Part II.—Bibliography and Epitome.*

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

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.* (now *Biblio. Psychiat. Neur.*).
Abh. Psychother.
2 *Acta Luso-Española Neur. y Psiquiat.*
2, 4 *Acta Neurol., Naples.*
2, 4 *Acta Neurol. Psychiat. Belg.*
1, 2, 3, 4 *Acta Psychiat. et Neurol., Copenhagen.*
3 *Acta Psychol., Hague.*
Acta Psychol., Keijo.
Alcohol Hyg.
Alg. Neder. Tijdschr. Wijsbegeert. Psychol.
2 *Alienist v. Neurologist.*
Aliéniste Français.
2 *Allg. Ztschr. f. Psychiat.*
2 *Altersprobleme.*
Amer. Imago.
1, 2, 4 *Amer. J. Ment. Def.*
1, 2, 3, 4 *Amer. J. Orthopsychiat.*
1, 2, 3, 4 *Amer. J. Psychiat.*
Amer. J. Psychoanal.
2, 3, 4 *Amer. J. Psychol.*
2 *Amer. J. Psychotherap.*
Amer. Psychologist.
An. Bras. Hig. Ment.
An. Istit. Psicol. Univ. B. Aires.
4 *An. Port. Psiquiat.*
An. Psicotec., Rosario.
2 *Anal. Inst. Neurol., Montevideo.*
Analele Psihol. (Rumania).
Anales de psicol., Buenos A.
2, 3 *Année Psychol., Paris.*
2, 3, 4 *Ann. Méd.-Psychol., Paris.*
2 *Ann. Osp. psichiatr., Perugia.*
2 *Appl. Psychol. Monogr., Washington.*
Arb. Psychiat. Inst., Sendai.
2 *Arch. Antrop. crim.*
2 *Arch. Argent. de Neurol.*
Arch. Argent. Psicol. norm. pat.

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

https://doi.org/10.1192/bjp.96.402.298 Published online by Cambridge University Press

2 *Arch. Bras. de Neur. e Psiquiat.*
Arch. Brasil Hig. Ment.
Arch. Chilenos de Crim.
Arch. Ital. di Studi Neuropsich.
2 *Arch. Neurobiol.*
2 *Arch. Neurocir., Paraguay.*
2, 4 *Arch. Neurol., Paris.*
2 *Arch. de Neurol. de Bucarest.*
Arch. de Neurol.
2 *Arch. di Antropol. Crim.*
Arch. di Crim. Neuropsiquiat. y Disc. Con., Quito.
4 *Arch. di Sci. Cereb. Psich., Salerno.*
2, 4 *Arch. f. Psychiat.*
2, 3 *Arch. ges. Psychol.*
2, 4 *Arch. Internat. de Neurol.*
Arch. Ital. Psicol.
Arch. Ital. Sci. Neur.
Arch. Krim. Anthrop.
1, 2, 3, 4 *Arch. Neurol. Psychiat., Chicago.*
Arch. Neur. Psychiat., Mex.
2, 3, 4 *Arch. Psicol., Neurol., Psichiat. e. Psicoter., Milan.*
2, 3 *Arch. Psychol., Genève.*
2, 3 *Arch. Psychol., N.Y.* (ceased publication 1945).
Arch. Relig. psychol.
Arch. Speech.
4 *Arch. Venez. Soc. Oto.-rino.-lar.-oft.-neur. Caracas.*
Arq. do Assist. a Psicop. de Pernambuco.
4 *Arq. Assist. Psicop. Sao Paulo.*
2, 4 *Arq. de Neuro-psiquiat., Brasil.*
3 *Aust. J. Psychol. Phil., Sydney.*

Behaviour.
Beih. Schweiz. Z. Psychol. Anwend.
Beih. Zbl. Psychother.
Biblio. Psychiat. Neur.
Bol. Inst. Psiquiat., Rosario.
Bol. Inst. psicopedag., Lima.
1, 2, 3, 4 *Brain.*
Brit. J. Addiction.
3 *Brit. J. Educ. Psychol.*
4 *Brit. J. Hypnot.*
2, 4 *Brit. J. Inebriety* (now *Brit. J. Addiction*).
1, 2, 3, 4 *Brit. J. Med. Psychol.*
2, 3, 4 *Brit. J. Psychol.*
2, 3 *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.
2 *Bull. industr. Psychol., Melbourne.*
2, 4 *Bull. Los Angeles Neurol. Soc.*
2, 4 *Bull. Menninger Clin.*
Bull. Milit. Clin. Psychol.
Bull. Soc. Psihol. med., Sibiu.

4 *Canad. Journ. Occup. Ther.*
4 *Canad. J. Psychol., Toronto.*
2, 4 *Cervello.*
2, 3 *Character and Per.* (now *J. Personality*).
2 *Child Develpm.*
2 *Child Develpm. Abstr.*
Child Develpm. Monogr.
Child Study.
Chin. J. Educ. Psychol., Chungking.
Chin. J. Psychol.

https://doi.org/10.1192/bjp.96.402.298 Published online by Cambridge University Press

3 *Comp. Psychol. Monogr.*
2, 4 *Confinia Neurol.*
Contr. del Lab. di Psichol.
Contr. Lab. Psicol., Milan.
Contr. psychol. Theor.
4 *Criança Portuguesa.*
Criminalia, Mexico.

2, 4 *Deutsche Ztschr. f. Nervenh.*
2, 4 *Dig. Neurol. Psychiat.*
2, 4 *Dis. Nerv. Syst.*

3 *Educ. psychol. Measmt., Washington.*
Egypt. J. Psychol., Cairo.
2, 4 *Electroenceph. Clin. Neurophys., Boston.*
1, 2, 3, 4 *L'Encéphale.*
2 *Epilepsia.*
2 *Evolut. Psychiat.*
1, 2, 4 *Excerpta Medica, Sec. VIII, Neur. Psychiat.*

Fiziol. Th. S.S.S.R.
2 *Folia Neuropath. Esthon.*
2, 4 *Folia Psychiat. Neur. Neurochir., Amsterdam.*
2, 4 *Folia Psychiat. et Neurol. Jap.*
2 *Fortsch. Neur. Psychiat.*

2, 3 *Genet. Psychol. Monogr.*
2, 4 *Geriat., Minneapolis.*
4 *Giorn. di Psich. e di Neuropat.*

4 *Howard Journal.*
1 *Hum. Factor.*
2 *Hum. Relat., Cambridge, Mass.*
2, 3, 4 *Hyg. Ment.*

4 *Illinois Psychiat. J.*
2, 4 *Index Neurol. y Psiquiat., Buenos Aires.*
Indian J. Neur. Psychiat, Calcutta.
3 *Indian J. Psychol., Calcutta.*
4 *Indiv. Psychol. Bull., Chicago*
3 *Industr. Psychol.*
3 *Industr. Psychotech.*
1, 2, 3, 4 *Int. J. Psychoanal., London.*
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., Washington.*
J. Amer. Soc. Psychic. Res.
3 *J. App. Psychol., Washington.*
2, 4 *J. Belge Neur. Psychiat* (now *Acta Neurol. Psychiat. Belgica*).
4 *J. Bras. Neur., Rio.*
2, 4 *J. Bras. Psiquiat.*
2 *J. Child. Psychiat.*
2 *J. Clin. Psychol., Burlington.*
2, 4 *J. Clin. Psychopath., Montecello, N.Y.*
2, 4 *J. Comp. Neur.*
2, 3 *J. Comp. Physiol. Psychol., Washington.*
2 *J. Consult. Psychol., Washington.*
J. Crim. Law and Criminol., Chicago.
2 *J. Crim. Psychopathol.* [now *J. Clin. Psychopath.*].
2 *J. de Psychiat. Infant* [now *Zeit. f. Kinderpsychiatrie*].

3 *J. Educ. Psychol., Baltimore.*
J. Except. Child.
3, 4 *J. Exp. Psychol., Washington.*
2 *J. f. Psychol. u. Neurol.*
3 *J. Gen. Psychol., Provincetown, Mass.*
2, 3 *J. Genet. Psychol., Provincetown, Mass.*
2, 4 *J. Geront., St. Louis.*
J. Juvenile Res.
1, 2, 3, 4 *J. Ment. Sci., London.*
1, 2, 3, 4 *J. Nerv. Ment. Dis., New York.*
1, 2, 3, 4 *J. Neurol. Neurosurg. Psychiat., London.*
J. Neuropath. and Psychiat., Leningrad.
1, 2, 4 *J. Neuropath. Ex. Neur.*
2, 4 *J. Neurophysiol.*
J. Neuropsychiat. du Pacifique.
2, 4 *J. Neurosurg.*
3 *J. Parapsychol., Durham, D.C.*
2 *J. Personality, Durham, D.C.*
J. of Psychic. Res.
J. Psihoteh.
2 *J. Psycho-Asthen.* [now *Amer. J. Ment. Deficiency*].
1, 2, 3 *J. Psychol.*
J. Psychol., Moscou.
2, 3 *J. Psychol. Neurol., Leipzig.*
2 *J. Psychol. Norm. Path., Paris.*
J. Soc. for Psychic. Res.
3 *J. Soc. Psychol., Provincetown, Mass.*
2, 4 *J. Speech Dis., Danville, Ill.* (now *J. Speech, Hearing*).

Kriminal.
3 *Kwart. Psychol.*

4 *Maan. Geest. Voltis., Amsterdam.*
Mag. psychol. Szle.
Menn. Miljφ.
1, 2, 4 *Ment. Health, London.*
Ment. Health Obs.
1, 2, 4 *Ment. Hyg., N.Y.*
Ment. Hyg. Rev.
Ment. Hyg. Bull., Indiana.
3 *Mind, London.*
4 *Monats. Psychiat. Neurol.*
Monogr. Soc. Res. Ch. Devel.
Mschr. Krim. Biol.
2 *Mschr. Psychiat. Neurol.*

Ned. Tijdschr. Psychol.
2 *Neopsichiat.*
2, 4 *Nervenarzt.*
2, 4 *Nerv. Child, New York.*
3 *Neue psychol. Stud.*
2, 4 *Neurbiol., Pernambuco.*
4 *Neur. a Psychiat., Česká.*
Nevrasse.
2, 4 *Nevropath. i. Psikhiat.*
2, 4 *Note e Riv. di Psichiat.*
Now. Psychjar.
Nuova Riv. di Clin. ed Assist. Psichiat.

Obshch. Klin. Nevropat.
2, 3, 4 *Occup. Psychol., London.*
2, 4 *Occup. Ther. and Rehabil.*
2 *Onder. Psychiat-Neur. Klin., Utrecht.*
2 *Osped. Psichiat.*

Personnel Psychol.
3 *Person. J.*
Pisani.
Polsk. Arch. Psychol.
Prace Psychol.
2 *Proc. Amer. Assoc. Stud. Ment. Def.* [now *Amer. J. Ment. Deficiency*].
2 *Proc. A. Res. Nerv. and Ment. Dis.*
3 *Proc. Soc. Psych. Res., London.*
Prog. Neurol., Psychiat.
3 *Psichotec.*
4 *Psicoanalisi, Rome.*
Psicoter., Cordoba.
Psyche, Cambs., Mass.
Psyche, Heidelberg.
Psyché, Paris.
Psychiat. Monogr.
2 *Psychiat. en Neurol. Bl., Utrecht.*
2 *Psychiat. et Neurol. Jap.*
2 *Psychiat. Neurol. Wchnschr.*
2, 4 *Psychiat., Washington.*
1, 2, 4 *Psychiat. Quart., Utica, N.Y.*
2, 3, 4 *Psychoanal. Quart., Albany, N.Y.*
2, 3 *Psychoanal Rev., New York.*
Psychodrama Monogr.
1, 2, 3 *Psychol. Abstr., Washington.*
2, 3, 4 *Psychol. Bull., Washington.*
Psychol. Clin.
Psychol. Exch.
3 *Psychol. Forsch.*
Psychol. Index.
2 *Psychol. Monogr., Washington.*
Psychol. Rec.
1, 3, 4 *Psychol. Rev., Washington.*
3 *Psychol. Rev. Monogr.*
Psychol. Sborník.
Psychol. Stud. Kbh.
Psychol. Stud. Univ. Bp.
Psychol. wychow.
3 *Psychomet., Chicago.*
Psychometr. Monogr.
2, 4 *Psychosom. Med., New York.*
2, 4 *Psychosom. Med. Monogr., New York.*
Psychotechnick.

Quart. J. Child. Behav., New York.
Quart. J. Ex. Psychol.
4 *Quart. J. Speech.*
2, 4 *Quart. J. Stud. Alc., New Haven, Conn.*
2, 4 *Quart. Rev. Psychiat. Neur., Washington.*

4 *Rass. Neurol. Veget., Firenze.*
2, 4 *Rass. Neuropsichiat. Sci. Aff., Salerno.*
4 *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.
4 *Rev. Espan. de Oto-neuro-oftam. y Neurocir.*
3 *Rev. Franç. Psychoanal.*
Rev. Ibero-Amer. de Anal. Biblio. de Neurol. y Psiquiat.
4 *Rev. Mex. Neurol. Psiquiat. y Med. Legal., Mexico.*
1, 2, 4 *Rev. Neurol.*
2 *Rev. Neurol. de Buenos-Aires.*
Rev. Neurol. Psychiat., Praha.

