*, **104**, 411–25 (1946); *cf. C. A.*, **29**, 4667[8]; **40**, 1923[8].]

The enzyme was obtained by extraction of acetone-dried tissue with 0·9 per cent. NaCl. Its activity was greatly increased by adenosine triphosphate (ATP), citric acid, and a third substance referred to as the activator and which was found in the juice prepared by extraction of the brain tissue with boiling saline solution. The activator was dialyzable, was not mineral nor identical with choline, ATP, glutathione, cozymase, or thiamine. It was present in liver in approximately the same concentration as in brain, and small amounts were in muscle and yeast. The activating effect of citric acid was not due to its Ca-binding capacity, and the effect was not abolished by malonic acid. None of dl-glutamic, l(+)-glutamic, aconitic, succinic, fumaric, acetic, oxalic, tartaric, pyro-phosphoric, malonic, pyruvic, or oxaloacetic acids showed an accelerating effect comparable to that with citric acid. If ATP, citrate, and the activator were added together to the saline extract from acetone-dried brain, as much as 1·8 mgm. acetylcholine/gm. powder/hour at 37° was formed, or 6 mgm./gm. dry weight of extract. The dialyzed extract was inactive and was not reactivated by any one of the three activators alone, but was by citrate and activator, and in particular by citrate, activator, and ATP. The concentration of enzyme in the nervous tissue varied widely between species, and between parts of the nervous system of a given species. The activity was greater in cerebrum than cerebellum, greater in efferent cholinergic fibers than in afferent fibers. For a given rate of synthesis the corresponding content of acetylcholine was higher in the peripheral than in the central nervous system.

H. L. WILLIAMS (Chem. Abstr.).

A New Method of Estimation of Acetylcholine with the Isolated Duodenum of the Rat. *Bayo Bayo, J., García de Jalón, M., and García de Jalón, P.* (*Lab. Farmacol. exptl. facolta med., Madrid*). [*Farmacoterap. actual* (*Madrid*), **2**, 622–8 (1945).]

The duodenum of the rat gives very regular responses to acetylcholine (I) but the voluntary movements of the muscle are of such amplitude that the interpretation

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is difficult. It was found that the addition of 0·15 per cent. $MgSO_4$ to Ringer solution causes the gut to relax but does not prevent a prompt response to (I). In tyrode solution the relaxation and reduction of amplitude are still better, but the Mg in presence of the Ca of the solution also depresses the response to (I). The response can be re-established by simultaneous sensitization with pantocaine and eserine. The most favorable conditions are obtained with a tyrode solution containing 0·01 per cent. $CaCl_2$ and 0·1 per cent. $MgSO_4$. After submerging the strip of duodenum in the bath of 80 c.c. at 37–8° 0·2 mgm. of pantocaine-HCl and 0·01 mgm. of eserine salicylate are added. The test is performed after 5 minutes. Equal doses of (I) between 0·1 and 1γ give identical contractions. After each test the gut is washed with 300 c.c. of the bath fluid. A. E. MEYER (Chem. Abstr.).

Acetylcholine and Choline Esterase Contents of Small Intestine. Cheng, C. P., Hsin, H. Y., and Hsu, F. H. [*Proc. Chinese Physiol. Soc.*, **1**, 27–30 (1942).]

In rabbits and dogs, choline esterase activity is higher in the muscle than in the mucosa, and in the lower parts of the small intestine than in the duodenum. Acetylcholine can be extracted both from muscle and from the mucosa, and in greater quantity from the upper part of the intestine than from the lower segments. In a rabbit, vagotomized, atropinized, and eserinized, 25·4 per cent. of acetylcholine were synthesized in 10 minutes by the jejunum. These findings are probably related to the higher motor sensitivity of the upper gut to acetylcholine in comparison with the lower gut. B. A. (Chem. Abstr.).

Cholinesterase in Cerebrospinal Fluid. Glasson, B., and Mutrux, S. (*Univ. Geneve*). [*Helv. Physiol. Pharmacol. Acta*, **4**, C12–14 (1946) (*in French*).]

The manometric method of Ammon (liberation of CO_2 from $NaHCO_3$ by the freed acid) was used with two substrates, acetylcholine (I) and monobutyrin (II). Human blood serum was quite active on both (I) and (II) and in both cases the action was almost completely inhibited by eserine (III). Red corpuscles and normal cerebrospinal fluid were strongly active on (I) and this action was inhibited by (III). Corpuscles showed a slight, and cerebrospinal fluid a variable activity towards (II) and this action was unaffected by (III). Cerebrospinal fluid from three cases of oligophrenia was strongly active on (I) and only slightly active on (II).
L. E. GILSON (Chem. Abstr.).

Influence of the Anticholinesterase, Diisopropylfluorophosphate, on Cardiovascular and Respiratory Reflexes of Carotid Sinus Origin. Heymans, C., and Pannier, R. (*Univ. of Ghent, Belgium*). [*Proc. Soc. Exptl. Biol. Med.*, **26**, 228 (1946).]

In dogs under chloralose anesthesia, intravenous injections of diisopropylfluorophosphate (0·12–1·0 mgm./kgm.) inhibiting completely the cholinesterase activity of the blood do not stimulate the respiratory center and do not affect either the heart rate or the blood pressure. The cardiovascular and respiratory reflexes induced by increase or decrease of the intracarotid sinus pressure are unaffected. These results do not support the theory of a cholinergic transmission of the respiratory and cardiovascular reflexes induced by a physiological stimulation of the carotid sinus pressoceptors. L. E. GILSON (Chem. Abstr.).

Studies on Cholinesterase. II. Enzyme Activity and Voltage of the Action Potential in Electric Tissue. Nachmansohn, David, Coates, C. W., and Rothenberg Mortimer A. (*Columbia Univ.*). [*J. Biol. Chem.*, **163**, 39–48 (1946); *cf. C. A.*, **39**, 3798[1].]

The activity of cholinesterase and the voltage of the action potential were measured simultaneously in the electric organ of a number of specimens of *Electrophorus electricus* confirming and extending previous findings indicating a close parallelism between the two. The direct connection of acetylcholine with the nerve action potential and nerve condition is highly probable.
DAVID B. SABINE (Chem. Abstr.).

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Cholinesterases in Degenerating and Regenerating Peripheral Nerves. Sawyer, Charles H. (Duke Univ., Durham, North Carolina). [*Am. J. Physiol.*, **146**, 246–53 (1946).]

The guinea-pig sciatic nerve contains both true cholinesterase (I) and pseudocholinesterase (II). Extracts of unoperated nerve hydrolyze mecholyl nearly 60 per cent. as readily as (I) and benzoylcholine only 20 per cent. as rapidly as (I). Wallerian degeneration of the tibial and peroneal nerves is attended, within the first few days after section of the sciatic nerve, by a 60 per cent. loss of their true (I) content. Further loss of the enzyme fails to occur even with the complete disintegration of axis cylinders. (II) activity is unaffected by the operation while (I) hydrolyzing capacity, dependent on the two enzymes, drops 30 per cent. Nearly two-thirds of the true (I) is secreted by the axis cylinders, but more than one-third is produced by some other element or elements, perhaps sheath cells. (II) is probably completely resident in the connective tissue.

E. D. WALTER (Chem. Abstr.).

Choline Esterase and its Specificity. Augustinsson, K. B. (Chem. Inst. of Vet. Coll., Stockholm). [*Nature*, **157**, 587–8 (1946); *cf. C. A.*, **39**, 5258[3].]

A specific cholinesterase is an enzyme which hydrolyzes acetylcholine at a higher rate than any other esters. The whole animal of *Patella vulgata* and the ampulla and podia of *Asterias rubens* from the west coast of Sweden split acetyl-β-methylcholine at a higher rate than acetylcholine. Benzoylcholine is very little split. In contrast, *Terebratulina caput serpentis* hydrolyzes acetylcholine but neither acetyl-β-methylcholine nor benzoylcholine. The reactions used to distinguish between a true and a pseudocholinesterase with the help of two choline esters do not tell the complete story about the types of this enzyme.

E. D. WALTER (Chem. Abstr.).

Specificity of Cholinesterase. Cholinesterase Activity and Phosphatase Activity. Cristol, P., Passouant, P., Benezech, C., and Dutarte, Jeanne (Univ. Montpellier). [*Compt. rend. soc. biol.*, **139**, 312–13 (1945).]

No correlation was found between the variations in cholinesterase activity and phosphatase activity in normal and pathological human serums.

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

Inhibition of Cholinesterase and Activation of Phosphatase in Blood Serum. [*Ibid.*, 314–15.]

Eserine sulfate has no effect on serum phosphatase activity but largely inhibits the cholinesterase activity. $MgSO_4$ increases the phosphatase activity but not the cholinesterase activity. L. E. GILSON (Chem. Abstr.).

Absence of Pseudocholinesterase from the Tissues of Ruminants. Gunter, Josephine M. (Univ. of Toronto, Can.). [*Nature*, **157**, 369 (1946).]

Tissues of ox and sheep which, by their inability to hydrolyze benzoylcholine, were shown to contain no pseudocholinesterase are liver, spleen, pancreas, muscle, gastric mucosa, intestinal mucosa, thyroid gland, and lachrymal gland. This is a further indication of the irrelevance of pseudocholinesterase to the process of nerve impulse transmission. E. D. WALTER (Chem. Abstr.).

Determination of Histamine in Cerebrospinal Fluid and Blood in Some Cases of Cerebral Disease. Businco, Lino, and Visalli, Felice (Univ. Rome). [*Boll. soc. ital. biol. sper.*, **16**, 258–9 (1941).]