2, 4 *Rev. Neuro-Psiquiatr., Lima.*
2, 4 *Rev. Oto-Neur.-Oftal. Cir. Neur. Sud-Am.*
4 *Rev. Oto.-neur.-ophtal., Paris.*
4 *Rev. Neuropsiquiatr.*
2, 4 *Rev. Psicol. Gen. Aplic., Madrid.*
Rev. Psicol. Pädag.
Rev. Psicoanal., 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. Psychol., Montreal.
Rev. Sudam. Psicol. Pedag
Rev. Tchèque de Neurol. et de Psychiat.
Ric. Psich.
2, 3, 4 *Riv. di Neurol.*
Riv. di Neuro-psiquiat., Peru.
4 *Rev.-Oto.-neur.-oftal., Bologna.*
3 *Riv. di Psichol.*
Riv. Ital. di Endocrin. e Neurochir.
2, 4 *Riv. Patol. nerv. ment., Siena.*
Riv. Psichol. Norm. Pat., Ferrara.
2, 4 *Riv. Sper. Freniat.*
1, 4 *Rocz. Psychjat.*
2 *Rorschach Res. Exch. J. Prog. Techn., New York.*

Samiksā.
1 *Schizofrenie.*
2, 4 *Schweiz. Arch. Neurol. Psychiat.*
Schweiz. Z. Psychol.
Schweiz. Z. Psychol. Anwend., Bern.
Sistema nerv. Genoa.
Skand. Arch. Psychol.
Sovet. nervropatol., psichiatri, psichoguoguia.
Sovet. Psichoneurol.
Sovet. Psikhotekh.
Speech Monogr.
2, 3 *Stud. Psychol. Psychiat. Cathol. Univ. Amer.*

Tidskr. Psykol. Pedag.
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.

4 *Wein. Z. Nerven., Vienna.*
Wien Z. Phil. Psychol. Pädag.
3 *Z. angew. Psychol.*
Z. Arb. Psychol.
Z. Berufs. des Pflegepers.
Z. Individ. Psychol.
2, 4 *Z. ges. Neurol. Psychiat.*
2, 4 *Z. Kinderpsychiat., Basle.*
3 *Z. Pädag. Psychol.*
Z. Parapsychol.
2 *Z. Psych. Hyg.*
Z. Psychiat. Neurol. Klin. Psychol.
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.

ACT. LUSO-ESPAÑ. NEUR. PSIQUIAT.

VOL. VIII. APRIL, 1949.

ACTA NEUR. PSYCHIAT. BELG.

VOL. XLIX. JANUARY, 1949.

FEBRUARY.

MARCH.

APRIL.

MAY.

JUNE.

JULY.

ACTA PSYCHIAT. NEUROL.

VOL. XXIV. 1949.

Investigations into the Colour-form Reaction of Epileptics.

The tendency to observe the colour rather than the form, or vice versa (colour-form reaction) of objects perceived has been the subject of a considerable number of investigations from the standpoints of genetic psychology and characterology. The influence of various mental disorders on this reaction hitherto has been investigated only in schizophrenics and epileptics—in the latter by means of the Rorschach method.

The present writer has studied the colour-form reactions of 213 epileptics ranging from 6–55 years of age and institutionalized at the State Institution for Epileptics, Vilhelmsro, Jonkoping, Sweden. Since colour-form reaction varies according to differences in intellectual level and constitutional type, both intelligence tests and the determination of constitution were undertaken, in addition to which a survey is given of earlier investigations in this field.

The I.Q. has been determined by means of "A Point Scale for Measuring Mental Stability," by Yerkes, Bridges and Hardwick, translated into Swedish by Wahlen. The mean I.Q. of the entire material was found to be $67{\cdot}1 \pm 1{\cdot}5$.

The determination of constitution was carried out according to Stromgren on the adult epileptics, totalling 116 cases. Mean index $= -0{\cdot}9$, i.e. a pronounced leptosome index. Only three patients showed a pyknic index.

The colour-form reaction was investigated with Lindberg's Ring Test, the resultant initial colour responses being distributed between different intelligence and constitution groups.

Initial colour responses were given by 49·1 per cent. of the female patients and 54·4 per cent. of the male patients (according to Lindberg's evaluation method, 60 and 67 per cent. respectively).

In the under eighteen age groups the material in the different intelligence groups is too scanty to permit of any comparison with Lindberg's material; but on the other hand a comparison is possible between the colour reactions of the adult epileptics (66·9 per cent.) and of Lindberg's surgical case material (21·5 per cent.). The difference amounts to $45{\cdot}4 \pm 2{\cdot}7$ per cent.

The leptosomatic epileptics exhibit a colour reaction $32 \pm 6{\cdot}7$ per cent. higher than Lindberg's leptosomatic mentally-insufficient patients.

The marked tendency towards colour reaction of the epileptics is regarded by the author as a pathoplastic phenomenon, associated with the general " fall in level," as in the case of schizophrenics. (Author's abstr.)

The Duration of Experimental Disturbances in the Cerebrovascular Permeability Due to Circumscribed Gross Damage of the Brain.

Relatively gross damage was produced experimentally by electrocoagulation in a circumscribed region of the brains of rabbits and cats. The purpose in view was to determine the duration of the disorder in the vascular permeability subsequent to such injury. It was found that in the beginning a pronounced disorder of the vascular permeability was demonstrable. Later, during the first day after the injury an increasing stasis arose in the vessels in the centre of the lesion so that finally the vessels contained no circulating blood. On the other hand, in the peripheral area of the damaged zone there were passable vessels which showed evidence of a disorder in their permeability lasting as long as one week subsequent to the damage. The reason why this disorder should last so long is discussed, and it is suggested that this persistence might be due to a toxic lesion ascribable to autolytic processes.

In the present investigation no dural vessels growing into the brain scar were demonstrable, but the existence of such vessels in certain gross cicatrical formations in man is a well-known fact that may be of physiopathological importance not only mechanically but also from the point of view of permeability. If the experimental damage is infected so that abscesses develop, the perifocal disturbance in the vascular permeability will persist probably as long as the abscess lasts. In the authors' experiments the disturbance in the permeability was limited to the thin capsule of the abscess. (Authors' abstr.)

ACTA PSYCHIAT. NEUROL., SUPPT.

1949.

AMER. IM.

VOL. V. 1948.

AM. J. MENT. DEF.

VOL. LIII. 1949.

AM. J. ORTHOPSYCHIAT.

VOL. XIX. JULY, 1949.

AM. J. PSYCHIAT.

VOL. CVI. AUGUST, 1949.

A Statistical Study of Psychoses due to Drugs or other Exogenous Poisons.

1. Psychoses due to drugs or other exogenous poisons represent very few first admissions to hospitals for mental disease in New York State. Such psychoses average only about 0·2 per cent. of all first admissions.

2. Despite the small number of such first admissions, it is evident, nevertheless, that psychoses due to drugs, etc., are more frequent among females than among males.

3. Very few cases of such psychoses arise from poisoning due to a metal or to a gas. The largest simple category is probably that arising from the use of opium or its derivatives.

4. The average age of first admissions with psychoses due to drugs, etc. is approximately 46 years. Females averaged about a year older than males. Approximately 60 per cent. of such admissions are found within ages 35 to 54.

5. There was a high percentage of intemperate users of alcohol among first admissions due to drugs, etc.

6. When classified as to marital status, all first admissions with drug psychoses, etc., except the married group, were in relative excess of the numbers expected when compared with similar marital groups in the general population. The excess was especially marked among those who were separated or divorced.

7. When compared with the general population, first admissions with drug psychoses, etc., did not show any significant differences with respect to degree of education. However, they included higher percentages with a high school or college education than is found among all first admissions.

8. Psychoses due to drugs, etc., occurred more frequently among the urban than among the rural population. With respect to the latter, they were confined to the non-farm group.

9. Negroes had a higher rate of first admissions with drug psychoses, etc., than whites. This was due to a great excess among Negro males. Negro females on the contrary contributed less than their quota of such first admissions.

10. Foreign-born whites had less than their quota of first admissions with psychoses due to drugs, etc. Native whites had an excess of such admissions.

11. There is a suggestion of a relatively high rate of first admissions with psychoses due to drugs, etc., among Jews, in contrast with unusually low rates of first admissions with alcoholic psychoses among them. (Author's abstr.)

Wave and Spike Discharges in the EEG.

The wave and spike formations occur usually on the background of a normal basic cortical rhythm, are remarkably constant in their occurrence in a frequency range of 2·5–3·5 cycles per second, but show considerable variations in the position and amplitude of the spike component in the same record, and indeed within the same discharge. The wave and spike formations are seen most frequently during hyperventilation. Other paroxysmal disturbances occur in 50 per cent. of the records, and the most frequent of these are single or multiple spiking discharges and paroxysmal bursts of 4–6-per-second activity. A surprisingly high incidence of wave and spike formation occurs after the age of 30 years. (Authors' abstr.)

Involutional Melancholia and Convulsive Therapy.

Results of 61 involutional cases discharged from the Institute of Living, 1935–37, prior to convulsive therapy, are compared with those of 347 involutional cases treated with convulsive therapy between 1945 and 1947. The average number of electric treatments for melancholia cases was 10·8; for paranoid cases 16·2; for mixed cases 11·8. In both series of pre-shock and shock eras, the patients benefited from estrogen therapy and the re-educational and vocational programs, the latter with progressively increased facilities.

The average duration of hospital stay was 1½ years in the pre-shock era as compared to 6 months with convulsive therapy.

The combined percentage of patients recovered and improved was essentially the same in both series (92 per cent. in 1935–37 and 90 per cent. in 1945–47). However, in the pre-shock era, 30 per cent. of melancholia cases were considered as recovered as compared to 62·5 per cent. of those receiving convulsive therapy; 62 per cent. were considered as improved as compared to 27·5 per cent. with convulsive therapy. The criteria of recovery not being the same in both series, these latter figures cannot be compared without reservation. However, even allowing for the difference in criteria of what constitutes recovered, there is material evidence to indicate that the shock-treated group left the Institute in better condition than the non-shock group. On the other hand, there is conclusive evidence that the length of stay in the combined recovered and improved group was materially shortened. The non-shock-treated group remained in the Institute on an average of three times as long as the shock-treated group. This difference in the length of hospitalization can hardly be accounted for by the improved and increased ancillary therapies which had undergone considerable development with the shock-treated group. (Author's abstr.)

SEPTEMBER.

Electroencephalographic Effects of Bilateral Prefrontal Lobotomy.

1. A selected group of 25 chronic psychiatric cases who manifested seizures after lobotomy and 46 chronic psychiatric cases who were seizure-free after lobotomy were studied clinically and electroencephalographically.

2. An analysis of the prelobotomy EEGs of the seizure and seizure-free group revealed only slightly increased abnormality in the seizure group (33 per cent. as compared to 24 per cent.), more slow dysrhythmia in the seizure group, and more fast dysrhythmia in the seizure-free group.

3. Considering only the cases who had electric shock treatments before lobotomy, those in the seizure group averaged approximately twice as many treatments as those in the seizure-free group (31 as compared to 15). However, the difference in amount of shock treatments given to the two groups was not reflected in the prelobotomy EEGs.

4. Immediately after lobotomy almost all patients exhibited a drowsy akinetic state accompanied by diffuse slow potentials of increased amplitude. When the patients were aroused by means of various types of stimuli, the slow waves in the posterior leads were temporarily replaced by alpha rhythm, but slow activity in the anterior leads remained. As the akinetic state gradually subsided, the occipital slow waves were replaced by normal alpha rhythm, but the frontal slow activity persisted for a longer period of time.

5. Following lobotomy, there was an extremely high incidence of very slow " rolling activity " which was observed in frontal, precentral, and to a lesser extent in motor and temporal leads.

6. Slow activity from leads about the injured area was at its height within the first two weeks after lobotomy and then began to decline. This decline was well marked in the seizure-free cases, but in the seizure cases slow wave abnormality tended to persist.

7. One month or more after lobotomy the incidence of abnormal EEGs was much higher in seizure cases than in seizure-free cases (76 per cent. as compared to 38 per cent.).

8. One month or more after lobotomy the incidence of focal EEGs was much higher in seizure cases than in seizure-free cases (41 per cent. as compared to 12 per cent.).

9. EEG abnormality in the seizure cases was in part dependent upon the interval between the seizure and the recording of the EEG. When the interval was short (days), the EEG was usually more abnormal than when the interval was long (months or years). However, the majority of EEGs recorded more than one year after a seizure were still abnormal.

10. Clinical analysis of cases with postlobotomy seizures revealed the following :

(*a*) The interval between lobotomy and onset of seizures varied from days to years, with a peak at 3 months to 1 year.

(*b*) During a follow-up period varying from 3 months to 4 years, the majority of patients experienced only 1 or 2 seizures. However, some patients had frequent convulsions, and status epilepticus occurred in 2 cases.

(*c*) In the large majority of cases, attacks were of the *grand mal* type. Two

patients had Jacksonian seizures in addition to *grand mal*, and 2 patients had tonic seizures. No *petit mal* or psychomotor seizures were observed.

11. Postlobotomy seizures did not negate the beneficial effect of lobotomy upon the mental disorder. The clinical follow-up study after lobotomy revealed that the seizure cases were doing as well as, or better than, the seizure-free cases.

12. There was no correlation between EEG findings (either before or after lobotomy) and the beneficial effect of lobotomy upon the mental disorder.

13. There were 4 patients with severe chronic postlobotomy headaches, all of whom had abnormal focal EEGs in addition to postlobotomy seizures.

(Authors' abstr.)

Experimental Observations on the So-Called Senile Changes of Intracellular Neurofibrils.

Various observations reported in the literature suggest that the so-called Alzheimer changes of the intracellular neurofibrils in senile and presenile conditions are associated with dehydration.

In the present experiments rabbits' brains were dehydrated *in vivo* by intravenous administration of 25 per cent. glucose solution. Microscopic examination showed that in some nerve cells, irregularly scattered in the brain, the intracellular argentophile fibres become coarse, and have a tendency to become matted strands. The nucleus remains intact. These changes correspond to the initial stages of Alzheimer's cell change in man as encountered in senile and presenile conditions. These findings are discussed in the light of the literature on this subject.

The histological findings in experimental hydration are also reported.

(Authors' abstr.)

A Comparison of the Test Performances of the Brain-Injured and the Brain-Diseased.

This paper is the fourth in a series dealing with encephalopathy due to brain injury and brain disease. The test results indicate that the functioning and efficiency of the patients in both groups follow a similar pattern: Full-scale I.Q.'s within the normal limits, verbal scores significantly higher than performance ratings, and low inter-group differences for full-scale, verbal and performance scales. The brain-injured patients on the average, showed more marked discrepancies between the verbal and performance subtests. The functions that are impaired or inefficient are discussed. The author holds that the statistical findings do not fully justify the quantitative interpretation that such rigid constructs as mathematical limits would imply. The effects of an encephalopathic process are serious, but an unmeasurable consideration must be given to the impact upon the personality structure of the patient. It is questionable whether any statistical technique exists at present which could factor out this pervasive element satisfactorily.

A comparison of the performances of both groups on the Bellevue Intelligence Scale shows similar function losses in addition to a consistent mean subtest weighted score pattern. For those interested in designing scatter-patterns the procedure suggested previously still holds: Use information weighted score as the basal point for deviation computation of the other 10 subtest weighted scores. The order of deviation of these 10 subtests from information—from most to least deviated—is: Digit symbol, digit span, block design, object assembly, and picture arrangement. (Author's abstr.)

A Psychometric Determination of Alcoholic Addiction.

There is a need for an objective test to identify the alcoholic addict. Such a test undoubtedly would conserve personnel and time within large screening programs. In clinical practice, such a test might be valuable as a therapeutic device.

A 60-question test, called the Alcadd Test, was constructed and used. In the present study, 123 alcoholics were compared with 159 nonalcoholics on the Alcadd Test. The 2 groups were relatively comparable in age, intelligence and socioeconomic status. Illiterates, mental defectives, psychotics and deteriorated individuals were not used.

The following conclusions have been drawn:

1. A paper-and-pencil test, simple to administer, score, and interpret has been found useful in the identification of the alcoholic addict.

2. Highly significant statistical differences in mean scores on this test were found to exist when alcoholics were compared with social drinkers and abstainers. Significant differences also were found when social drinkers and abstainers were compared.

3. The Alcadd Test made approximately 97 per cent. correct predictions of alcoholics and 94 per cent. correct predictions of social drinkers. It predicted 100 per cent. correctly for the abstainers.

4. Using the shorter approximation of the Richardson-Kuder formula, a coefficient of reliability of ·92 was found for the males and ·96 for the females.

5. A subjective analysis of the 60 items on the Alcadd Test revealed 5 characteristics of the alcoholic addict. They were as follows: (*a*) Regularity of drinking, (*b*) preference for drinking over other activities, (*c*) lack of controlled drinking, (*d*) rationalization of drinking and (*e*) excessive emotionality.

6. Male alcoholics appear to be more consistent drinkers and show stronger preferences for drinking than do female alcoholics. Female alcoholics show much less control over their drinking, more rationalizations for their drinking and more emotional immaturity than male alcoholics.

7. Abstainers consistently make lower scores on the 5 alcoholic characteristics than do both alcoholics and social drinkers.

8. The Alcadd Test can be completed in less than 10 minutes and scored in 2 or 3 minutes. A profile of the 5 traits can be immediately constructed.

(Author's abstr.)

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Presenile Sclerosis (Alzheimer's Disease) with Features Resembling Pick's Disease.

Two cases of presenile dementia are presented. In each of these cases the brain manifested pronounced focal atrophies affecting chiefly the temporal lobes, in addition to generalized atrophy of the cerebral cortex. Histologic examination revealed the presence of numerous senile plaques and neurofibrillary changes throughout the cortex. Concomitant with these typical Alzheimer features were focal accumulations of inflated cells limited to the focally atrophic gyri and sparing the precentral, postcentral, transverse, temporal and occipital gyri. These inflated cells as seen in Nissl sections were ballooned cells with clear cytoplasm and an eccentric nucleus. Silver stains disclosed that they were not morphologically uniform but were in various stages of degeneration. Some contained argyrophil inclusions which made them resemble the cells of Pick's disease. Others had cytoplasmic structures which appeared transitional between neurofibrillary degeneration and the argyrophil bodies. Finally, some had a clear, unstained cytoplasm in both Nissl and silver preparations.

These cases were unique in that they were characterized by typical changes of presenile sclerosis concomitant with inflated cells similar in appearance and distribution to those seen in Pick's disease. The presence of intermediary forms linking the fibrillary cytoplasmic changes with the argyrophil suggests that they may be phases of the same degenerative process. Furthermore, the inflated cells as seen in Nissl sections may be morphologically variable, for silver methods revealed that some contain argyrophil bodies and others showed only a clear, unstained cytoplasm.

From these cases and others described in the literature, there is evidence of considerable variability in the presenile degenerations known as presenile atrophy

(Alzheimer's disease) and Pick's disease. There may be conditions which are intermediate between the two diseases. (Author's abstr.)

MAY.

Relation of Changes in Carbohydrate Metabolism to Psychotic States.

A study was made of the possible effect of changes in the mental status on the glucose tolerance (Exton-Rose technic) in 28 patients, (11 men and 17 women). The agent used to produce the psychiatric changes was electric shock therapy. A variety of psychotic types were investigated, the commonest classification being schizophrenia (16 cases).

In the majority of cases the glucose tolerance as a whole was little affected by the degree of improvement shown, but in individual cases there was a notable relation between the emotional disturbances and the response to sugar. This relation was due not to the presence or absence of psychotic manifestations but to the underlying residue of emotional tension. Only those patients who had apparently made some solution of their problems showed a "normalizing" of their glucose tolerance. It is concluded that the decrease in the glucose tolerance which is commonly seen in psychotic patients is primarily due to the anxiety underlying the mental disturbance. (Authors' abstr.)

JUNE.

The Superiority Attitude and Rigidity of Ideas.

The superiority complex is described as a more or less completely developed trend toward a secure and gratifying, even exalted, self-estimation. A certain uniformity of personality traits is detectable, and an attempt is made to delineate them as a type. Any disruption of the superiority feeling is exceedingly painful, and the possessor, therefore, is distinguished by his inability to adapt himself to discordant realities. As a protection against these realities, he is totally committed to a fixity of views that will afford absolute security. These rigid formulations pervade the entire mental life. In the effort to eliminate uncertainties, a cosmology of cause and effect relations approaching the metaphysical may be constructed, while efforts to control events may show magic components.

Psychoneurotic phenomena appear when the superiority system has been breached by the impact of the circumstances. The simplest is the anxiety-depressive reaction, indicating the direct reaction to the imminence of psychic pain. More spectacular, however, are the obsessions and compulsions, in which protection is achieved by one's accepting the offending agent into consciousness only in the form of a symbol, thus permitting its vicarious destruction.

It is suggested that involutional depression arises from external trauma to some phase of the security organization, with complete failure of all protective mechanisms. An explanation is offered for the rapid spread of the depression to involve every aspect of the personality. (Author's abstr.)

VOL. LXII. JULY, 1949.

Analysis of a Prefrontal Lobe Syndrome and its Theoretic Implications.

The study reported has emphasized the importance of a severe limitation in associative range as a determinant of the behavioral disturbance after damages of the prefrontal regions. The hypothesis is offered, on the evidence from this case study, that symbolic activity is affected after damage to the prefrontal lobe because of the diminution in ability to shift from one associational trend to another. It is considered possible that the limitation of associations and the ensuing degradation of behaviour are dependent on the failure of symbols as links with or pivots for related experiences. Although it has been clear that lesions producing aphasia disrupt symbolic activity at more primary level, the limitation of associative range and of the use of symbols following lesions of the prefrontal regions has not been so apparent. Such limitations may not be noted readily both because of the diversity and breadth of human facilities, and because the limitations are masked by knowledge and behavioral patterns acquired prior to damage to the brain. In some cases, as in F.'s, the intellectual defect is obvious. In other cases in which the impairment is slight, little may be noted other than some degree of short-sightedness—an absence of consideration of the total circumstances and prior experience. As stated by Freeman, the thinking is finished more quickly and with greater satisfaction and assurance than in normal persons. In all cases of dysfunction due to lesions of the prefrontal lobes it may be that symbols, although readily available, are deprived of their full associative value, and that this limitation may affect the ability to utilize other assets. (Author's abstr.)

Combined Lateral and Ventral Pyramidotomy in Treatment of Paralysis Agitans.

1. A new surgical technique for the treatment of paralysis agitans is presented. In this procedure, combined lateral and ventral pyramidotomy, the lateral pyramidal and the opposite ventral pyramidal tract are sectioned through a single incision in the cord.

2. Use of this procedure in the treatment not only of tremor, but of rigidity and poverty of movement as well, is demonstrated in a series of 11 cases.

3. One case is presented in which combined lateral and ventral pyramidotomy was performed bilaterally, with good result.

4. Application of this procedure to the treatment of the major symptoms of paralysis agitans affecting one side or both sides is discussed. (Author's abstr.)

Plasma Calcium Fractions after Electric Convulsion Treatment.

Plasma calcium levels determined immediately before and immediately after an electrically induced *grand mal* convulsion showed an increase after the convulsion in each case, the average increase being 15·2 per cent. The initial increase in the total calcium of the plasma immediately after the convulsion was followed by a gradual decrease, which varied with the patient, and the preconvulsion calcium level was reached in all cases within 90 minutes after the convulsion.

The aforementioned increase in total plasma calcium after the convulsion may be attributed to an increase in the non-ultrafiltrable calcium fraction (largely protein-bound calcium); the ultrafiltrable calcium fraction remained essentially the same before and after the convulsion. Plasma protein levels were also increased immediately after an electric convulsion treatment, and the electrophoretic patterns of the plasma of one patient under the same conditions indicated that within the limits of experimental error, all fractions of protein were increased proportionally.

Hematocrit determinations on the blood of 5 patients showed an increase in the ratio of red blood cells to plasma in each case. An average decrease of 10·9 per cent. in circulating plasma volume was found.

It seems that hemoconcentration occurs after an electrically induced *grand mal* convulsion, thus accounting for the reported increase in several constituents of the plasma. However, some other mechanism may operate to account for the fact that the plasma calcium fractions do not increase proportionally after the convulsion.

(Authors' abstr.)

AUGUST.

Effects of Ultraviolet Radiation on the Exposed Brain.

The recent introduction of ultraviolet radiation for antibacterial purposes has led to its use on the exposed human brain during neurosurgical procedures. Since the need for antisepsis is more important in the longer neurosurgical procedures, the necessity of knowing the prolonged effect of radiation on the exposed animal brain seems to be of immediate importance.

Ten cats were subjected to bilateral craniotomies. Ultraviolet radiations, having 80 per cent. of their wavelengths in the vicinity of 2,537 angstrom units, were allowed to project on to one hemisphere for five hours, while the other hemisphere, used as a control, was shielded from the radiations. Pathologic studies were made after the animals were killed, on the third, fourth or fifth post-operative day.

Exposure of the cat brain to ultraviolet radiation under actual human operating conditions produced dilation of meningeal and cortical vessels, usually within 30 to 60 minutes. Gross congestion subsided in 20 minutes by elimination of the ultraviolet radiation. Re-irradiation, however, provoked a rapid redilation.