In 5 patients with brain tumors the blood contained 0·04–0·10γ of histamine per c.c. and the cerebrospinal fluid not more than 0·02γ/c.c.

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

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Free Aneurine Content of Cerebrospinal Fluid. Cacioppo, Filippo (*Univ., Palermo, Italy*). [*Boll. soc. ital. biol. sper.*, **20**, 83–4 (1945).]

In a study of 29 children, the cerebrospinal fluid showed no appreciable variations in aneurine content in cases of diphtheria or poliomyelitis. The aneurine titer was greatly increased in cases of tubercular meningitis.

HELEN LEE GRUEHL (Chem. Abstr.).

Effect of Morphine on Cerebrospinal Fluid Pressure. Chou, C. [*Proc. Chinese Physiol. Soc.*, **1**, 97–101 (1942).]

In dogs under urethan, paraldehyde, or phenobarbital, morphine (0·25–1 mgm. per kgm.) decreased cerebrospinal fluid pressure simultaneously with blood pressure in 9 out of 16 animals. In the remaining 7 animals, cerebrospinal fluid pressure increased, while the blood pressure remained unaltered or decreased. Changes of cerebrospinal fluid pressure are the result of the balance of the action of the drug on local cerebral circulation and on systemic blood pressure.

B. A. (Chem. Abstr.).

The Specific Gravity of Spinal Fluid. A Review of Methods and the Application of a Newer Micromethod in Spinal Anesthesia. Wolman, Irving J., Evans, Barnett, and Lasker, Sigmund (*U.S. Public Health Service Hospital, Sheepshead Bay, Brooklyn*). [*Am. J. Clin. Path., Tech. Sect.*, **10**, 33–9 (1946).]

The following method was simple and accurate. Fill the lower part of a 2,000 c.c. graduated cylinder with paraffin so that the mouth of a 250 c.c. graduated cylinder centered in it will protrude about 1 inch. Fill the space between the cylinders with water for insulation, adding a few drops of H_2SO_4 and a few crystals of $CuSO_4$ as preservative. Cover with paraffin. Prepare the gradient mixture by blending 31 c.c. of bromobenzene (specific gravity 1·94) with 94 c.c. of tech. kerosene (sp. gr. 0·81) producing a light mixture (sp. gr. 0·97). To produce a heavy mixture (sp. gr. about 1·05), blend 47·5 c.c. bromobenzene with 77·5 c.c. of kerosene. For a gradient between sp. gr. 1·000 and 1·0200 with extremes from 0·97 to about 1·05, pour the heavier mixture into the inner cylinder to the 125 c.c. mark, and overlay it with the lighter mixture to the 250 c.c. mark. To produce the gradient insert a piece of 16 gauge Cu wire coiled to form a disc with a long handle to the level of the interface two or three times with a 1 inch stroke into each phase. Increase the stroke by 1 inch and repeat. Continue this until only 1 inch of the top and bottom is undisturbed. Calibrate with NaCl solutions of known sp. gr., checking each day of use for changes due to diffusion, temperature, etc. Add drops of the spinal fluid to be tested, of the same size used in calibration and read the sp. gr. from the graduations on the cylinder. The sp. gr. of spinal fluid from persons free from disease of the central nervous system was found to be 1·0062–1·0082, with a mean of 1.00697, at 25 degrees. A few specimens from subjects with syphilis of the central nervous system were elevated above this range.

JOHN T. MYERS (Chem. Abstr.).

Action of Aneurine and Riboflavin on Isolated Intestine and their Functional Relationships to Acetylcholine and Adrenaline. Sanz Sanchez, F. [*Farmacoterap. actual* (*Madrid*), **1**, No. 3, 43–51 (1944).]

Aneurine inhibits the isolated intestine of rabbits and increases the action of adrenaline. Acetylcholine is less effective after administration of aneurine. Lactoflavin inhibits the intestine and does not modify the action of adrenaline or acetylcholine.

B. A. (Chem. Abstr.).

Mental Response to Added Thiamine. Harrell, Ruth F. (*Columbia Univ.*). [*J. Nutrition*, **31**, 283–98 (1946).]

During three periods of time the effect was observed of daily supplementation of the dietary of an orphanage with 2 mgm. of thiamine per child. Thirty-seven to 55 carefully matched pairs of children were used, one member of each pair receiving thiamine and the other a placebo. Measurements of performance included acuity of vision, skills at games, reaction time, reading, arithmetical processes,

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memorizing and forgetting, intelligence tests, and other recognized measures in current use by psychologists. In the first period of 6 weeks the vitamin-fed group made superior average gains in performance in every one of the 18 test tasks. The individual variations in gains within each group were within the limits of probable error in the case of 7 of these tasks. In the second period of 1 year, 15 activities were used and the vitamin-fed group again surpassed the control group in gains in performance in every activity. The superior gain for the vitamin-fed group was statistically significant for 8 of the 15 activities. In the third period of 1 year, without knowledge of the participants, 20 pairs of children were continued with the same regime as in the previous year while 20 pairs were reversed, so that those who had received the thiamine now received the placebo and *vice versa*. The unreversed pairs continued to show superiority of average performance for the thiamine-fed group in all 11 tests used; the reversed pairs showed reversals of superior gain in 7 activities but failed to show adverse effects of withdrawal of thiamine in intelligence tests, educational achievement, reaction time, height or weight gains. The children received from 0·9 to 1·0 mg. thiamine daily in their normal diet. W. B. Esselen, Jun. (Chem. Abstr.).

Disturbances of Neuromuscular Excitability During Dietary Imbalance and Avitaminoses. VI. Antialkalosic Action of Ascorbic Acid and Vitamin D (Calciferol, Lecoq, Raoul, Chauchard, Paul, and Mazoue, Henriette (La Sorbonne, Paris). [*Bull. soc. chim. biol.*, **27**, 358–63 (1945); *cf. C. A.*, **40**, 1911[9].]

For action of ascorbic acid, *see C. A.*, **40**, 3513[9]. Calciferol acts in a similar fashion. L. E. Gilson (Chem. Abstr.).

VII. Alkalosis in Experimental Rickets. [*Ibid.*, 364–76.]

In rickets the bone lesions and the neuromuscular troubles due to alkalosis develop independently. Administration of ascorbic acid prevents only the latter, vitamin D prevents or cures both. L. E. Gilson (Chem. Abstr.).

Vitamins Antagonizing the Action of Thyroxine on Nerve Chronaxia. Chauchard, Paul, and Mazoue, Henriette (École hautes études, Paris). [*Compt. rend. soc. biol.*, **139**, 594–5 (1945).]

The shortening of nerve chronaxia in pigeons produced by thyroxine is antagonized by ascorbic acid, thiamine, riboflavin, nicotinamide, or pantothenic acid, but not by vitamins A or D, or glutathione, cysteine, or methionine.
L. E. Gilson (Chem. Abstr.).

Biochemical Study of Avitaminoses of the Pellagra Group. IX. Influence of Riboflavin on the Nicotinamide Content of Livers of Rats on Certain Synthetic Diets. Raoul, Y. (Hôpital Foch, Paris). [*Bull. soc. chim. biol.*, **27**, 371–3 (1945); *cf. C. A.*, **40**, 3510[2].]

The distribution of nicotinamide (plus nicotinic acid) in different parts of the rat liver is very irregular; hence an accurate estimate cannot be made from analysis of a small sample from one lobe. The entire liver should be sampled. A deficiency of riboflavin in the diet frequently causes an important decrease in liver nicotinamide. This should be of interest in connection with human pellagra.
L. E. Gilson (Chem. Abstr.).

Biochemical Study of Avitaminoses of the Pellagra Group. X. Is Cutaneous Excretion of Porphyrin by the Rat a Sign of Pantothenic Acid Deficiency? Raoul, Y., and Marnay, C. (Hôpital Foch, Paris). [*Bull. soc. chim. biol.*, **27**, 502–8 (1945); *cf. C. A.*, **40**, 3510[2].]

The absence of either pantothenic acid or pyridoxine, especially the latter, in the diet results in cutaneous excretion of protoporphyrin by the rat. Choline may have a slight protective action. L. E. Gilson (Chem. Abstr.).

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XI. Changes in the Porphyrins of Biological Media under the Influence of Dietary Imbalance. [*Ibid.*, 509–13.]

Rats on diets deficient in pantothenic acid or pyridoxine, or both, showed cutaneous excretion of protoporphyrin without other porphyrins and at the same time an increase in urinary excretion of coproporphyrin. Their livers contained free protoporphyrin, while livers from normal rats contained none. Feeding rats a Goldberger maize diet did not cause cutaneous excretion of protoporphyrin and did not increase the urinary excretion of coproporphyrin (normal is 4·8–5·8γ per cent.). L. E. GILSON (Chem. Abstr.).

Structure of the Liver in Pellagra. Gillman, J., and Gillman, T. (*Univ. Witwatersrand, Johannesburg, S.A.*). [*Arch. Path.*, **40**, 239–63 (1945).]

By means of an improved biopsy technique, the changes in the liver in pellagra have been studied. Fatty livers are found in children, and fat and pigment (cytosiderin and cytolipochrome) are found together or separately in the livers of adults. Pigmentary cirrhosis is common in adults, and is indistinguishable from hemochromatosis. These changes represent the reaction of the liver to acute or chronic malnutrition. M. L. C. BERNHEIM (Chem. Abstr.).

Ultraviolet Absorption of Living Nerve Fibers. Lüthy, H. (*Hallerianum, Bern*). [*Helv. Physiol. Pharmacol. Acta*, **4**, C20–2 (1946) (*in German*).]