Petechial hemorrhages were observed on the irradiated side in all animals at autopsy, on the third or fifth post-operative day. In one animal cortical petechiae were already visible towards the end of the five-hour exposure.

On inspection of the exposed cortex slight edema was suspected in only a few instances on the irradiated side. It was difficult to evaluate and recognize during exposure. However, in all the cases gross edema was observed at autopsy and was greater on the irradiated side.

Cortical electrograms were taken throughout the exposure in 4 animals. In 3 experiments depression of activity occurred after an exposure of one hour. In one animal epileptiform spikes developed after one hour, and this animal had a clinical seizure at the end of five hours. The electrographic alterations supervened during the stage of vascular congestion.

An intense inflammatory response was observed in the irradiated area. This consisted of vascular congestion of meningeal and cortical vessels; transudation of serum; cellular exudate, with cuffing of vessels; diapedesis, and numerous petechial hemorrhages.

Within the irradiated hemisphere ganglion cells were reduced in number, showed swelling and disruption of cell membranes and occurred as pyknotic forms in the more superficial layers.

Fibrous astrocytes showed evidence of mild hyperplasia of their projections on the irradiated side. The oligodendroglia remained normal, except about cysts and hemorrhages, where acute swelling occurred. The microglia was reactive, especially in the plexiform layer over the irradiated hemisphere.

It is concluded that prolonged ultraviolet irradiation of the brain is harmful and that further investigation is necessary to establish how long the brain may be irradiated without showing irreversible pathologic effects. Physiologic and electrographic observations during ultraviolet irradiation of the exposed brain suggest that with frequent irrigation of the brain, exposure of 30 to 45 minutes will not result in permanent harmful effects. (Authors' abstr.)

Electroencephalographic Changes after Prefrontal Lobotomy, with Particular Reference to the Effect of Lobotomy on Sleep Spindles.

1. Patients with lobotomy have generalized slowing, most prominent in the frontal leads, in the first few post-operative days. Slowing decreases in degree and extent slowly in the group, abruptly in individuals, and there is little change after three months.

2. The commonest long term electroencephalographic residuum of lobotomy consists in ½ to 1 per second base line sway with normal frequencies superimposed.

3. Runs of 12 per second waves in sleep are most prominent in anterior leads before operation and virtually do not occur after lobotomy. It is assumed that they originate in the nucleus centralis medialis of the thalamus and that their absence in all leads after operation is due to degeneration of this nucleus.

4. Runs of 14 to 15 per second waves in sleep are most prominent in central leads before lobotomy, are more frequent in central leads and are less frequent in frontal leads after than before lobotomy. It is assumed that they originate in the nucleus centralis lateralis of the thalamus. When they are still present anterior to the lobotomy incision, it must be assumed that severance of fiber tracts between the cortex and thalamus is incomplete. When they are absent in frontal leads and present in central leads after lobotomy, it must be assumed that thalamofrontal fibers are completely severed (unless orbital fibers have been spared).

5. Persistence of normal alpha activity in the frontal leads of patients whose sleep records demonstrate complete severance of corticothalamic fibers casts serious doubt on the theory that alpha activity is maintained solely by thalamocorticothalamic reverberating circuits. (Authors' abstr.)

The Phobic Syndrome: A Study of Eighty-Six Patients with Phobic Reactions.

This report on 86 patients with phobic reactions indicates that the phobic person is fundamentally apprehensive, with an overactive sympathetic nervous system and a cyclothymic make-up. He is usually too closely attached to one parent and has not entered into mature adult relationships with other persons, even though he may have married and become a parent. Having been brought up "soft" he reacts to the dangers of everyday living by acquiring protective phobic reactions. His fears are merely symptoms of an underlying neurotic state; therefore the treatment of an adult with phobias consists in emotional retraining and reconditioning of specific fears through psychologic re-education or brief psychotherapy. In order that he may reap the rewards to which his exceptionally good personality entitles him, a potentially valuable but immature person must be assisted to grow up, to assume an adult's place in society. To bring this about, he must be taught to accept no orders from unreasonable fears.

Psychologic re-education requires approximately two months of intensive treatment which should be followed by several months of medical supervision to make sure that the patient does not regress into phobic or other neurotic reactions. This follow-up treatment is important, since phobic patients have a tendency to mislead the physician in an attempt to escape further treatment by making the doctor think that they are more nearly well than they actually are. The physician must be sufficiently experienced not to be deceived by such a person, who is frequently a master in deception. As long as any phobia is active, the patient is not cured. (Author's abstr.)

Studies on the Occipital Lobe. (1) *Significance of Small Areas of Preserved Central Vision.*

Six cases of occipital lobectomy are presented in detail, and their results with regard to preservation of central vision are summarized.

The various theories advanced to explain macular sparing are discussed. Previous series of occipital lobectomy are reviewed with reference to the present status of the doctrine of cortical localization of the macula.

It is concluded that there is no evidence to support a theory of bilateral macular or foveal representation.

Preservation of central vision ranging from 0·25 to 1·5 degrees was observed in the authors' patients with occipital lobectomy. However, the same degree of macular sparing was found in eight control patients with chiasmal lesions. It was felt that the situation and extent of the lesion in the optic pathways, as well as a minimal eccentricity of the fovea with slight physiologic variation in fixation, explain the results most satisfactorily. (Authors' abstr.)

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Tridione Therapy in Minor Epilepsy.

Tridione has been satisfactory in the treatment of minor epilepsy in 38 out of a series of 45 cases. In 12 the attacks were abolished and in another 15 almost complete control was achieved. In 13 further cases there was a significant reduction in the incidence of attacks.

Of the remaining five cases three showed slight improvement. Two failed to respond.

Five patients with psychomotor epilepsy were made worse by tridione.

Minor side-effects—photophobia, mild sore throat, a few papules on the face, chest or arms—were not uncommon. In two patients there was an urticarial reaction which subsequently developed into generalized exfoliative dermatitis necessitating discontinuation of tridione. In a third case treatment was temporarily discontinued following the appearance of facial erythema and oedema. At a later date tridione was successfully used in these three cases without harmful effects.

Apart from transient eosinophilia in four patients the authors observed no significant alteration in the leucocyte counts.

The authors conclude that tridione is the most effective substance as yet available for the treatment of minor epilepsy. Although serious toxic reactions sometimes occur, they are not common and in hospital out-patient practice tridione is the drug of choice in the treatment of minor epilepsy. (Authors' abstr.)

The Orbital Gyri.

(1) The functions and connections of the orbital gyrus have been studied in a series of cats, dogs and monkeys.

(2) Electrical stimulation of the orbital gyrus affected blood pressure and respiration, the effects varying with the anaesthesia, the frequency of the stimulus, and the wave form of individual current pulses.

(3) Blood pressure and respiratory effects were obtained independently.

(4) The excitable area for these various autonomic responses corresponds to Area 13 of Walker, or " FF " of von Bonin and Bailey, and within this area there are discrete foci of maximal sensitivity.

(5) Stimulation of the anterior perforated substance is usually followed by a rise in blood pressure and respiratory arrest, as well as pupillary dilatation.

(6) Varying the frequency or the stimulus in the orbital gyrus and in the anterior perforated space may reverse the blood pressure reaction.

(7) Dilatation of the pupil eye movements, salivation, lacrimation and coarse motor movements were obtained by stimulation of the orbital gyrus and the anterior perforated space.

(8) The blood pressure and respiratory responses could be obtained after section of both vagi, the splanchnic nerves, and after removal of the adrenals, and destruction of both paraventricular nuclei of the hypothalamus, and after paralysing doses of tetra-ethyl-ammonium chloride.

(9) No suppression of somatic movements or of ECG could be obtained.

(10) Strychnine neuronography demonstrated connections to both homolateral and contralateral putamen, contralateral caudate nuclei, corpus callosum, H Field of Forel, median forebrain bundle, and lateral preoptic area, as well as the contralateral orbital gyrus. (Authors' abstr.)

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VOL. III. 1949.

CERVELLO.

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SEPTEMBER.

CRIANÇA PORT.

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Rhythmic Electrical Activity from Isolated Cerebral Cortex.

Bremer has demonstrated that rhythmic activity of the cerebral cortex, similar to the alpha rhythm, represents an inherent property of the cortex itself. In spite of Bremer's findings there has been considerable disagreement as to whether the rhythmic cortical activity depends upon the interaction of the cortex and subcortical structures, or whether the cortex is able to produce spontaneous rhythms.

Spontaneous and induced cortical activity has been investigated in cats in intact hemispheres, in hemispheres after thalamectomy, and in portions of the cortex which have been completely isolated from the rest of the brain.

Both after thalamectomy and after severance of all subcortical and intracortical connections to a small portion of the cortex, spontaneous rhythmic cortical activity was recorded, resembling the normal alpha rhythm.

Electrical activity induced by electrical or chemical stimulation in such preparations was indistinguishable from activity induced by the same means in normal hemispheres.

The results of this study give support to Bremer's views on the origin of the rhythmic cortical activity, and have led to the following conclusions:

1. Rhythmic electrical activity, closely related to the alpha rhythm, is an inherent property of the cortex itself. It is not necessarily dependent upon thalamo-cortical circuits, though these circuits, when present, may modify its form and amplitude.

2. The explanation of contradictory results obtained by some authors may be due to conditions of the cortex following operative procedures. The facility with which the effect of such procedures can be reversed by chemical stimulants like eserine and acetylcholine should be emphasized.

3. Rhythmic electrical activity in response to electrical or chemical stimulation of the cortex has the same characteristics in an isolated portion of the cortex as in normal cortex with intact connections, under the conditions of the authors' experiments. (Authors' abstr.)

The Electroencephalogram following Occipital Lobectomy.

The electroencephalograms of six patients whose entire left occipital lobe has been removed at operation have been reported.

Three abnormalities have been observed: (*a*) Diminished alpha activity in the left occipital area; (*b*) the occurrence of sharp waves and fast beta activity in the temporal, central and frontal areas; (*c*) over-activity of the left temporal lobe with the presence of abnormal waves, increased alpha-like waves and large voltage sleep waves. It is suggested that some of the increase in activity is probably the result of scar tissue. Although it is possible that some of the remaining irregularity might be the result of anatomical changes resulting from operation, it is suggested that there is some evidence that the increased activity represents a release phenomenon; (*d*) it is concluded that following complete removal of the striate area, the remaining portion of the hemisphere, especially the temporal lobe, is capable of generating an alpha-like rhythm. (Authors' abstr.)

Electrographic Study of the Convulsant Action of Intravenously Administered Acetylcholine.

1. Intravenous administration of large doses of acetylcholine induces convulsive respiratory movements, occasionally followed by tonic spasm of the limbs and tremor. In only one case a convulsive seizure clinically similar to a strychnine tetanus was observed.

2. Injections of convulsant doses of the drug induce arrest of the heart, which is followed by flattening of the cortical potentials and by spinal hyperactivity.

3. The changes in the electrical activity of the central nervous system evoked by intravenously administered acetylcholine are similar to those induced by asphyxiation.

4. It is concluded that the convulsive movements resulting from intravenous administration of acetylcholine are not of cortical origin, and that the hyperactivity of the spinal cord is probably a consequence of the deficient oxygenation which follows the circulatory changes induced by the drug. (Authors' abstr.)

Influence of Ammonium Chloride on the Electrical Activity of the Brain and Spinal Cord.

1. Intravenous injections of suitable doses of ammonium chloride in lightly anaesthetized dogs evokes various circulatory effects which may be only slight or sufficient to stop the heart altogether with conspicuous drop of the general blood pressure.

2. The occasional changes in the electrical activity of the cerebral cortex following the administration of the drug are probably a consequence of these circulatory changes.

3. Intravenous as well as local administration of ammonium chloride induces modifications of the spinal cord activity, which are probably independent of circulatory changes and consist of a simple activation or of an activation accompanied by synchronization of the spinal rhythms.

4. The activation accompanied by synchronization induced in the spinal cord by ammonium chloride resembles in many respects the spinal activation following strychnine administration, and possibly is due to increase in the number of active neurones and in their degree of synchronization.

5. Ammonium chloride produces a marked potentiation of strychnine action on the brain and spinal cord.

6. It is concluded that ammonium chloride fits are not of the epileptic type. (Authors' abstr.)

The Electroencephalogram in Cerebellar Seizures.

1. The EEG recorded in a series of cats undergoing cerebellar seizures was not specific in type.

2. It was unlike the EEG accompanying clonic seizures elicited by cerebral stimulation.

3. Involvement of a specific limb in the slow march of the seizure was not accompanied by specific alteration of the EEG from either side of the brain.

4. The EEG during a cerebellar seizure was best described as an activation pattern, being made of low amplitude fast waves.

5. When tracings from the cerebellum were recorded, no alterations were observed during a seizure, but the type of apparatus used did not allow complete analysis of the waves out of the range of the ordinary frequencies of the EEG. (Authors' abstr.)

Effects of Thalamic Stimulation in Unanaesthetized Animals.

A cinematographic analysis of the responses of cats to local stimulation of various portions of the thalamus and hypothalamus has been varried out with histological control and with electroencephalography correlation both during operation and post-operatively in an attempt to investigate further the hypothesis of a diencephalic origin for *petit mal* epilepsy. The results of this investigation may be summarized as follows :

(1) Electrical stimulation within the intralaminar nuclei of the thalamus, especially rostrally and bordering upon the dorso-medial and antero-medial nuclei,

produced a sudden arrest of responsiveness which the authors have called the "arrest reaction." During this arrest the animal would remain immobile, with no loss of tone, and failed to respond to usually effective stimuli.

(2) The "arrest reaction" would persist beyond the period of thalamic stimulation in the form of an after-discharge resembling very closely a *petit mal* type of seizure in man, including the slight twitching movements about the eyes and face so commonly seen in these attacks, and would cease abruptly.

(3) More intense and more rapid stimulation of the same regions producing the "arrest reaction" would produce first a *petit mal*-like attack followed by a major generalized convulsion or "*grand mal*" seizure.

(4) Electrocorticograms showed that stimulation of the intralaminar regions of the thalamus which produced a generalized recruiting type of control of the rhythmic activity of the cortex also produced the generalized changes in behaviour which the authors have termed the "arrest reaction." During the *petit mal*-like seizures a bilaterally synchronous 3 per second wave and spike discharge was recorded from the cortex and was associated with a similar type of wave form in the thalamus. During the *grand mal*-like attacks the electrocorticogram and electrothalamogram showed rapid high-voltage activity simulating closely the form of the EEG at the onset of such attacks in man.

(5) Autonomic system responses including a complex defensive pattern of behaviour with sham rage or a fear pattern associated with flight were reproduced particularly from the hypothalamus and occasionally from the overlying midline nuclei of the thalamus. These results corresponded closely with the detailed analysis of such responses as reported by Hess.

(6) Extrapyramidal types of movement such as tonic postural states, turning of the head and body, circling movements, alternating movements of a limb, and so forth were reproduced from what was presumed to be the thalamic or subthalamic extrapyramidal system. These results also correspond closely to those described by Hess.

(7) Control studies in which stimulation of sensory relay nuclei was carried out produced no such reactions but only responses which gave one the impression of a paraesthesia or of a reaction to a sensory stimulus. On no occasion was any loss in general responsiveness or change in emotional behaviour observed when stimulating sensory relay nuclei.

(8) It is concluded from observations in the cat that the diffuse thalamo-cortical projection system which the authors have called the *thalamic reticular system* has a very widespread and profound effect on behaviour as a whole and may be involved in the mechanism of *petit mal* and generalized convulsive seizures as seen in man.

(Authors' abstr.)

Changes in the Electroencephalogram following Administration of Mesantoin (Methyl-phenyl-ethyl Hydantoin).

Electroencephalograms were obtained before, during and after administration of mesantoin (methyl-phenyl-ethyl hydantoin).

Reversible changes were noted as follows:

	Percentage.
Decrease in alpha rhythm	58
,, slow activity	38
Increase in fast activity	62
No change	12

The dose of the drug and duration of administration seemed to have the closest correlation with these changes. There was little or no correlation between the changes noted and the age, sex or race of the patient, or with the duration, severity, type or degree of control of the convulsive disorder.

The changes observed necessitate recognition that when mesantoin is being administered progressive clinical improvement may be associated with increasing abnormality in the electroencephalogram. (Author's abstr.)

Continuous Measurement of Alveolar CO_2 Tension during the Hyperventilation Test in Routine Electroencephalography.

1. A method is described for automatic and continuous measurement of the alveolar carbon dioxide during the hyperventilation test in routine electroencephalography. It depends upon the specific absorption of infra-red by CO_2, as measured

by an infra-red analyzer (K. Luft) which transforms changes in absorption into deflections of a membrane-condenser. The CO_2 concentration is recorded through the EEG amplifier on the ink-writer tape, and it appears as an alternating voltage of 7 cycles per second, the amplitude of each cycle representing the instantaneous CO_2 concentration in the analyzer.

2. Using this method, 98 healthy flying cadets were tested during a routine three-minute hyperventilation. There was a wide distribution of hypocapnic levels reached, covering the range of 10–28 mm. of Hg. The median of CO_2 tensions after one minute was 22·5 mm., after two minutes it was 20·5 mm., and after three minutes, 18·5 mm. Hypocapnia persisted in each case for more than two minutes after " normal breathing " was demanded.

3. The first appearance of slow wave activity in the EEG occurred over a wide range of alveolar CO_2 tensions. Whereas a number of cases went as low as 10–15 mm. without exhibiting slow waves, others showed a response as high as 25–28 mm. Hg. when hyperglycemia was not assured. With a selected group of five subjects it was demonstrated that raising the blood sugar above 130 mgm. per cent. necessitated a lower CO_2 tension for the elicitation of a given slow wave response, as compared to the fasting state. The progress through the different degrees of slow wave build-up was accompanied by only slight changes in alveolar CO_2 tension. In half of the cases the transition from 5–8 per second activity to 2–4 per second activity occurred within a range of $\pm$ 1 mm.

4. The disappearance of slow wave activity after termination of hyperventilation generally occurred at a higher CO_2 level than that of its appearance during hyperventilation. In the mean, 2–4 per second waves disappeared at a tension 2·3 mm. higher and 5–8 per second waves required an increment of 3·2 mm. Persistence of slow waves beyond two minutes post-hyperventilation indicated that there was either a facile EEG response at a relatively high CO_2 level or an inadequate recuperation from a low CO_2 level. (Authors' abstr.)

Path of Current Distribution in Brain during Electroconvulsive Therapy.

When the minimal electric current necessary to produce a clinical *grand mal* convulsion is administered to the head of a corpse, the current does not travel in a spindle or " onion "-shaped pattern between electrodes, but travels principally along neuronal paths, concentrating in such structures as the corpus callosum and traversing both sides of the head even when one electrode is applied to the vertex and the other to the parietal region. The extent to which it invades structures relatively deep in the brain stem may be of clinical significance.

(Authors' abstr.)

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Liver Function and Nutrition in Relation to Toxic Psychoses.

(1) Toxic psychoses on a nutritional basis may occur without any physical signs of vitamin deficiency.

(2) If nicotinamide therapy is started immediately after the onset of psychosis and adequate diet is restored, the response may be very rapid and dramatic.

(3) In cases of longer duration, response to nicotinamide may be slow or even not occur until the adequate and normal liver metabolism is restored and recovery may occur only after three or four months' treatment.

(4) Several patients recovered on general measures aimed at restoration of liver function without massive doses of nicotinamide.

(5) All of our severe cases of long standing showed an accompanying liver insufficiency and it was only after the improvement in the liver function that the patient's mental functions were restored to normal.

(6) In depressed patients with an accompanying nutritional deficiency, electric shock treatment is of no value until after the nutritional deficiency has been adequately corrected.

(7) Heavy insulin shock is definitely contraindicated in toxic psychoses of nutritional origin.

(8) Vitamins alone have no value in restoring severe cases to normalcy.

(9) We feel that the term " toxic psychosis " is misleading and a more descriptive term, " nutritional psychosis " would be more appropriate for psychosis accompanying deficiency diseases. (Author's abstr.)

Brief Stimulus Electric Shock Therapy.

(1) Brief stimulus therapy is electroconvulsive therapy by means of current so modified that the average electrical energy necessary for production of the seizure is very much less than that necessary with classic sine wave currents.

(2) The clinical improvement obtained by use of BST is at least as good as that produced by the classic type of current.

(3) The chief advantage of the use of BST is the marked diminution and, at times, complete absence of confusion associated with electric shock therapy.

(4) The disadvantage of the treatment with BST is the increase of fear engendered in the patient; this, however, can be readily controlled by the use of pentothal sodium before the treatment. (Author's abstr.)

AUGUST.

NEW YORK NEUROLOGICAL SOCIETY AND THE NEW YORK ACADEMY OF MEDICINE, SECTION OF NEUROLOGY AND PSYCHIATRY.

SEPTEMBER.

NEW YORK ACADEMY OF MEDICINE, SECTION OF NEUROLOGY AND PSYCHIATRY AND THE NEW YORK NEUROLOGICAL SOCIETY.

J. NEUR. NEUROSURG. PSYCHIAT.

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Experimental Radio-Necrosis of the Brain in Rabbits.

A single X-ray dose of 2,850 *r*, under the physical conditions that have been defined, will produce delayed irradiation-necrosis in the rabbit's brain after a latent period of approximately one hundred days.

The latent period is lengthened by reducing the dose to 2,440 *r*.

The initial histological changes consist of minute foci of haemorrhage and necrosis which are intimately related to the perforating vessels and, in particular, capillaries. Increase of capillary permeability accompanied the established lesion but was not demonstrated at earlier stages.

The lesions when once initiated, appear to extend rapidly and to progress to non-irradiated parts of the brain. They are characterized, in later stages, by pronounced gliosis and by progressive degeneration and sclerosis of the adjacent vessels.

The neurones are only focally and secondarily affected.

The meninges and their blood vessels are unaffected and the superficial parts of the cerebrum escape although the cerebellar cortex may be extensively damaged.

(Authors' abstr.)

Clinical and Pathological Observations on Relapse after Successful Leucotomy.

Six patients are described in whom mental illness recurred some considerable time after apparently successful leucotomy and in whom the precise extent of the brain lesions was subsequently determined in the laboratory. Anatomical features of the six cases are discussed, together with those of other " psychosurgical " cases in the literature in which the extent of the lesion was determinable during life. Clinical features of the six cases are reviewed together with those of other relapsed cases in the literature of " psychosurgery " in which diagnostic details are available. The following conclusions are come to :

1. Many of the symptoms and syndromes characteristic of affective and schizophrenic functional psychoses can recur after practically complete isolation of the prefrontal cortex from its fibre connections. Some chronic schizophrenic symptoms, at least, can not only persist but can disappear and then recur after full bilateral prefrontal lobectomy. Many neurotic symptoms can recur after disappearance following removal of more limited amounts of prefrontal cortex.

2. The illness which recurs is practically always of the same type as the preoperative one.

3. There are indications that the power of environmental influences for both good and ill may be augmented after the operation.

4. Case 1 also touches on the problem of localization in the prefrontal regions in relation to type of personality change and to creative ability.

5. The facts demand a generous conception of the degree of plasticity within both the abnormally and the normally functioning brain. (Authors' abstr.)

Observations on the Wave and Spike Complex in the Electroencephalogram.

The EEGs of 100 cases showing typical wave and spike complexes were examined. Seventy-five of the patients were aged from 5 to 20 years inclusive.

Normal alpha rhythm was seen in 88 cases. Apart from the wave and spike complexes, 58 records were otherwise normal. Abnormal rhythms seen are described.

In 80 cases the wave and spike complexes occurred without overbreathing, but in 20 cases they appeared only when the patient overbreathed.

Overbreathing was done by 82 of the patients, and 74 of these developed wave and spike complexes in relation to the overbreathing.

The effect of opening and closing the eyes was observed in all the 100 cases. In 8 cases wave and spike complexes followed opening, and in 15 they followed closing of the eyes. In 4 of these cases there was a response both to opening and closing movements. In 3 cases opening of the eyes was seen to inhibit an existing wave and spike outburst.

The average number of wave and spike outbursts in a record before overbreathing was between 3 and 4.

The wave and spike outburst might last for less than a second or be almost continuous for several minutes. The total average duration of a single outburst per record was 8 seconds.

The length of individual outbursts in a single record showed much variation, but in some cases there was a striking constancy of duration of the outbursts.

In 94 cases the wave and spike complexes were entirely generalized. In 3 cases localized as well as generalized outbursts were seen. In 3 cases the complexes were localized to the frontal regions only. No special clinical features distinguished the few cases in which localized outbursts occurred.

In most cases the wave and spike outbursts appeared abruptly and synchronously in all leads. The complexes themselves might be preceded and followed by a short burst of irregular waves, and there was then a tendency for the wave and spike complex to assume its characteristic form more rapidly and more clearly in the fronto-parietal regions. The wave and spike complexes tended to persist longest in the area in which they were first seen. In about 10 per cent. of cases the outburst was predominantly fronto-parietal, and in 5 per cent. or less it was predominantly occipital.

During long outbursts the wave and spike complex tended to decrease slightly in frequency.

Twenty-nine patients showed obvious clinical attacks of *petit mal* during the recording. All clinical attacks were associated with wave and spike outbursts,

and these were usually of longer duration than the average. The liability to clinical attacks seemed to be rather more closely related to the duration than to the number of outbursts in the record, but there was no constant relationship between the severity of the clinical symptoms and the extent of the EEG changes.

So-called " pyknolepsy " could not be sharply distinguished from *petit mal* by the EEG any more than it could by clinical standards.

The clinical response of cases of *petit mal* to treatment with " tridione " was not necessarily associated with parallel changes in the EEG.

A random 20 minutes EEG record is of limited value in assessing the frequency of attacks, prognosis or progress of a patient with *petit mal*. It may, however, have great diagnostic value in doubtful cases. (Authors' abstr.)