The apparatus and method are described. The ultraviolet absorption curve of a frog nerve is shown for the range 250–320 mμ. The curve shows a small peak at 290 mμ due to tyrosine, a high peak at 265–70 mμ due to unknown constituents, a dip at 258 mμ, and complete absorption at 250 mμ.

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

Indicanorachia under Physiological and Pathological Conditions. Tinelli, Giuseppe. [*Ibid.*, 172–3.]

Indican was present in normal human cerebrospinal fluid in amounts of 23.7γ/100 gm. Under various pathological conditions (meningitis, luetic nervous conditions, etc.) the indican increased to an average of 67.5γ/100 gm.

The Effect of Removal of the Suprarenal Capsule on the Neuromuscular System and the Effect of the Injection of Desoxycorticosterone Acetate. Chauchard, Paul (*École hautes études, Sorbonne, Paris*). [*Ann. d'endocrinol.*, **4**, 257–8 (1943); *Chem. Zentr.*, **11**, 227 (1944).]

Chronaximetric measurements on young rats from which the suprarenal capsule had been removed showed that the asthenia observed in such animals is of encephalic origin and that the musculature does not experience a depressor but rather an excitor action. The principal effect of desoxycorticosterone acetate on normal animals is always a muscular excitation and, depending on the dose, an excitation or depression of the nervous centers. In the case of animals from which the suprarenal capsule had been removed or in scorbutic animals the above hormone showed a corrective effect on the chronaxia phenomena.

M. G. MOORE (Chem. Abstr.).

The Diencephalic-hypophysial Centers and Uric Acid Exchange. Ferrannini, Alfredo (*Univ. Bari, Italy*). [*Boll. soc. ital. biol. sper.*, **15**, 782–4 (1940).]

Blood uric acid curves after intravenous injection of uric acid (50 c.c. of Koehler's solution) were determined for patients with lesions of the diencephalic-hypophysial region and for normal subjects. The results were irregular and inconclusive.

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

Uric Acid Exchange in Chronic Encephalitis. [*Ibid.*, 785–6.]

Patients with lesions in the diencephalon resulting from encephalitis were given uric acid intravenously (50 c.c. of Koehler's solution) and the rate of disappearance of the uric acid from the blood determined. The rate seemed to be a little slower than in normal subjects. L. E. GILSON (Chem. Abstr.).

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Influence of Magnesium on Chronaxia of Nerve and Muscle. Bartorelli, Cesare (Univ., Bologna). [*Boll. soc. ital. biol. sper.*, **15**, 621–2 (1940).]

Frog nerve-muscle preparations were used. Addition of $MgCl_2$ to the bath solution prolonged nerve chronaxia and shortened muscle chronaxia, but increased the rheobase of both nerve and muscle. The effects were reversible.

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

Effect of Anoxia on Nervous Excitability in Man. Beyne, J., Chauchard, B., and Chauchard, P. (Sorbonne). [*Compt. rend. soc. biol.*, **139**, 929–30 (1945).]

Partial anoxia without hypercapnia prolongs the chronaxia of the motor nerves.

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

Effects of Aminothiazole and Thyroxine on Chronaxia. Chauchard, Paul (Sorbonne). [*Compt. rend. soc. biol.*, **139**, 925–6 (1945).]

In pigeons the injection of 50 mgm. of aminothiazole daily for 2 days causes a decrease in basal metabolism and a lengthening of nerve and muscle chronaxia. The oral administration of thyroxine to normal pigeons or pigeons treated with aminothiazole causes an increase in basal metabolism, and a decrease in chronaxia to considerably below normal. L. E. GILSON (Chem. Abstr.).

Oxygen Poisoning. III. The Effect of High Oxygen Pressures upon the Metabolism of Brain. Stadie, William C., Riggs, Benjamin C., and Haugaard, Niels (Univ. of Pennsylvania, Philadelphia). [*J. Biol. Chem.*, **160**, 191–208 (1945); *cf. C. A.*, **38**, 1560[2].]

The metabolic activity *in vitro* of tissues surviving exposure to high O pressures in comparison with similar tissues under normal O tension was studied in an effort to determine the mechanism of O poisoning. Toxic effects developed rapidly in rats exposed to 8 atmosphere of O culminating in convulsions, and practically all animals exposed for 30 minutes died, either during exposure or within a few hours after decompression. Oxygen poisoning, however, was completely reversible up to a certain point, because if animals were decompressed relatively early, many recovered even though severe convulsions had occurred. Hemorrhagic patches in the lungs and intestines were observed at autopsy. Gross examination of the brain, on the other hand, revealed no changes. Thin slices of tissue from animals killed by high O pressures showed no significant changes in total O intake or respiratory quotient when examined in the Warburg apparatus under normal O tension. Similar preparations from animals exposed during a preliminary period to high O pressures and later observed in the Warburg apparatus under normal O tension manifested a steadily decreasing rate of O intake proportional to the O pressure employed and the time of exposure. Inhibition of brain metabolism by high O pressure was similar with glucose, fructose, pyruvate, or lactate as substrate. Similar findings were obtained when normal tissue was observed at 8 atmosphere of O, a 50 per cent. reduction in the initial O uptake being detected within 1 hour. The fact that the time required for appreciable reduction in the rate of O uptake was far greater than for the production of serious symptoms, convulsions and death when intact animals are subjected to the same O pressures appears significant in connection with the mechanism of O poisoning. Brain slices subjected to relatively high pressures of CO_2 (up to 107 mm. of Hg) in addition to 8 atmosphere of O were no more susceptible to O poisoning than those exposed to 8 atmosphere of O alone. Furthermore, the metabolism of brain tissue *in vitro*, although diminished in rate, was not altered from its predominantly high carbohydrate character by high O pressures. Attempts to reverse the lowered O uptake of tissue poisoned by O with early return to normal O tension failed. The rate of O uptake by tissues *in vitro* in a normal atmosphere was not affected by 7 additional atmospheres of N or H. The data obtained are discussed in relation to the mechanism of acute O poisoning. D. W. MCKINSTRY (Chem. Abstr.).

Suction Type of Electrode for Electroencephalography. Rice, H. V. [*Can. J. Research*, **23E**, 19–21 (1945).]

The electrode is constructed from Pb-coated brass tubing with an upper rubber diaphragm to facilitate application by suction. B. A. (Chem. Abstr.).

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Photosensitivity as a Cause of Falsely Positive Cephalincholesterol Flocculation Tests. Moses, C. [*J. Lab. Clin. Med.*, **30**, 267–9 (1945).]

The photosensitivity of the cephalin-cholesterol flocculation reaction is confirmed. Since this reaction occurs in artificial light as well as in sunlight, it is not due to any property peculiar to the latter. It is not due to heat generated in the sample by light. B. A. (Chem. Abstr.).

Consumption of Oxygen and Muscular Work During the Crisis of Electric Shock. Delmas-Marsalet, P., Servantie, L., Faure, J., and Pouyanne, H. (*Hôpital Saint-André, Bordeaux*). [*Compt. send. roc. biol.*, **139**, 845–6 (1945).]

During the first minute of convulsions produced by electric shock treatment O consumption often increases to 6–8 times the basal rate. At the same time blood lactic acid increases to 2–3 times normal. These effects are due to the intense muscular activity. A 52-year old man was studied.

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

Biochemical and Physiological Differentiation During Morphogenesis. VI. Succinic Dehydrogenase and Succinic Oxidase in the Cerebral Cortex of the Fetal Pig, Flexner, Louis B., and Flexner, Josefa B. (*Carnegie Institution of Washington, Baltimore, Md.*). [*J. Cellular Comp. Physiol.*, **27**, 35–42 (1946); *cf. C. A.*, **36**, 1075[1].]

In early fetal life succinic dehydrogenase activity of the cerebral cortex is approximately 35 per cent. of the adult value. Between 68 and 75 days the activity begins to increase and reaches the adult level at birth. Succinic oxidase activity is low or absent up to about 66 days. It then increases to birth when it is equal to that of the adult. The low activity in early stages of gestation is ascribed to the low cytochrome c activity. H. L. MASON (Chem. Abstr.).

Contributions to the Study of the General Physiology of Nerve Centers. I. Central Summation. Bremer, F., Bonnet, V., and Moldaver, J. (*Lab. Path. Generale of Univ. of Brussels*). [*Arch. Intern. Physiol.*, **52**, 1–56 (1942).]

The latent central addition of two impulses of afferent influx has been studied in the frog and toad (spinal animals, decerebrated or intact) for homolateral and contralateral reflexes of the posterior limb. The non-barbiturate narcotics, as well as convulsive agents in doses affecting an isolated stimulus, do not change the shape of the summation curve; they only modify the heights of the ordinate; barbiturates (particularly veronal) depress the second summit of the curve. Physiological aspects of the subject are reported. D. A. MEYER (Chem. Abstr.).

II. Inhibition Reflex. Bremer, F., and Bonnet, V. [*Ibid.*, 153–94.]

The inhibition reflex is provoked in the spinal frog by a single induction shock to a sensitive nerve. While the reflex of the flexion of a posterior member is disturbed by the latent addition of two rupture shocks applied on the afferent sciatic and while the summation curve of this reflex is of a simple type, the precision of an identical or feebler induction shock (stimulus S_1), exciting the same sensitive nerve, has the regular effect of inhibition of response to the stimuli $S_2 + S_3$. This inhibition can proceed to the complete suppression of the reflex. Strychnine and catelectrotonus intensify the excito-motor effect of $S_2 + S_3$ and the inhibiting effect of S_1, depressor agents (central fatigue, narcotics, anelectrotonus) weaken one another. D. A. MEYER (Chem. Abstr.).

III. The After-discharge Reflex and the Neuro-chemical Theory of Central Activation. Bremer, F., Bonnet, V., and Moldaver, J. [*Ibid.*, 215–48.]

Data indicate that the primary discharge reflex and the after-discharge are the central determinants, distinct and relatively independent: (*a*) they have different laws of summation; (*b*) they have different sensitivities to various physical and pharmacological agents, in the sense of a reinforcement or weakening more or less elective of the after-discharge by certain agents, and reciprocally of a much more marked action of other agents on the primary discharge; the after-discharge is

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notably strengthened electively by catelectrotonus, K, nicotine, acetylcholine, choline, eserine, and prostigmine, while the action of strychnine applies especially to the primary discharge; it is electively depressed by anelectrotonus and by the barbiturates, especially by veronal. D. A. MEYER (Chem. Abstr.).

Pentosenucleoproteins and Neural Induction. Brachet, J. [*Bull. classe sci., Acad. roy. Belg.*, **29**, 707–18 (1943) (*Pub.*, 1945).]

Nucleoproteins, nucleic acids and certain of their derivatives have induction capacities for the nervous system of amphibians. During invagination of the batrachian embryo, the amount of ribonucleic acid increases in the embryonic nervous system, while it diminishes elsewhere. Zymonucleic acid, in yeast, has its origin in the active derivatives of glucidic metabolism, notably 5-adenylic acid. The hydrocarbon metabolism of the gastrula is most intense in the region where the amount of ribonucleic acid in the cells varies the most; this denotes growth and organization. The synthesis of proteins has been correlated with the presence of large amounts of pentosenucleic acid, although the manner in which it acts is obscure. The splitting of proteins plays a major role in muscular contraction and presumably also in cell division. Recent researches have shown the existence of elongated molecules in the neural plaque of the hen, and the particular form of these cells of the medullary plaque is in harmony with the existence of this type of molecule. D. A. MEYER (Chem. Abstr.).

Changes in the Excitability of the Respiratory Centers of Aviators in a State of Fatigue. Franck, C., Grandpierre, R., Santenoise, D., and Stankoff, E. [*Compt. rend. soc. biol.*, **139**, 835–6 (1945).]

The fatigued state is characterized by an increase in blood CO_2 and a decrease in the sensitivity of the respiratory centers to changes in alveolar CO_2. Normally, the threshold of increase in pulmonary ventilation is 1–2 per cent. of CO_2 in the inspired air, but in the fatigued state this is raised to 3–6 per cent.
L. E. GILSON (Chem. Abstr.).

Urinary Excretion of Estrogenic Substances by Women Before and After Electric-shock Treatment. Bergouignan, M., and Demange, R. [*Compt. rend. soc. biol.*, **139**, 848–50 (1945).]

The results of electric shock treatment as practiced in insane hospitals were inconclusive. Possibly the excretion of estrogens was slightly increased in a few cases. L. E. GILSON (Chem. Abstr.).

Cerebral Lipides in Acute Aniline Poisoning. I. Determination of Cerebral Lipides by the Method of Frankel. Tarsitano, Francisco (*Univ. Naples*). [*Folia med.* (*Naples*), **27**, 830–5 (1941).]

Since the nervous system is particularly involved in aniline (1) poisoning, an examination of the changes which are produced in the brain was undertaken. Rabbits given subcutaneously 1·5 gm. of (1) per kgm. of body weight died within 24 hours. Following the method of Frankel, the brains of rabbits treated with (1), and of controls were subjected to (1) cold extraction by Me_2CO for 24 hours; (2) hot extraction by Me_2CO for 24 hours; (3) extraction by petr. ether for 48 hours; (4) extraction by C_6H_6 for 48 hours; (5) extraction by absolute alcohol for 48 hours, and (6) extraction by Et_2O for 48 hours. In (1) and (2), H_2O, cholesterol, and some unsaturated phosphatides are separated; in (3) unsaturated phosphatides and cerebrosides; in (4), (5) and (6) the saturated phosphatides and remaining cerebrosides. The values found for 5 treated and 5 control animals are, respectively, H_2O, 76·80–77·42 per cent. and 74·90–77·20 per cent.; (1) and (2) (per cent. of dry weight) 22·15–25·00 and 23·90–26·62; (3) 24·97–26·08 and 22·55–26·40; (4) 12·66–15·03 and 11·90–13·58; (5) 4·59–5·55 and 4·44–5·74, and (6) 0·69–1·39 and 0·73–1·45. The total lipides, then, range from 68·35–71·18 in treated animals and from 68·08–71·40 in controls on a dry weight basis. The effects on the brain of treatment with (1) appear to be an increase in H_2O, decrease in (1) and (2), increase in (3) and (4), with little change in (5) and (6) or in the total lipides.
WILLIAM F. BRUCE (Chem. Abstr.).

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Mechanism of Action of Sympathomimetic Amines. III. Oxidation of Tyrosine by Rat-liver Homogenates. Govier, Wm. M., Grelis, Mary E., Yanz, Naomi S., and Beyer, Karl H. (Sharp and Dohme, Inc., Glenolden, Pa.). [*J. Pharmacol.*, **87**, 149–58 (1946); *cf. C. A.*, **40**, 1228⁹.]

The oxidation of tyrosine by rat liver homogenate is stimulated by cytochrome c, coenzyme (1), α-keto acids, low concentrations of methylene blue, and by Al and Ca ions. p-Hydroxy-phenylacetic acid is strongly inhibitory to both tyrosine oxidation and tissue respiration. A possible mechanism of the reactions is discussed which suggests that " amine oxidase " is a mixture of several enzymes.

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

Action of Some Zoösteroids on the Excitability and Fatigability of Neuromuscular Preparations. Borgatti, G. [*Arch. sci. biol. (Italy)*, **29**, 160–88 (1943).]

Some zoösterols, and more specifically cholesterol, cholic acid, corticosterone, and testosterone were studied by chronaximetric and ergographic methods in regard to the excitability and work done by nerve-muscle preparations. All exhibited a positive neuromyotropic effect on the excitability and contractility of muscle. Corticosterone tended to hasten the return of isochronism between nerve and muscle which is necessary for transmission of an impulse, and the muscle under its influence also did more work. Cholic acid also favored the renewal of nervous excitability after fatigue, but exerted a negative action on work performance. Cholesterol and testosterone did not have a clear-cut effect. The common action of the various compounds on excitability is attributed to the common nucleus of these sterols, while the differences between them in respect to other neuromuscular activities are ascribed to the peculiar properties introduced by the different chemical side chains.

D. I. MACHT (Chem. Abstr.).

The Chemical Excitation of the Cerebral Cortex. Moussatche, H. (Inst. Oswaldo Cruz, Rio de Janeiro). [*Mem. inst. Oswaldo Cruz*, **42**, 365–400 (1945); *cf. C. A.*, **40**, 1935⁹.]

Solutions of acetylcholine were applied directly to the motor cortex of dogs, cats, monkeys and rabbits. When 10 per cent. solutions were used, localized muscular twitchings occurred in all animals, and 44·4 per cent. of the animals underwent generalized epileptic convulsions. Convulsions also occurred in some of the animals when 1 per cent. solutions were used and definite responses were generally observed with 0.2 per cent. acetylcholine. Convulsions were easily produced by rapid, light mechanical stimulations of the skin covering the muscles connected to the motor points. Eserine (1 : 10,000) increased the acetylcholine-induced muscular twitchings, and decreased the acetylcholine concentrations normally necessary to produce generalized epilepsy. Prostigmine injections had no effect, but morphine followed by motor cortex stimulation caused epileptiform convulsions. Nicotine in concentrations from 0·002 per cent. to 50 per cent., was a strong convulsant when applied to the motor cortex. Atropine either applied to the motor points in low concentrations (1 : 1,000) or injected in high concentrations (3–5 mgm. atropine sulfate) did not prevent the motor reactions of acetylcholine.

BRUNO VASSELL (Chem. Abstr.).

A Fourth-action Substance of Nerves. Weidmann, S. (Hallerianum, Bern). [*Experientia*, **1**, 61–2 (1945) (*in German*).]

A fourth, unknown substance (A_4) has been found polarographically in solutions containing cut nerves. More A_4 is found when the nerve is stimulated. An increase is again noted on stimulation after a period of rest. In degeneration the amount steadily decreases. The polarogram of Ringer solution is changed by A_4 so that the rise occurs 1·9 v. earlier. Some properties of A_4 were determined from nerve extracts which showed appreciable amounts of A_4 polarographically. The substance is dialyzable, remains in solution with sulfosalicylic acid precipitation; hence it is not a protein. It is thermostable, its activity not being changed after 2 hours heating at pH 7 in 0·1 N HCl or 0·1 N NaOH. It is destroyed by ultraviolet light. Its activity is dependent on pH.

A. EDELMANN (Chem. Abstr.).

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A Depressor Substance Extracted from Rat Intestine. Kahane, Ernest, and Levy, Jeanne (Univ. Paris). [*Bull. soc. chim. biol.*, **27**, 164–7 (1945).]

When dog intestine or stomach or rat intestine, lung, or brain is ground with Tyrode solution there passes into solution a substance M which, when the extract is heated to 70°, liberates an unidentified substance X. The latter provokes the contraction of leech muscle with or without pretreatment with eserine. The substance X is stable upon boiling in weakly acid or alkaline solution. Its chemical and pharmacological properties show it is not choline or a choline ester, a K salt, histamine, adenosine, the substance P of Euler and Gaddum (*C. A.*, **25**, 5440), the substance R of Chang and Wong (*C. A.*, **27**, 5395), or the depressor substance extracted from blood by alcohol by Fleisch (*Schweiz. Med. Wochen.*, **68**, 81 [1938]). In some respects it resembles the vasodilator substance extracted from fresh blood by Fleisch and Gavin-Franel (*C. A.*, **30**, 4221[8]). L. E. GILSON (Chem. Abstr.).