Psychiatric Changes Associated with Friedreich's Ataxia.

1. Some cases of mental disturbances in association with Friedreich's ataxia have been described. These comprise the rare form of great severity, characterized by paranoid feelings and outbursts of excitement, in addition to depressive and confusional states.

2. The association with epilepsy is discussed, and abnormal electroencephalogram findings are recorded. It is suggested that these may help to account for some features of the symptomatology in certain of these cases. (Author's abstr.)

J. NEUROPATH. EXPER. NEUR.

VOL. VIII. JULY, 1949.

Pick's Disease.

1. Seven cases of lobar sclerosis are reported. They fall into anatomic groups :

(*a*) In one form there is marked focal cortical devastation with loss of nerve cells and axis cylinders. Remaining nerve cells are swollen and may exhibit eccentric nuclei and argyrophilic cytophilic inclusions. There is astrocytosis of the cortex, and isomorphous fibrillary gliosis of the white matter. Demyelination and reactive gliosis parallel each other in severity. Damage to subcortical centers is not prominent. This type generally affects polar regions, either of the frontal or temporal lobes.

(*b*) In the other group there is widespread gliosis, cellular and fibrillary, of the subcortex, out of proportion to demyelination of these areas. Cortical damage is less prominent ; there are nonspecific alterations to the nerve cells. Subcortical centers are frequently involved, including degeneration of descending motor pathways. The atrophy is generally not polar in distribution. Disturbances in motor and sensory spheres are observed rather than in brain structures associated with " higher " mental processes.

2. The literature is reviewed, and reported cases classified according to the above grouping, where this is possible from the original author's description.

3. It is suggested that these two types of circumscribed cerebral atrophy have a different etiology. (Author's abstr.)

The Central Nervous System in Hepatic Disease.

1. The central nervous system in 8 out of 18 cases of severe liver disease revealed extensive alterations.

2. The changes involved all regions of the brain and consisted of nerve cell damage and widespread areas of demyelination.

3. The tissue alterations were predominantly perivascular in nature, suggesting a toxic etiology.

4. From a review of the literature and a study of these cases it is felt that the liver damage enables some endogenous toxin to reach the brain and produce the widespread damage.

5. Two cases are reported in detail. (Author's abstr.)

Pathologic Changes in the Central Nervous System Resulting from Experimentally Produced Hyperpyrexia.

The brains of animals dying shortly after subjection to hyperthermia demonstrated hyperemia, edema, and an acute disease process of the parenchyma without signs of beginning repair.

The brains of the animal living for several months after subjection to hyperthermia demonstrated evidence of parenchymatous damage undergoing repair. Clinically, this group of animals showed no disturbance in motor function or behaviour. They appeared and behaved like normal animals.

The application of a frozen carbon dioxide temperature collar about the neck, in the manner described, did not lower the temperature of the head or prevent heat stroke. (Authors' abstr.)

J. NEUROPHYSIOL.

VOL. XII. JULY, 1949.

Effect of Curare on Cortical Responses Evoked by Afferent Stimulation.

1. Intocostrin and d-tubocurarine chloride are each able to block synaptic conduction in the central nervous system.

2. After conduction over sensory pathways is blocked, impulses are still propagated retrograde over the corticospinal tract.

3. Spontaneous cortical electrical activity does not persist after synaptic conduction in the central nervous system has been blocked.

4. Depression of synaptic conduction always requires doses greater than those required for blocking neuromuscular conduction. In cats with intermittent administration of small doses intravenously, a per kilo. dose of 10 to 15 times the usual clinical dose was effective in eliminating all cortical response to stimuli conducted over sensory pathways. Occasionally, much smaller doses sufficed. (Authors' abstr.)

Unipolar Electromyograms of Normal and Denervated Human Muscle.

The form of normal human muscle action potentials obtained with a unipolar micro-needle electrode may be described as follows:

1. A negative spike of 2–3 msec. duration preceded and followed by a positive potential of variable size, form and duration, depending upon the position of the needle point relative to the conducted spike process in the muscle.

2. Prolongation, distortion and attenuation in voltage results from spikes derived from fibres at a distance from the needle point and are considered to be largely derivation artefacts.

3. Small fibre potentials may occasionally appear superimposed upon the negative spike potential due to imperfect fusion of the many fibre potentials making up the normal motor unit but the duration of a normal unit does not exceed 4–5 msec. These irregularities may also be considered derivation artefacts since slight movements of the needle point may result in their disappearance.

4. Other apparent "polyphasic units" are due to either grouped firing of several adjacent motor units or occasional repetitive firing of single units.

5. In muscles of the face and in certain portions of limb muscles, motor units appear to have a more complex organization with overlapping of the distribution of terminal axonal networks causing poorly integrated multiple fibre potentials to appear.

6. Action potentials from single denervated muscle fibres during spontaneous fibrillation are characterized by monophasic, diphasic and rarely by triphasic spikes of 0·5–1·5 msec. duration and voltages usually about 50–100 μV. but reaching 500 μV. in exceptional cases. Prostigmine increases fibrillation in completely denervated muscle, but it may abolish traces of fibrillation in a muscle being re-innervated by regeneration of an interrupted nerve supply.

7. Following re-innervation the separate fibre potentials composing a motor unit may appear poorly synchronized and spatially dispersed within the muscle causing a polyphasic action potential in which the individual units may be separated as much as 10 msec. or more, but maintaining a constant time relation with each other upon repeated firing of the same unit. These disintegrated units are thought to be due to abnormal ramification of the terminal axonal branches in the regenerated nerve terminals in addition to their abnormally slow velocity of conduction.

8. In muscles sensitized by denervation, mechanical stimulation by the needle tip serves to initiate not only a series of fibre potentials of the usual form, but also a series of repeated sharp waves usually of positive electrical sign at the needle tip. These appear to be non-propagated local potentials with form similar to a condenser discharge. They were not observed in normal muscle. These potentials are distinct from any other form of muscle action potential and they appear to be associated with local contractile processes induced by mechanical stimulation in a sensitized muscle. (Authors' abstr.)

Action Potentials of Cerebellar Cortex in Response to Local Electrical Stimulation.

1. Electrical stimulation at or near the surface of a folium of the cerebellum results in the activation of elements thought to be the molecular fibers.

2. These conduct in a lateral direction at a rate of 0·35–0·50 meters per second and to a distance usually not exceeding 5 mm.

3. They, in turn, are capable of synaptically affecting the Purkinje cells.

4. Purkinje cells exhibit a negative potential in or near their dendritic processes whose duration is from 5 to 25 msec.

5. This potential may or may not be associated with an axon spike in the Purkinje cell fiber.

6. Evidence is obtained which leads to the belief that the synaptic delay for this Purkinje cell potential is of the order of 1 msec. (Author's abstr.)

Selective Destruction of Large Motoneurons by Poliomyelitis Virus. (1) *Conduction Velocity of Motor Nerve Fibers of Chronic Poliomyelitis Patients.*

1. Maximal muscle action potentials evoked by appropriate motor nerve stimulation and conduction velocities of the fastest nerve fibers innervating these muscles have been studied in 26 human chronic poliomyelitis patients.

2. The conduction velocities of the fastest residual motor nerve fibers are slower than normal in this disease and vary directly with the action potential amplitude (i.e. functional ability) of the muscles they innervate.

3. Explanations for the slower than normal conduction rates are considered. The most likely cause is thought to be the selective destruction by the virus of those motoneurons with the thickest axons.

4. The mechanism of this proposed preferential invasion and killing by the virus—e.g. metabolic, mechanical, etc.—requires further study.

5. The results are discussed in the light of some of the pathological physiology of the disease. (Author's abstr.)

Synaptic Facilitation in the Crayfish.

The same efferent fibers in the third root of the abdominal ganglia of the crayfish, *Cambarus clarkii*, can be activated by four independent preganglionic pathways.

These are the homolateral lateral giant, the two medial giants and the homolateral first root of the same ganglion.

In preparations in which the third root no longer responds to a single impulse in any preganglionic fiber, two shocks with variable time intervals were given to various pairs of pathways. With most combinations it was found that no summation occurs when the shocks follow each other closely. The length of this "inert period" depends on the distance between the synapses, being longest between the synapses that are furthest apart.

It is concluded that the normal summation periods are caused by a process which spreads along the postganglionic fiber from the first synapse stimulated. This process increases the excitability of the fiber at the neighbouring synapses. It diminishes in intensity with distance as well as with time. Transmission is not possible at the second synapse activated until this process has reached it. With certain combinations, especially that of the two medials, summation does occur when the impulses arrive at the ganglion at the same time. This summation most likely involves the excitation of only one synapse, which is partially activated by the first pathway stimulated and is then fully activated by the action potential of the second giant stimulated. (Author's abstr.)

Excitability Cycle and Interaction in Geniculate-striate System of Cat.

1. The postsynaptic negative spike observed in the lateral geniculate exhibits the general properties of soma potentials.

2. The potential magnitude recorded from a group of units (geniculate or cortex) shows a summation or recruitment interval extending to 30 msec. and a subnormal phase lasting for seconds.

3. The relative recruitment is greater in the subnormal phase and proportional to it by a function not yet defined. It is partly proportional to subliminal fringe and hence an inverse function of strength of stimulus, or of excited zone dimensions.

4. The subnormal phase in the geniculate has never been observed to develop fast enough to quench the first three responses occurring within the initial period of about 5 msec. This initial extinction phenomenon is not necessarily part of the conventionally defined subnormality, however.

5. It is not certain that the first cortical spike is entirely due to radiation fiber action potential.

6. Bilateral interaction occurs at cortical level for all components, but comparable interaction has not been demonstrated anywhere in the geniculate.

(Author's abstr.)

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PROC. AM. PSYCHOPATH. ASS.

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Inheritance of Electroencephalogram Patterns in Children with Behavior Disorders.

1. In the families of children with behavior disorders there is a marked similarity in EEG pattern in the various members of a single family. This is very obvious in members of a family who are of similar age. It is present even in the EEG patterns of parents and their children. A similar familial patterning has been shown in the families of epileptics.

2. There is a higher incidence of dysrhythmic and hence abnormal records in the patients (60 per cent.), than in their " normal " relatives (40 per cent.).

3. The incidence of EEG's in the " normal " relatives having 40 per cent. abnormality is different from that of the total normal population, which has only 10–15 per cent. of abnormal records.

4. It is suggested that anxiety or tension may be related to dysrhythmic and unstable EEG patterns, and that, in consequence, a very high incidence of abnormal EEG's appears in the neurotic or psychotic members of families with relatively unstable psychological background. This is reflected more in the EEG's of children than of adults because the cortical potentials of the former are, under any circumstance, more labile than are those of the latter. (Author's abstr.)

Q. J. ALC.

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REV. NEUROL., B. AIRES.

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Z. GES. NEUROL. PSYCHIAT.

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VOL. CLXXV. 1942

Z. PSYCHOL.

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VOL. CLIV. 1942.

Z. TIERPSYCHOL.

VOL. VI. 1944.

1. Psychiatry.

Frontal Lobe Ablation in Chimpanzee : A Résumé of " Becky " and " Lucy." Crawford, M. P., Fulton, J. F., Jacobsen, C. F., and Wolfe, J. B. [*The Frontal Lobes*, 3.]

The reactions of 2 adolescent chimpanzees in problem-solving situations (tool usage, problem-box, delayed reaction set-up) were studied prior to and after unilateral ablation and following bilateral lobectomy. Follow-up observations were

made at intervals for 3 years. Protocols are given and results at retest periods are presented in detail. In general, bilateral ablation, as opposed to unilateral, resulted in " profound behavioural deficit," but continued post-operative training was accompanied in some problems by improvement. Variability in performance remained " the most clearcut feature." Discussions by W. Freeman, C. F. Jacobsen.
L. A. PENNINGTON (Psychol. Abstr.).

Specialization of Behavioral Functions and the Frontal Lobes. Halstead, Ward C. [*The Frontal Lobes*, 59.]

A summary of the author's monograph (1497) is given with special emphasis placed upon the 4-factor theory of biological intelligence, upon the measurement of these factors in relation to the calculation of an impairment index. The inferences are made that biological intelligence is cortically represented as a gradient maximal in the frontal lobes, and that the four factors in unified fashion shed light upon ego functions. L. A. PENNINGTON (Psychol. Abstr.).

Some Connections of the Frontal Lobe Established by Physiological Neuronography. McCulloch, Warren S. [*The Frontal Lobes*, 95.]

Ten figures are presented to illustrate in summary the neural relations between the two or more sectors of the frontal lobes and between these and thalamic nuclei as inferred from the electrical reactions to local strychninization (neuronography). Brodmann's numbers are used, although no histological differentials are implied. The mechanism by which frontal lobe suppressor areas (8, 4s, 24) operate is considered. L. A. PENNINGTON (Psychol. Abstr.).

The Non-pyramidal Motor Projections from the Frontal Cerebral Cortex. Mettler, Fred A. [*The Frontal Lobes*, 162.]