Chemical Bases of Nerve Impulse Transmission. Rosenbaum, H. [*Compt. rend. ann. et arch. soc. turque sci. phys. nat., Nos.* 8–9, 92–104 (1939–42) (*Pub.*, 1943) (*in German*).]

Review and critic discussion. Conclusion: transmission of nerve impulses is not entirely dependent upon chemical reactions; other non-chemical factors play an important part. BRUNO VASSEL (Chem. Abstr.).

Modification of the Regnier Chronaxia Method for the Measurement of the Excitability of Sensory Nerves under Local Anesthetics. Quevauviller, A. (Faculté de Pharmacie de Paris, France). [*Arch. Intern. Pharmacodynamie*, **69**, 361–81 (1944).]

A modification of the method (*C. A.*, **39**, 3356[3]) and a theoretical discussion of chronaxias and a calculation of the electric energy necessary to elicit the crossed extension reflex of the frog under certain conditions are given. Ringer-glucose solutions are necessary for stabilization of the nerve. Novocaine first lowers then raises the chronaxia, but the phase of lowered chronaxia does not correspond to the phase of pre-anesthetic hyperexcitability.

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

Toxic Effects of Oxygen and of Hydrogen Peroxide on Brain Metabolism. Mann, P. J. G., and Quastel, J. H. (City Mental Hosp., Cardiff). [*Biochem. J.*, **40**, 139–44 (1946); *cf. following abstract.*]

The respiration of minced brain tissue, especially in the presence of glucose, Na lactate or pyruvate, is smaller in O_2 than in air. In the presence of Na succinate the respiration is not quite so sensitive to O_2. Neither the lactic dehydrogenase nor diaphorase is inhibited by exposure of brain tissue to O_2. It appears that the part of the brain respiration system most sensitive to O_2 is the pyruvate oxidizing system. Xanthine oxidase is poisoned by O_2 probably through the intermediate formation of H_2O_2, while d-amino acid oxidase, though it is poisoned by O_2, can be protected by its substrate. The thiol enzyme pyruvic oxidase is thought to be the factor inhibited by high O_2 tensions, but this may be due either to O_2 or to H_2O_2. The toxic effect of the latter is especially noted in the presence of NaN_3.

S. MORGULIS (Chem. Abstr.).

Toxic Effects of Oxygen on Brain Metabolism and on Tissue Enzymes. I. Brain Metabolism. Dickens, F. (Natl. Inst. Med. Research, London, N.W.3). [*Biochem. J.*, **40**, 145–71 (1946); *cf. preceding abstract.*]

Respiration of brain tissue is slowly and irreversibly poisoned by exposure to high O_2 pressure (H.O.P.). The injury begins at 1 atmosphere and the rate steadily increases with rising pressure. At pressures of 1 to 5 atmospheres the time of exposure necessary to produce a certain percentage of poisoning is more or less inversely proportional to the O_2 pressure. A plot of the course of poisoning of the respiration of brain tissue against time of exposure gives a sigmoid shape similar to that of a probability curve. The metabolism of the O_2 poisoned brain tissue could not be restored to normal by any methods tried, including return to normal

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pressure. This poisoning is probably responsible for the convulsions under H.O.P. The order of sensitivity of the respiration of rat tissues to O_2 is as follows :—Brain cortex (finely ground) > brain cortex (slices) > spinal cord > liver > testis > kidney > lung > muscle. The oxidation of glucose, fructose, lactate, and pyruvate by poisoned brain tissue are all affected, but the ability to oxidize succinate is much less affected. The ability of the brain tissue to oxidize glucose decreases at the same rate as the ability to form lactic acid, and in each case the effect is irreversible. Traces of H_2O_2 poison brain respiration, the succinate oxidation being more sensitive. Poisoning with H_2O_2, unlike that by O_2, is much less severe in a catalase-rich tissue (liver) than in a catalase-poor tissue (brain). But no accumulation of H_2O_2 could be demonstrated in brain tissue under H.O.P. and the poisoning effect could not thus be ascribed to the formation of H_2O_2. Neither was the course of H.O.P. poisoning of the brain appreciably affected by the addition of vitamins B_1, B_2, B_6, and C, pantothenic acid, nicotinamide, adenylic acid, adenine, adenosine, cozymase, flavoprotein, fructose, pyruvate, α-ketoglutarate, NaBr, luminal, chloretone, or EtOH. Glutathione, SH-compounds, NH_4Cl, glutamic acid, or glutamine (0·001 M) increased the susceptibility of brain tissue to poisoning by H.O.P. The respiration of brain-cortex slices was protected against injury by H.O.P. by the following divalent metals : Mn > Co > Mg > Ca. Fe, Ni, or Cu had a slight protective action, but not Zn. In 5×10^{-5} M or higher concentration Co and Mn were very effective or almost completely counteracted the toxic action of H.O.P. but they could not restore an already poisoned respiration. It is suggested that H.O.P. poisons primarily the pyruvate oxidase system, probably by attacking the SH-groups of the enzyme, which are both activated and protected by Mg, Mn, and Co. Inhibition of this enzymic system would interfere with the oxidation of glucose, fructose, and lactate by blocking the regeneration of adenosine triphosphate. It would also inhibit lactic acid formation from glucose. However, it is not denied that other O_2 sensitive enzymes may also be affected by the toxic action of H.O.P.

S. MORGULIS (Chem. Abstr.).

II. Tissue Enzymes. [*Ibid.*, 171–87 (1946).]

At least 5 enzymes (phosphoglucomutase, fructose-6-phosphate phosphorylase, triosephosphate dehydrogenase, succinic dehydrogenase, and pyruvic oxidase) concerned in the carbohydrate metabolism of brain tissue can be inhibited by O_2, and of these, 4 are known to depend upon intact SH-groups for their activity. The connection is clear between their — SH nature and liability to O_2 poisoning, which agrees well with the knowledge of O_2 sensitivity of other — SH enzymes. Succinic oxidase system is inhibited by exposure to H.O.P. Its most sensitive component, the dehydrogenase, is apparently irreversibly poisoned. The accumulation of oxalacetic acid probably accounts only for a very small part of the poisoning effect. Both malonate (10^{-3} M) and Mn ions ($2{\cdot}5 \times 10^{-4}$ M) protect the dehydrogenase against the H.O.P. Cytochrome oxidase may also be somewhat poisoned by prolonged exposure to H.O.P. Lactic and malic acid dehydrogenases from animal tissues are very resistant to inactivation by H.O.P. Triosephosphate dehydrogenase is poisoned only when exposed to H.O.P. in the absence of cozymase. Typical flavoproteins (d-amino acid oxidase, diaphorase) are not affected by H.O.P., even up to 4·4 atmospheres O_2, while the flavine-adeninedinucleotide content of the brain tissue is not affected by exposure to 3·3 atmospheres O_2. Catalase is insensitive to O_2 but choline oxidase which is a — SH enzyme is poisoned. Yeast hexokinase is not affected by O_2 in the presence of glucose. The relationship between the presence of essential — SH groups in the enzymes sensitive to O_2 exposure is stressed.

S. MORGULIS (Chem. Abstr.).

Metabolism of Brain Suspensions. III. Respiration at Low Oxygen Tension. *Elliott, K. A. C., and Henry, Maryon* (*McGill Univ., Montreal.* [*J. Biol. Chem.*, **163**, 351–60 (1946) ; *cf. C. A.*, **37**, 924[4].]

The rate of respiration of brain tissue has been studied with Barcroft differential manometers, and suspensions of whole rat brain homogenates in warm Ringer, 0·033 M phosphate, 0·01 M glucose medium. The gas to be used was passed in series through the vessels before warming it in the 38° bath. Small O concentrations were determined in Barcroft manometers with N NaOH in both

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vessels. The gas was passed through the manometers in series and after equilibrium in the bath 0·8 c.c. M NaCl in 0·002 N H_2SO_4 + 0·1 M pyrogallol was added from the side arm of one vessel. Formulas are given for determination of O. The results show the rate of respiration is unaffected by reduction of O tension to as low as 4 mm. of Hg provided the rate of diffusion of O into the suspension medium does not limit O uptake. The rate of diffusion can be increased by decreasing the tissue concentration or by increasing the rate of shaking. The full rate of respiration was obtained with a glucose concentration as low as 10^{-4} M.

E. G. SHIPLEY (Chem. Abstr.).

IV. Anaerobic Glycolysis. [*Ibid.*, 361–74.]

Homogenized rat brain was suspended in warm Ringer-bicarbonate-glucose medium, and glycolysis was followed manometrically in the Barcroft apparatus. The results showed that the maximum stimulation to glycolysis by pyruvate was reached with 5×10^{-5} M pyruvate. A preliminary period of aerobiosis briefly stimulated, and a trace of O will continuously stimulate glycolysis. The rate of glycolysis was stimulated by washing the suspension; the effect was due to removal of a system which destroys pyruvate. Washed suspensions were found to contain increased amounts of pyruvate during the period of stimulated glycolysis. By adding the washings to fresh suspensions of brain glycolysis was inhibited. The inhibitory effect was largely abolished by adding pyruvate. An increase in concentration of tissue decreased glycolysis per unit weight of tissue, and was not all due to an increase in pyruvate destruction. Mg was found to be necessary for stimulation of glycolysis after washing the suspension. Added lactate did not inhibit glycolysis in the absence of added pyruvate, but inhibited glycolysis when pyruvate was added. Rat and horse serum inhibited glycolysis although serum did not destroy lactate and in itself contained enough pyruvate to stimulate glycolysis. The nature of the inhibitor in serum was not determined. In Ringer-phosphate-glucose medium the lactic acid production was lower than in Ringer-bicarbonate-glucose, but large amounts of unidentified acid were formed. The anaerobic glycolysis of suspensions of whole brain was found to be less variable than that of slices and duplicates agreed very closely, and were nearly the same as the average rate for slices. The possibility that glycolysis is a normal functional process is discussed.

E. G. SHIPLEY (Chem. Abstr.).

The Neuro-muscular Transmission: Curarization, Decurarization, Reinforcement at the Myo-neural Junction. *Coppée, G.* (*Fredericq Inst., Liege*). [*Arch. intern. physiol.*, **53**, 327–507 (1943); *cf. C. A.*, **38**, 4319[2].]

Extensive experiments were performed on cat and frog (*Rana esculenta* and *R. temporaria*) muscles, either isolated or *in situ*. The mechanical response of the muscle was registered isometrically upon a smoked cylinder while the electric response was analyzed after amplification by a cathode oscillograph. A functional organelle (designated " motor plate ") was found between the termination of the motor nerve and the muscle fibers. Curarization of muscle, just adequate to abolish the mechanical response, followed by an indirect stimulus, produced in the motor plate a brief electric potential followed by a negative after-potential. During neuro-muscular block by K^+ ions or acetylcholine (1) in the presence of eserine, or after section of the motor nerve, the electric response of the muscle to a motor volley is composed of an action potential of the motor plate on which is superimposed an action potential not originating in the muscle fiber; the negative after-potential will be weak, or replaced by a positive after-potential. The response of the motor plate is not determined by (1) liberated at the end of the nerve fiber; the transmission from nerve to plate is assured by an electric mechanism and is characterized by a measurable synaptic delay. The transmission of the excitation from the plate to the muscle fiber occurs as soon as the slow wave exceeds critical voltage; at that moment a brief potential of muscle fiber becomes superimposed on the slow wave. That brief potential may be diffused and accompanied by contraction or not diffused and not accompanied by contraction. The " critical voltage " is conditioned by the excitability of the muscle fibers. Two factors in transmission from plate to muscle fibers are: (1) the energy of the stimulus measured by the amplitude of the slow wave; (2) the excitability of the muscle fibers for that

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stimulus. Most of the decurarizing agents (e.g., phenols, guanidine, veratrine, negative polarization) augment the amplitude. K^+ ions augment and Ca^{++} ions depress the excitability of the muscle fibers, and veratrine appears to produce no change in their excitability. The transmission from plate to muscle fiber occurs normally by an electric mechanism similar to that of the nerve impulse along the axone, and is characterized by a non-measurable latent period. Agents which increase the amplitude of the slow wave decrease the delay. (1) is liberated in the motor plate and acts upon the muscle fibers, but it is too weak or too late to assure transmission, which has been assured by the electric mechanism. In specific cases (as with certain muscles of the cat) and on muscle treated with eserine, the (1) liberated by the plate effects excitation of muscle fibers. All the effects from excitation of muscle fibers, of potentiation or depression of neuromuscular transmission produced by (1), can be explained by the intervention of K^+ liberated by the (1). The (1) unites with a radical (R —) of an organic K complex to displace K^+. Normally the R — (1) is rapidly hydrolyzed in the presence of choline esterase, but the compound is not stable and R — K would become reconstituted. The inhibitors of choline esterase hindered the hydrolysis of (1) and stabilized R — (1) and blocked the radical R — for the final reaction (desensitization of muscle to (1)); in addition, the excess of K^+ alters the functioning of the transmission apparatus (this explains the similarity of electric manifestations in muscles curarized by excess K^+ and by (1) in presence of eserine). When stable non-hydrolyzable excitatory substances (quaternary ammonium, nicotine, cytisine) act upon muscle the excitations are equally due to K^+ displaced at the beginning of the R — K complex, but the depression of the neuro-muscular transmission is not due to an excess of the ions. The curarizing agents act upon the motor plate to depress the response to a motor-nerve impulse. The curarizing agents (except the metallic ions) unite with R — of the organic K complex, and produce a progressive leak of muscle K, responsible for depolarization of the motor plate, for lowering the amplitude of the wave from the motor plate, and for the block of transmission from plate to muscle fiber. Ca^{++} and Mg^{++} act as direct antagonists to muscle K^+ Summation occurs at the interface of nerve and plate and not at plate and muscle fiber. The increased reaction of the plate to the second impulse is due to the negative after-potential produced by the first impulse. When the negative after-potential is absent, there is only a long refractory period. (1) intervenes in summation only at the myo-neural junction. Neuro-muscular transmission can be restored either by increase in amplitude of the slow wave (type: phenol, guanidine, veratrine) or by negative polarization. Decurarization by eserine and the anti-curare action of adrenaline are discussed. Some agents such as guanidine and veratrine are extremely active because they re-establish transmission in myo-neural units and render the muscle fibers repetitive. Reinforcement is concerned either with the repetitive response of muscle fibers (type: eserine, sensitivity to K^+ ions) or with a modification of the muscle contractile apparatus (as by K^+ and an acid pH). Veratrine action is very specific; reinforcement by eserine is due to its anti-esterase activity.

ELVA G. SHIPLEY (Chem. Abstr.).

Modifications of the Phosphorus in Plasma Caused by Anoxia. Cicardo, Vicente H. [*Pubs. centro invest. tisiol. (Buenos Aires)*, **9**, 271–81 (1945).]

In dogs in which muscular contraction is abolished by spinal cord destruction or anesthesia the total acid-soluble plasma P increases during asphyxia. Hepatectomy does not modify this increase but surgical shock produces a further rise. Curarizing drugs which cause a drop in plasma P prevent the liberation of P from the tissues. Anoxia by HCN produces an increase of the total acid-soluble plasma P which occurs by way of depression of the respiratory center as is proved by the fact that the increase does not appear if artificial respiration is applied.

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

Further Studies on the Biochemistry of Epilepsy. Madsen, Jrøgen (St. Hans' Hosp., Roskilde, Denmark). [*Acta Psychiat. Neurol.*, **18**, 257–337 (1943) (*in English*).]

Hyperammoniurics (usually with epilepsy) showed essentially low normal serum Na, K and Ca, with the Na and Cl falling under the influence of theophyllin, particularly in patients in the convulsive phase. The hyperammoniurics showed

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lowered water and increased Na excretion during water diuresis and the excretion of water, Na and Cl after salyrgan injection was variable, and after theophyllin injections the excretions varied with the Na and Cl not always parallel. The normal morning urine Cl/Na ratio is 1·26, but in the epileptics it was found to be 1·55; this indicates a tendency for retention. The ratio fell after diuretic administration and increased after the injection of pitressin. The hyperammoniuria decreased during liver therapy (exhepa).[6] The mechanism of the hyperammoniuria and its control are discussed. H. L. WILLIAMS (Chem. Abstr.).

Reaction of the Blood in Epilepsy. Faurbye, Arild (Hosp. for Epileptic Dis., Dianalund, Denmark). [*Ibid.*, 419–38 (*in English*).]

The pH is and remains normal. H. L. WILLIAMS (Chem. Abstr.).

New Studies on the Variations in Nervous Excitability under the Influence of Different Sugars. Chauchard, Paul, Mazoué, Henriette, and Lecoq, Raoul. [*Compt. Rend.*, **221**, 643–5 (1945).]

White rats were given daily intraperitoneal injections of 50 mgm. glucose, levulose, galactose, saccharose, maltose, lactose, sorbitol, or mannitol. After the fifth day there was observed a chronaxial effect on the nerves indicative of a depression of the nerve centers. The injection of glucose or levulose into rats produced an immediate chronaxial increase even though the animals were regularly receiving these sugars. The other sugars did not produce this immediate effect.

H. L. G. (Chem. Abstr.).

2. Pharmacology and Treatment.

Experimental Therapy of Acute Barbiturate Poisoning, II. Zancan, Lanfranco (Univ., Padova, Italy). [*Boll. soc. ital. biol. sper.*, **15**, 591–2 (1940).]

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

Modification of Chronaxia of Subordination by Diallylbarbiturate. Bartorelli, Cesare (Univ., Bologna). [*Boll. soc. ital. biol. sper.*, **15**, 624–5 (1940).]

In rabbits Na 5,5-diallylbarbiturate abolished the state of subordination, of chronaxias producing an equalization of the antagonistic muscles of the hind leg.

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

Toxicity and Narcotic Action of Some Derivatives of Barbituric Acid. Donatelli, L., and Abbate, R. (Univ. Firenze, Italy). [*Boll. soc. ital. biol. sper.*, **15**, 838–9 (1940); *cf. C. A.*, **33**, 3885[7].]

Effects of some common barbiturates on rats and rabbits are described.

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

Action on the Heart of Some Derivatives of Barbituric Acid Used as Narcotics. Donatelli, L. [*Ibid.*, 839–40; *cf. C. A.*, **38**, 2387[1].]

Effects of several commonly used barbiturates on isolated frog, rabbit, and guinea-pig hearts, and on the rabbit heart *in situ* are described.

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

Pyruvic Acid Antagonism to Barbiturate Depression. Westfall, B. A. (Univ. of Missouri, Columbia). [*J. Pharmacol.*, **87**, 33–7 (1946).]