By comparing the capacities of primates bilaterally devoid of frontal cortex with primates sustaining pyramidotomy the author reviews the recent experimental and selected clinical literature on (1) the non-pyramidal motor functions of the frontal cortex, and (2) the pathways, as far as these are known or suspected, originating in the cortex by means of which the non-pyramidal motor functions are mediated. Brodmann's area designations and their subcortical terminations are tentatively specified and charted. Stress is placed upon the specialists' need to consider both cortical pyramidal and non-pyramidal factors in cases of motor dysfunctions.
L. A. PENNINGTON (Psychol. Abstr.).

The Thalamic Projection to the Frontal Lobe. Freeman, Walter, and Watts, James W. [*The Frontal Lobes*, 200.]

By recourse to methods of defibrillation dissection, myelinization and retrograde degeneration changes in the thalamus following frontal lobotomy in 14 cases, the cortico-thalamic relationship is described at the human level. It is concluded, to illustrate, that all parts of the frontal lobe receive projection fibers from the thalamus ; that " there is no frontal association area " ; that retrograde degeneration of the medial thalamic nucleus " may be an important factor " in relief from emotional tension after lobotomy. Projection of this nucleus upon the cortex is described and charted. L. A. PENNINGTON (Psychol. Abstr.).

The Motor Functions of the Agranular Frontal Cortex. Denny-Brown, D., and Botterell, E. H. [*The Frontal Lobes*, 235.]

Reports of clinical observations made upon the macaque monkey exposed to ablation studies (with appropriate histological and other controls) provide a " reappraisal of some of the facts of motor disorder from frontal lesions " with special reference to areas 4 and 6. Selected results show that " what is represented in area 4 is the whole motor mechanism, and especially the red nucleus, reticular formation and propriospinal internuncial neurons of the cord. The direction of patterns of behavior is determined elsewhere." Ablation of all of area 4 gives rise to spasticity. These and other findings are discussed clinically, theoretically, and in relation to Betz-cell distribution. Comments by J. F. Fulton, P. C. Bucy, F. A. Mettler. L. A. PENNINGTON (Psychol. Abstr.).

Movement of Head and Eyes from Stimulation of Human Frontal Cortex. Rasmussen, Theodore and Penfield, Wilder. [*The Frontal Lobes*, 346.]

In a series of 208 electrical stimulations made during craniotomies performed under local anesthesia, eyeball movement was elicited in 48 stimulations in 31 cases. A third of the points were anterior to the central sulcus; the remainder were located in the anterior half of the precentral gyrus or in the caudal segment of the adjacent frontal convolutions. Head movements, nonspecific in type, were aroused from stimulation of the precentral gyrus at the central sulcus. It is suggested that eye movement elicitation cannot be limited to any one fronto-cortical area inasmuch as areas 4, 6 and 8 are in different cases responsible.

L. A. PENNINGTON (Psychol. Abstr.).

Some Forebrain Mechanisms Involved in Expression of Rage, with Special Reference to Suppression of Angry Behavior. Bard, Philip, and Mountcastle, Vernon B. [*The Frontal Lobes*, 362.]

By means of surgical, observational and histological methods the effects of extirpation of neocortical tissue upon rage behaviour of 5 cats are studied. Results suggest that these animals become quite placid after removal of the neocortex, but that rage reactions are easily aroused following injury to the limbic lobe in these animals or in other subjects with intact neocortex. It is held that under normal circumstances the limbic lobe inhibits the rage mechanisms centered subcortically, and that the cells of the limbic lobe are in turn inhibited by the cells of the neocortex. These and other findings are related to current studies and theory wherein continued research is held necessary to clarify controversial points. Comments by P. C. Bucy, Margaret A. Kennard, R. M. Brickner.

L. A. PENNINGTON (Psychol. Abstr.).

Stimulation and Regional Ablation of Orbital Surface of Frontal Lobe. Livingston, Robert B., Fulton, John F., Delgado, Jose M. R., et al. [*The Frontal Lobes*, 405.]

One hundred controlled observations of preoperative and normal monkeys (15) and chimpanzees (2), the animals subjected to ablation of all or parts of the orbital surface (Walker's area 13), suggest that bilateral extirpation is accompanied by hyperactivity (8 to 16 times the normal), by heightened body-extremity temperature and by augmented reflex vasodilatation. After indicating the limitations of the electrical and ablative techniques the authors conclude by presenting detailed analysis that the orbital surface " is related both to autonomic and somatic mechanisms " perhaps by way of mesopallial connections with the cortex, thalamus, hypothalamus, and extrapyramidal systems, and that this area can be considered a part of the " highest representation of visceral functions."

L. A. PENNINGTON (Psychol. Abstr.).

The Anterior Cingulate Gyrus and Personality. Ward, Arthur A., jun. [*The Frontal Lobes*, 438.]

The neuroanatomy of the anterior limbic region (Brodmann's area 24) is described and the effects both of electrical stimulation and ablation in the monkey are reported. Evidence shows the region to have two powerful functions, viz., " suppression " of cortical activity and motor performance as well as " domination " of the autonomic nervous system. Absence of data at the human level precludes concise statements with reference to progressive personality changes, although the " mental " effects of tumors of the anterior corpus callosum might perhaps be due to limbic-lobe involvement. L. A. PENNINGTON (Psychol. Abstr.).

Effect of Extirpation of Frontal Areas upon Learning Performance of Monkeys. Harlow, Harry F., and Settlage, Paul H. [*The Frontal Lobes*, 446.]

The effects of unilateral and bilateral cortical lesions (especially frontal in locus) on the learning behaviour of 8 monkeys and their controls in 5 test situations are summarized. Results indicate a quantitative and significant inferiority for the operated animals on all tests, with greatest deficit on reversal problems. Whether this partial loss is " general " or a function of " special abilities " is as yet unknown. It is concluded that the results predicate similar deficits in man following frontal lobectomies and lobotomies. L. A. PENNINGTON (Psychol. Abstr.).

Normal and Pathological After-discharge from Frontal Cortex. Walker, A. Earl, and Johnson, Herbert C. [*The Frontal Lobes*, 460.]

The electroencephalographic recordings (8-channel machine) of waves immediately following experimental electrical stimulations of the frontal lobes in 6 normal monkeys, in 20 animals prepared by earlier placement of alum protein plaques on the frontal cortex, in 10 monkeys each with a coil beneath the scalp with terminals in the cortex, provide data that characterize "normal" and "abnormal" self-sustained responses. Selected results indicate the frontal cortex to have the highest threshold, the motor the lowest threshold, the longest acting and most radiating after-discharge. The waves of the "plaque" subject, however, showed a low threshold, wide areal involvement, marked variability in cessation (some stop, start up again and continue for 2 or more hours). Physiologically this datum appears "to play a large role in epileptic activity."

L. A. PENNINGTON (Psychol. Abstr.).

Report of Case of Bilateral Frontal Lobe Defect. Ackerly, S. S., and Bentor, A. L. [*The Frontal Lobes*, 479.]

The neurological, familial, psychological, nosological and etiological aspects, based upon repeated studies during the last 21 years of a 35-year-old white male suffering from bilateral frontal lobe defect, are discussed. Although most psychometric findings (Rorschach and Kohs Blocks, for example) were negative, the patient whose social behavior superficially suggested that the psychopathic personality was unable socially to plan, to pursue a remote goal. He lacked the "abstract attitude" described by Goldstein, was free from anxiety. The chapter supplies the literature with "confirmatory evidence that the frontal lobes are essential . . . for the amplification and elaboration of experience.

L. A. PENNINGTON (Psychol. Abstr.).

The Frontal Homolateral Syndrome in the Light of Observations made on 150 *Cases of Traumatic Lesions. Rouquier, A.* [*The Frontal Lobes*, 505.]

The author describes the subjective and objective "elements" that comprise the recently identified aforementioned syndrome, and relates these to neurological involvements in areas 9, 10, 11, 6α, β. The outward deviation of the outstretched arms (eyes closed), the lowering and falling of the corresponding limbs, homolateral trembling and the dysmyotonic reaction, as syndrome "signs," are described and clinical tests for each indicated. "The frontal convolutions . . . play a very important role in reactions of the head and trunk as well as in the limbs," and they are important "sympathetic and intellectual centers." The neural mechanisms mediating these signs are conjectured.

L. A. PENNINGTON (Psychol. Abstr.).

Symposium on Gyrectomy. Penfield, Wilder, Cameron, D. Ewen, et al. [*The Frontal Lobes*, 519.]

As a substitute for leucotomy procedures 7 patients exhibiting prolonged tension and anxiety were treated by bilateral symmetrical removal of frontal cortex in areas connected with the thalamus, and post-operatively compared with 8 patients of similar diagnosis treated by bilateral frontal lobotomy. Administration of a wide range of psychological and stress tests shows post-operatively a "consistent slight drop in general intelligence, a significant reduction in vocabulary" (in the direction of concreteness). These and other differences are considered as conceivably due to the patients' inability to maintain a "set in the face of interference." The chapter is divided into 3 parts. The first by Penfield discusses operative techniques and anatomy. The second, by D. E. Cameron and M. D. Prados, provides a summary of psychiatric findings, among which is the statement, "gyrectomy is of no greater value than lobotomy." The third, by R. B. Malmo, describes in detail the psychological approach to the problem.

L. A. PENNINGTON (Psychol. Abstr.).

Motor Aphasia and Agraphia Caused by a Small Vascular Lesion Confined to Third and Second Convolutions of Left Frontal Lobe. Scheinker, I. M., and Kuhr, B. M. [*The Frontal Lobes*, 582.]

A clinical description of the aphasic and agraphic behavior of a 61-year-old male is given. Upon his decease, macroscopic and microscopic studies were made

of the brain, revealing a "sharply demarcated area of hemorrhagic softening confined to the upper half of the third and lower third of the second frontal convolution on the left." These observations are related briefly to the historical findings by Broca, Marie and others. L. A. PENNINGTON (Psychol. Abstr.).

The Cortical Motor Pattern Apraxias. Nielsen, J. M. [*The Frontal Lobes*, 565.]

The 3 types (ideational, ideokinetic, cortical motor pattern) of apraxia are defined and described, the experimental and clinical approaches to the subject historically summarized. Major attention is given the latter type of apraxia wherein 3 subtypes are identified, their cortical centers specified on the grounds of clinico-pathological findings. Three illustrative case-histories are given.
L. A. PENNINGTON (Psychol. Abstr.).

Effect of Cortical Excision and Stimulation of the Frontal Lobe on Speech, with a Review of Aphasia and Cerebral Physiology Related to Speech. Robb, J. Preston. [*The Frontal Lobes*, 587.]

Following a review of the recent experimental and clinical literature summarizing the results accruing from cortical stimulation and ablation methods in relation to the anatomical basis of speech, the author sequentially reports observations made on 27 cases of right-handed patients who were treated (1930–1947) for focal epilepsy by surgical excisions from the left frontal lobes. Results include (1) no residual speech defect in 26 cases; (2) the left hemisphere was found dominant in 16 cases; (3) all of the lateral aspect of the lobe with the exception of the posterior part of the 3rd convolution can be ablated with, at worst, only transient aphasia; (4) speech recovery does occur after excision of the posterior part of the 3rd convolution. The value of electrical stimulation (Rahm stimulator) as an adjunct to surgery and research is stressed. Results are discussed briefly in relation to the problem of bilateral localization of speech functions.
L. A. PENNINGTON (Psychol. Abstr.).

Autonomic Functions of Frontal Lobes: Studies on Patients with Head Trauma. Netsky, Martin G., and Starr, Harry. [*The Frontal Lobes*, 610.]

Fifty servicemen, 19 to 38 years of age, and all having a year earlier sustained head injuries, and now all undergoing treatment for consequent post-traumatic seizures, were studied before and after craniotomies (performed by A. Earl Walker) with reference to selected autonomic functions, including sweating, skin temperature, blood pressure. Observations of control and experimental subjects indicate that autonomic changes, especially those of sweating and skin temperature, "were related to a contralateral area of the cortex, extending anteriorly from the post-central gyrus to an undetermined distance beyond the precentral gyrus . . . the representation of autonomic influence in the cortex was analogous in position to the somatic." These and other findings are related to recent research studies on cortico-autonomic correlations. L. A. PENNINGTON (Psychol. Abstr.).

Psychological Changes Produced by Frontal Lobotomy. Yacorzynski, G. K., Boshes, Benjamin, and Davis, Loyal. [*The Frontal Lobes*, 642.]

Detailed and repeated psychological examinations (21 different tests) of a 36-year-old male patient in good contact were made prior to and after lobotomy. Results include: (1) the observation that the operation was accompanied by none of the changes consequent to frontal lesions; (2) no evidence for a change in motivation (aspiration level and degree of effort tests); (3) a decrease of 17–18 I.Q. points on the Wechsler Scale with greater loss on the verbal scale—especially on arithmetic reasoning; (4) lowered performance on the Vigotsky test and in those reasoning test situations demanding the "use of concepts removed from immediate experience"; (5) while the Rorschach suggested the release of a psychosis, clinical impression and the MMPI psychogram suggested improvement. These and other test results are "speculated" upon in relation to theory and to the variant results that obtain from lobectomies. L. A. PENNINGTON (Psychol. Abstr.).