Data on 64 rabbits anesthetized with Na pentobarbital (40 mgm./kgm. intravenously) indicate that adrenaline increases the blood-sugar level and shortens the sleeping time, insulin decreases the blood sugar and also shortens the sleeping time, and intravenous injection of Na pyruvate decreases the depth of depression and shortens the sleeping time but does not materially alter the total depression time. Adrenaline probably exerts the above action by increasing blood pyruvate and insulin by increasing pyruvate metabolism. All these results suggest that utilization of

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pyruvic acid may be the step in carbohydrate metabolism depressed by pentobarbital. The experimental data indicate an inverse relationship between the available pyruvic acid in the blood and the degree of depression produced by the drug.
L. E. GILSON (Chem. Abstr.).

The Determination of Barbiturates in Toxicological and Clinical Investigations. Cohen, Eugene L. (Philadelphia Coll. of Pharmacy and Sci., Pa.). [*Am. J. Pharm.*, **118**, 40–62 (1946).]

The barbiturate-cobalt reaction can be used for quantitative estimations and the optimum wave length for the measurement of the color compound produced in the barbiturate-Co reaction is 5650 A., with 0·2 ml. of a 1 per cent. cobaltous acetate solution in absolute methanol and 0·6 ml. of 1 per cent. isopropylamine in absolute methanol. Read at this wave length, the color of the reacting agents does not interfere with the color produced in the reaction, and at concentrations of 1 to 10 mgm. of barbiturate per ml. of $CHCl_3$ solution, the colour produced in the reaction was proportional to the barbiturate content. It is not possible to employ one barbiturate as a standard for the determination of all barbiturates. In like manner, a factor based on the ratio of molecular weights, as is practiced in the determination of the sulfonamide drugs, cannot be employed in the quantitative estimation of the barbiturates. A procedure was devised, based on the Stas-Otto method (*cf.* Autenrieth, *Lab. Manual for the Detection of Poisons and Powerful Drugs*, *C. A.*, **22**, 1829) for the extraction of the barbiturate drugs from animal tissues, whole blood and urine, requiring a minimum of apparatus and technical skill. By this procedure, recoveries of the barbiturate compounds from tissues and fluids of 93 per cent. or more were obtained in 57 estimations. Possible sources of error were noted and recorded. A simple apparatus was also devised for the purification of the barbiturate drugs by sublimation, prior to identification by m.p. and other determinations. W. G. G. (Chem. Abstr.).

Effects of Continued Administration of Phenobarbital and Diphenylhydantoin. Hanzlik, P. J., and Laqueur, G. L. (Stanford Univ., San Francisco, Calif.). [*Stanford Med. Bull.*, **4**, 21–32 (1946).]

Phenobarbital (I) and diphenylhydantoin (II) were administered to white rats in concentrations of 0·1 and 0·01 per cent. in the diet over a period of 16 months. The greater injurious effect of (I) as compared with (II) was demonstrated by greater decrease in body weight and growth, greater mortality, more frequent nasal bleeding and encrustation of eyelids, and generally a decreased activity in the group receiving (I). No evidence could be found of antagonism to the effect of feeding (I) by administration of sodium succinate. Animals showing toxic effects of (I) had no anatomically demonstrable changes in the kidneys, liver, brain, heart, lungs, and gonads; however an enlargement of the parenchymatous liver cells was noted in the majority of these cases. D. A. SHIRLEY (Chem. Abstr.).

Site of Action of Narcotics on Brain Metabolism. Greig, Margaret E. (Vanderbilt Univ. School of Med., Nashville, Tenn.). [*J. Pharmacol.*, **87**, 185–92 (1946).]

Previous experiments have shown that the dehydrogenase and that part of the cytochrome system involved in the oxidation of succinate are relatively insensitive to narcotics. There are two possible oxidation-reduction reactions on which narcotics might exert their effect in inhibiting the carbohydrate metabolism of the brain. They are (1) the transfer of H from reduced cozymase to flavoproteins, and (2) the oxidation of flavoproteins by the cytochrome system by electron transfer. Experiments showed that reduced cozymase did not accumulate during the carbohydrate metabolism of brain in presence of nembutal, and that the oxidation of reduced cozymase by methylene blue catalyzed by flavoprotein was not affected by nembutal. This indicates that the block is not at the position suggested in (1). The oxidation of lactate by yeast is also inhibited by nembutal. The fact that the yeast lactic enzyme, which like the enzyme in animal tissues does not require cozymase for activity, was inhibited is further evidence that cozymase is not involved and that the block occurs at cytrochrome b. It is possible that the narcotic

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acts by binding the reduced flavoprotein with cytochrome b (or another intermediate) and that the affinity of nembutal for this complex is greater than for the succinic dehydrogenase-cytochrome b complex which is not affected by low concentrations of narcotics. L. E. GILSON (Chem. Abstr.).

New Ideas on Mechanism of Narcosis. Valázquez, B. L., and Elio Membrado, F. J. [*Farmacoterap. actual* (*Madrid*), **1**, *No.* 5, 28–33 (1944).]

The action of acetylcholine on *in vitro* muscle preparations of the frog and leech in the presence of chloral hydrate, thiamine, and morphine hydrochloride is described. Cerebrospinal fluid drawn from the cisterna magna of the anesthetized dog has less acetylcholine than normal. It is suggested that the immediate mechanism of narcosis involves the inhibition of acetylcholine synthesis by the direct action of the narcotic on cell metabolism. B. A. (Chem. Abstr.).

Electrocardiographic Studies During Convulsions Produced by Metrazole and Azoman (*3-ethyl-4-cyclohexyl 1,2,4-triazole*). *de Haene, A., Delaunois, A. L., and Pannier, R.* (*Univ. Gand, Belgium*). [*Arch. intern. pharmacodynamie*, **69**, 309–47 (1944).]

Azoman produces changes similar to metrazole on the human electrocardiogram, namely, tachycardia and changes in the T-wave and the S-T interval. Intravenous injection of 1·0 mgm. into frogs causes augmentation of the T-wave which is abolished when the cord is destroyed. 5 to 20 mgm. causes irregularities in the Q-R-S complex and depression of the T-wave. This occurs in normal as well as in sympathectomized frogs and also in those with the cord destroyed. The small doses increase the rate and amplitude of the beat, and the large ones depress. Strychnine behaves like small doses of metrazole. Azoman (1·0 mgm.) causes disappearance of the T-wave in normal frogs and a decrease in amplitude in frogs with the cord destroyed. In both cases, a small increase in the Q-R-S complex occurs. Azoman (5–20 mgm.) produces bradycardia and changes in the Q-R-S complex and T-waves whether the cord is destroyed or not or the frog is sympathectomized. In the dog, azoman causes tachycardia, shortening of the P-R and S-T intervals, and increase in the T-wave followed by a depression. M. L. C. BERNHEIM (Chem. Abstr.).

Localization and Origin of the Convulsions Produced by Metrazole and Coramine. ten Cate, J., and Swijgman, D. W. (*Univ. Amsterdam, Holland*). [*Arch. intern. pharmacodynamie*, **70**, 293–306 (1945).]

Metrazole (27 mgm./kgm.) injected intraperitoneally causes convulsions in cats. Removal of the motor cortex prevents convulsions, and in decapitate cats at least twice the dose is necessary to produce them. 80 mgm./kgm. coramine intravenously produce convulsions in both normal and decorticated cats. In decapitated animals smaller doses are effective, indicating that coramine acts primarily on the spinal cord. M. L. C. BERNHEIM (Chem. Abstr.).

The Alleged Convulsive Action of Pyridoxine. Saviano, Mario (*Univ. Naples*). [*Boll. soc. ital. biol. sper.*, **16**, 763–4 (1941).]

In mice, rats, and rabbits extremely heavy doses of pyridoxine increased neuromuscular excitability and occasionally provoked transient clonic spasms. Blood Ca and phosphate were not affected; blood sugar was slightly increased. L. E. GILSON (Chem. Abstr.).

Morphine Habituation and Abstinence in the Light of Kravkov's Theory of Stages in Toxic Action. Gorovoi-Shaltan, V. A. [*Farmakol. i Toksikol.*, **7**, *No.* 6, 26–30 (1944).]

Clinical evidence supports the following data concerning morphine effects in three stages, where + = stimulation, − = depression, + − = collapse:

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	Phase I. Euphoric (incipient).	Phase II. Narcotic (saturation).	Phase III. Abstinence (final).
Cortex	+	−	+ −
Subcortical	−	−	+
Vegetative nervous system :			
Parasympathetic	+	+	+
Sympathetic	−	−	+
Endocrine glands :			
Parathyroid and pancreas, thyroid	+	+	−
Suprarenal, cerebral appendage, reproductive glands	−	−	+

The mode of administering morphine influences both the rate of development and the duration of the first phase. Clinical results in studies of habituation and abstinence proved to be most informative when studied in the light of Kravkov's theory of phases in toxic action. JULIAN F. SMITH (Chem. Abstr.).

The Effect of Glutamic Acid on the Hydrogen Ion Concentration (pH) of the Urine in petit mal Types of Epilepsy. Spangler, Ralph H. (2221 *S. Broad St., Philadelphia, Pa.*). [*Ann. Allergy*, **3**, 241–57 (1945).]

Administration of 1·5–4·5 gm. of dl-glutamic acid-HCl daily resulted in urine with pH 4 on 59 of 114 days. d-Glutamic acid-HCl maintained pH 4 on 89 of 121 days in doses of 4·64–11·6 gm. Natural (+)-glutamic acid was less effective in doses of 10 gm. H. L. MASON (Chem. Abstr.).

Humoral Changes After Insulin Therapy. Delay, Jean, Soulairac, A., and Jouannais, S. (*Univ. Paris*). [*Compt. rend. soc. biol.*, **139**, 460–2 (1945).]

In human subjects given enough insulin to produce coma after about 3 hours, blood sugar descended to 36 mgm. per cent., alkali reserve decreased to 54 volumes per cent., blood proteins increased a little (maximum value observed was 8·8 gm./100 c.c.), and serum lipides showed a marked increase. Normal conditions were restored within an hour after administration of glucose.
L. E. GILSON (Chem. Abstr.).

Action of Transcerebral Electrophoresis of Calcium on the Ovaries of the Rat Before and After Hypophysectomy. Bourguignon, Georges (*Coll. France*). [*Compt. rend. soc. biol.*, **139**, 568–70 (1945); *cf. C. A.*, **40**, 1594[8].]

Two positive pad electrodes were placed on the eyes and the negative electrode on the base of the occiput, and 0·5–0·6 ma. d.c. was applied for 10–45 minutes. In intact rats passage of the current for 3–4 minutes produced turgescence first of the graafian follicles then of the entire ovary, and dilation of the blood vessels of the uterine horns. The maximum effect was reached in approximately 10 minutes. If the current was continued for 15 minutes or longer the turgescence and congestion gradually disappeared. In rats hypophysectomized 5–10 days previously no such effects were produced by the current. L. E. GILSON (Chem. Abstr.).

Neutralization of Nerve-exciting Effects of Caffeine by Thiamine and Adrenaline. Chauchard, Paul, and Mazoué, Henriette (*École hautes études, Paris*). [*Compt. rend. soc. biol.*, **139**, 640–1 (1945); *cf. C. A.*, **40**, 1932[1], 1933[6], 3528[5].]

Like the sugars, either adrenaline or thiamine counteracts the effect of caffeine on nerve chronaxia. L. E. GILSON (Chem. Abstr.).

Influence of Acute and Chronic Carbon Monoxide Poisoning on the Activity of Higher Nerves in Animals. Gorsheleva, L. S. [*Farmakol. i Toksikol*, **7**, *No.* 5, 47–51 (1944).]

Chronic poisoning by CO in doses too small to be detected by blood tests can be diagnosed by the method of conditioned reflexes. There is evidence of direct

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action on the central nervous system, with sharper reaction than in mild single-dose poisoning. The earliest effects in chronic CO poisoning include cortical changes in the upper parts of the central nervous system. Trophic changes in the skin also occur. JULIAN F. SMITH (Chem. Abstr.).

Idiopathic Hypoparathyroidism with Mental Deterioration: Effect of Treatment on Intellectual Function. Mortell, E. J. (Univ. of Virginia, Charlottesville). [*J. Clin. Endocrinol.*, **6**, 266–74 (1946).]

Serum Ca rose from 4·5 to 10·2, inorganic P fell from 9·6 to 3·1 mgm. per cent., and alkaline phosphatase fell from 5·6 to 3·8 Bodansky units during several months' treatment of a case of idiopathic hypoparathyroidism in a girl 19 years old with $CaCl_2$ and vitamin D given orally. The mental deterioration which had developed as the disease progressed showed no improvement. Intracerebral calcification was shown by X-ray examination. KATHRYN KNOWLTON (Chem. Abstr.).

Adrenal Function and Cholesterol Metabolism in Narcosis. Abelin, I. (Hallerianum Inst., Univ. Bern). [*Helv. Physiol. Pharmacol. Acta*, **4**, 1–9 (1946) (*in German*).]

In rats and guinea-pigs anesthesia by inhalation of Et_2O or $CHCl_3$ or by injection of numal causes a temporary increase of 100 per cent. or more in the cholesterol content of the adrenals. In guinea-pigs, blood cholesterol decreases as the adrenal cholesterol increases. Other known effects of narcosis on blood components are discussed. L. E. GILSON (Chem. Abstr.).

Pharmacology of Some Sympathomimetic Drugs Given by Cisternal Puncture (Dogs). Velázquez, B. Lorenzo, and de Armijo Valenzuela, M. [*Farmacoterap. actual (Madrid)*, **1**, *No.* 2, 29–34 (1944).]

Chloralosed dogs were given injections of 0·125–0·5 mgm. of adrenaline, 10–20 mgm. of ephedrine, or 2·5–5 mgm. of amphetamine, direct into the cisterna magna, blood pressure and respiration changes being recorded. The effect was in the order, ephedrine greater than amphetamine, greater than adrenaline. Repeated injections of the same or of the other drugs were also carried out, and the results are discussed. B. A. (Chem. Abstr.).

Expectorant Action of Parasympathomimetic Drugs. Boyd, Eldon M., and Lapp, M. Shirley (Queen's Univ., Kingston, Canada). [*J. Pharmacol.*, **87**, 24–32 (1946).]

Approximately 400 animals, including albino rats, guinea-pigs, rabbits, cats and dogs were arranged for collection of respiratory tract fluid (RTF). Eleven parasympathomimetic drugs, acetylcholine bromide, acetyl-β-methylcholine chloride (mecholyl), carbaminoylcholine chloride (carbachol), β-methylcholine urethan (urecholine), fur-furyltrimethylammonium iodide (furmethide), physostigmine, prostigmine, pilocarpine, arecoline, and the dimethylurethans of m-isopropyl-p-dimethylaminophenol methiodide and p-dimethylaminothymol methiodide, were given subsutaneously in a range of doses. Practically all of the drugs increased the volume output of RTF, some to over 1,000 per cent. Atropine abolished this effect when produced by pilocarpine, selected as an example of the group. Section of the cervical vagus nerve had no effect on the action of pilocarpine in cats. Pilocarpine was as effective in decerebrate as in urethanized cats. The mean specific gravity of cat RTF was slightly lowered by parasympathomimetic drugs, and the relative viscosity was increased. In general, the chloride content of RTF was increased by the drugs. L. E. GILSON (Chem. Abstr.).

Sympathomimetic Amines: The Relation of the Structure to their Action and Inactivation. Beyer, Karl H. (Sharp and Dohme, Inc., Glenolden, Pa.). [*Physiol. Rev.*, **26**, 169–97 (1946).]

The formation of pressor substances with a basic β-phenylethylamine nucleus is discussed. The nature of the etiologic agents in hypertension is considered as well as the hypothetical relation of dopa to essential hypertension. This naturally led to the use of phenol oxidase (tyrosinase) in the treatment of hypertension but the effects were shown to be non-specific. Much interest centers in quinones as

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antipressor agents. It was shown that 2-methyl-1,4-naphthoquinone (having vitamin K activity) reduces blood pressure of acutely hypertensive rats, but the quinones are not effective in human cases of hypertension or in dogs with renal hypertension. The mode of action of sympathomimetic amines is not well understood but more is known about the mechanism of their inactivation. Amine oxidase is widely implicated in this inactivation. The presence of amine oxidase in the body, particularly in the liver, determine whether pharmacologically active derivatives of β-phenylethylamine are effective and are excreted as such when administered by mouth. Derivatives, wherein the NH_2 group is on the terminal C of the side chain, are deaminated and are not active or excreted as such unless a hepatotoxic agent has been administered previously. But those in which the NH_2 group does not have the terminal position on the side chain are not appreciably deaminated and are active orally. There is a certain rough parallelism between rate of deamination and pressor activity. The relation of the ascorbic-dehydroascorbic acid system to the deamination of sympathomimetic agents is discussed, as well as the inactivation by conjugation. The renal excretion of the sympathomimetic amines depends upon the mode of inactivation. Since phenylethylamine derivatives, where the NH_2 is not on a terminal C, are refractory to deamination, they are generally excreted as such. But p-hydroxy derivatives in part, and compounds with a catechol nucleus predominantly, are excreted in conjugated form. As pressor agents, even the best of the aliphatic amines is less active than ephedrine and all demonstrate tachyphylaxis. In the presence of an aromatic nucleus, the NH_2 group on the side chain should be removed at least one C atom; aniline has no pressor activity while β-phenylethylamine has maximum activity. Substitution of alkyls or aryls on the α or β carbon reduces, abolishes, or reverses the sympathomimetic effect of the agent. Substitution of an alkyl group on the NH_2-N practically always decreases the pressor effect. The m-OH is the strongest while the o-OH compound is the weakest, but if 2 OH groups are introduced into the nucleus, the pressor activity depends on their mutual relation as well as on the relation to the side chain. The true sympathomimetic drugs have the o-dihydroxy substitution in the m- and p-positions. Arterenol, β-(3,4-dihydroxyphenyl)-β-hydroxyethylamine is more active than epinephrine and is supposed to be sympathin. S. MORGULIS (Chem. Abstr.).

Toxicity of Sulfanilamide on Higher Nervous Activity. Gantt, W. Horsley, and Marshall, E. K., jun. (*Johns Hopkins Med. School, Baltimore, Md.*). [*Bull. Johns Hopkins Hosp.*, **77**, 104–15 (1945).]

Sulfanilaminde in doses ranging from 0·2 to 1·2 gm./kgm. body weight (in excess of therapeutic dosages) has a depressing action upon all conditioned reflexes in two dogs with well-established salivary conditional reflexes and in two with cerebellar motor reflexes. The maximum effect is reached within 1 hour and disappears within 24 hours. A. EDELMANN (Chem. Abstr.).

Suppression of the Neuromuscular Troubles of Experimental Chronic Alcoholism by Injection or Ingestion of Vitamins B_3 and B_4. Lecoq, Raoul, Chauchard, Paul, and Mazoué, Henriette. [*Compt. rend.*, **222**, 414–6 (1946).]

Repeated amounts 5–10 gm./kgm./day, of alcohol were given to adult rats until they developed symptoms of polyneuritis. The neuromuscular conditions of the disease were not altered by giving thiamine or riboflavin, but they were improved by a combination of 100γ of vitamin B_3 (nicotinamide) and 150γ of B_4 (adenine). J. R. PORTER (Chem. Abstr.).

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