Telencephalization of Survival Characteristics. Brickner, Richard M. [*The Frontal Lobes*, 658.]

The absence of "neurological comprehension" of the manner by which the nervous system participates in the mediation of prehuman attributes leads the

author to propose and develop the thesis that the cortex " instead of being principally devoted to the *suppressing* of what we call primitive impulses, actually has as one of its chief functions the *expressing* of these impulses " (survival functions). " This results from . . . the familiar process of encephalization." These survival characteristics (rage, for example) are telencephalized by all persons, the form determined by the culture. Thus, telencephalization is the neural process underlying acculturation, wherein culture promotes repressions of certain ways of expressing the survival functions. It is then posited that most social and personal problems arise from normal rather than from abnormal behavior and that much needed social therapy must be telencephalized through insight.

L. A. PENNINGTON (Psychol. Abstr.).

Personality Analysis Before and After Frontal Lobotomy. Rylander, Gosta. [*The Frontal Lobes*, 691.]

An analysis of the " changes in detail " that accompany leucotomy is made by careful observational and psychometric pre-operative and post-operative study of 8 of a series of 32 patients in the group reported upon. Descriptions of interest loss, of " shallowness and forgetfulness " are given, as well as a characterization of the patients' " reduction " in attention, in word-naming, and in I.Q. scores (Stanford Revision). These and other findings lead to a conclusion of reluctance to use this surgical method upon nonpsychotic patients . . . and these " findings are at least a warning that intellectual damage may follow lobotomy." Discussion by R. M. Brickner, W. Freeman, D. M. Rioch, R. R. Grinker, J. M. Nielsen.

L. A. PENNINGTON (Psychol. Abstr.).

Relief of Intractable Pain of Organic Origin by Frontal Lobotomy. Falconer, M. A. [*The Frontal Lobes*, 706.]

Medical and psychometric descriptions are given of one case of post-herpetic neuralgia and of one of tabes dorsalis both successfully treated by lobotomy. The finding earlier reported by Freeman and Watts, that the chief effect of the operation is to modify the patient's reaction to pain rather than to abolish pain perception is reiterated. This surgical approach is suggested as a last resort in such cases because of the personality changes observed to accompany the operation.

L. A. PENNINGTON (Psychol. Abstr.).

Frontal Lobotomy in the Treatment of Unbearable Pain. Watts, J. W., and Freeman W. [*The Frontal Lobes*, 715.]

The positive (" striking ") effects of lobotomy upon a series of organic cases where unbearable pain, continuous or paroxysmal, was a major complaint are reported by describing the pre- and post-operative reactions of 9 patients (carcinoma, thalamic syndrome, tabes dorsalis, causalgia, phantom limb, atypical facial neuralgia). It is posited that the operation " interrupts the central process " responsible for reaction to pain as a threat. " Pain then becomes a sensation only."

L. A. PENNINGTON (Psychol. Abstr.).

The Psychological Effects of Frontal Lobotomy Performed for the Alleviation of Pain. Koskoff, Y. D., Dennis, Wayne, et al. [*The Frontal Lobes*, 723.]

The effect of lobotomy in the relief of chronic pain is studied, with appropriate controls, by reference to 10 patients (several " somatic," others " psychic ") whose symptoms are reported (1) medically and clinically, (2) psychometrically (7 cases). In the latter, the Wechsler, Babcock-Levy, Porteus Maze, Rorschach, TAT and Goodenough Drawing tests are administered to the " organic " patients before operation and again 3 months afterward. All organic-pain cases free from " mental abnormality " showed some loss in intellectual abilities of a general type followed by, it is believed, a gradual restitution. The danger of generalizing from so few cases is stressed, present results being reported as " trends." Data obtained from the administration of personality tests are to be reported later.

L. A. PENNINGTON (Psychol. Abstr.).

Measurements of Heat Stimulus Producing Motor Withdrawal Reaction in Patients following Frontal Lobotomy. Chapman, William P., Rose, Augustus S., and Solomon, Harry C. [*The Frontal Lobes*, 754.]

The pre- and post-operative reactions of 23 patients (19 schizophrenics, 1 involutional melancholia, 3 psychoneurotics) to heat stimuli are studied in order to measure experimentally one aspect of the clinical observation that such patients show "an increased withdrawal reaction . . . to discomforting stimuli." For this the Hardy-Wolff-Goodell pain threshold apparatus was used; the heat required on the forehead to induce a wince and a head withdrawal was recorded. Post-operative retests were continued at intervals for 12 months. Wince and pull-away responses were significantly more easily elicited (lower threshold) after operation with this difference still present (although less so) 12 months later. "We have no explanation for the apparent discrepancy between these results and the fact that frontal lobotomy relieves intractable pain."

L. A. PENNINGTON (Psychol. Abstr.).

A Co-operative Clinical Study of Lobotomy. Moore, B. E., et al. [*The Frontal Lobes*, 769.]

As a co-operative inter-agency research program initiated in 1946, 200 chronic institutionalized cases (163 schizophrenics, 10 manic-depressives, 8 involutionals, 4 psychoneurotics, 15 miscellaneous) were studied, pre- and post-operatively, according to uniform procedures previously agreed upon. Follow-up studies at 1, 3, 6 and 12 post-operative months were made. Selected results include, (1) the patient's condition by 3 post-operative months is a "good indication" of future developments in his case; those showing moderate to marked improvement continue to improve; (2) paranoid schizophrenics show the best prognosis, with catatonics second; (3) improvement among schizophrenics is unrelated to the length of hospitalization, but this is less true among non-schizophrenic patients; (4) 75 per cent. of the 200 patients showed an amelioration of those symptoms most troublesome to personnel (suicide attempts, etc.); (5) sex and intelligence can be ignored as prognostic criteria. L. A. PENNINGTON (Psychol. Abstr.).

2. Biochemistry, Physiology, Pathology, etc.

Determination of Proteins in Cerebro-spinal Fluid. R. G. Willcocks. [*Nature*, **163**, 329–30 (1949).]

A rapid, approximate method for medical use has been devised. The xantho-proteic reaction was made quantitative and compared with an artificial standard; mg. per cent. protein = T/S × c × 1·6, where T/S = ratio of color intensities of sample and standard and c = concentration of the standard $K_2Cr_2O_7$ in mg. per cent. A hollow wedge colorimeter was used. No data on tests of the method are included. E. NEWTON (Chem. Abstr.).

Chemical Analysis of Spectrophotometric Findings in the Cerebrospinal Fluid. Spiegel-Adolf, M., Wycis, H. T., and Spiegel, E. A. [*Science*, **109**, 335–6 (1949) *cf. C. A.*, **42**, 3480c.]

Cerebrospinal fluids from 40 patients with organic or functional nervous disorders were examined for ultra-violet absorption at 2650 A, ascorbic acid content, total protein, globulin, cell count, and Wassermann, and colloidal gold tests. Ascorbic acid values were converted into extinction coefficient (E) and subtracted from experimentally determined E of the cerebrospinal fluid, resulting in a difference D_1. Protein values were likewise converted, a residual value D_2 being obtained. Three groupings of D_1, 0–0·4, 0·41–0·8, 0·8–2·5, and D_2, 0·0–14, 0·14–0·61, 0·61–2·16 were made, the values varying in different types of disorders. Selective cerebrospinal fluid absorption is not always explained by ascorbic acid and protein content. D_2 values were particularly pronounced in central nervous system tumors, where destruction of nuclear substances may be expected. The differences between the mean E of normal cerebrospinal fluid, and that of fluid withdrawn 1 hour to 2 months after cerebral concussion were statistically significant, and D_2 values above normal were noted in the fluid shortly after such concussion. The D_2 values

are believed due primarily to nuclear substance constituents, such as purine-pyrimidine compounds, which may be nucleic acids or their cleavage products.
JOHN P. CRISPELL (Chem. Abstr.).

Thiaminase and the Action of Acetylcholine on the Heart. Bettencourt, J. Moniz de, and Abecasis, L. [*Compt. rend. soc. Biol.*, **142**, 1147–8 (1948).]

In the perfused turtle heart, added thiaminase destroys thiamine and thereby makes the heart less sensitive to acetylcholine. L. E. GILSON (Chem. Abstr.).

Behaviour of Ocular Tension and Retinal Arterial Pressure in Acetylcholine Shock. Rosso, Silvio. [*Rass. ital. Ottalmol.*, **16**, 357–69 (1947).]

In 15 young schizophrenic patients undergoing shock therapy by rapid intravenous injection of 0·5 gm. acetylcholine bromide in 2 ml. water, during 90 seconds after injection the systolic blood pressure usually fell 20 mm. Hg, the retinal arterial pressure decreased 5–15 mm., and the intraocular pressure was lowered 5 mm. Hg (average). The retinal arteries appeared to become first constricted and then dilated. W. M. GRANT (Chem. Abstr.).

Convulsant Action of Acetylcholine. Arduini, A., and Machne, X. [*Arch. fisiol.*, **48**, 152–67 (1949).]

In unanesthetized rabbits Jacksonian epilepsy was caused by acetylcholine 4–10 per cent. applied to the masticatory cortex. The effect was increased by 1 per cent. eserine, and prevented by intravenous atropine 1 mgm./kgm. No similar effect was seen after application of saline of equal tonicity.
H. L. WILLIAMS (Chem. Abstr.).

The Mechanism of Action of Barium Chloride and Acetylcholine in Biological Systems. Capraro, V., and Barilli, V. [*Arch. intern. Physiol.*, **56**, 241–4 (1949).]

The following observations demonstrate that $BaCl_2$ does not act entirely through the liberation of acetylcholine (1) : (1) $BaCl_2$ suppresses the action of (1) in the isolated frog or rabbit heart. (2) In the isolated frog heart, atropine inhibits the action of (1), but not that of $BaCl_2$. (3) KCl diminishes the contraction of the isolated small intestine of the guinea-pig produced by $BaCl_2$, but not that caused by (1). FRED H. SNYDER (Chem. Abstr.).

Cholinesterase in Blood and Spinal Fluid in Psychiatric Cases. Glasson, B., and Mutrux, S. [*Presse Méd.*, **55**, 230–1 (1947).]

See *C. A.*, **42**, 3480*d*. A. E. MEYER (Chem. Abstr.).

Thiamine and the Inhibition of Cholinesterase by Eserine. Bettencourt, J. Moniz de, and Cardoso, M. Rodrigues. [*Compt. rend. soc. Biol.*, **142**, 1149 (1948).]

In guinea-pig serum *in vitro* the activity of cholinesterase inhibited by eserine is not restored by addition of thiamine. L. E. GILSON (Chem. Abstr.).

Correlation between Signs of Toxicity and Cholinesterase Level of Brain and Blood during Recovery from Diisopropyl Fluorophosphate (DFP) Poisoning. Freedman, A. M., Willis, Alice, and Himwich, H. E. [*Am. J. Physiol.*, **157**, 80–7 (1949) ; *cf. C. A.*, **42**, 7450*a*).]

After the injection of large doses of DFP non-specific plasma cholinesterase regenerates more rapidly than the specific cholinesterases of the red blood corpuscles and the brain. A lag in regeneration of erythrocyte cholinesterase activity lasting from 24 to 48 hours occurs after injection of DFP, a period during which brain cholinesterase regenerates rapidly. Subsequently the rate of regeneration of erythrocyte cholinesterase becomes more rapid and surpasses that of the brain. A close relation appears to exist between the severity of the toxic signs of DFP poisoning and the level of brain cholinesterase during the regeneration period. The relation between toxic signs and erythrocyte cholinesterase activity is less exact, and fails altogether in the case of the plasma.
E. D. WALTER (Chem. Abstr.).

Relation between Cholinesterase Inhibition and Function in a Neuroeffector System. Riker, Walter F., jun., and Wescoe, W. Clarke. [*J. Pharmacol. Exptl. Therap.*, **95**, 515–27 (1949).]

The rate of irreversible inactivation of cholinesterase (I) in the submaxillary gland of the cat by diisopropyl fluophosphate (DFP) shows the characteristics of a first order reaction. Approximately 50 per cent. of the glandular (I) can be inhibited without affecting response to electric stimulation of the chorda tympani. Beyond this point changes in function accompany further progressive inactivation of (I). These changes include a decrease in the frequency of stimulation required to elicit a threshold response, a prolongation of the response to maximum stimuli, and eventually the onset of spontaneous salivary secretion when the (I) activity is reduced to 10 per cent. or less of the normal. These findings are discussed with respect to physiological and pharmacological interrelationships affecting chemical mediation at neuroeffector junctions. L. E. GILSON (Chem. Abstr.).

Tubocurarine Antagonism and Inhibition of Cholinesterases. Blaschko, H., Bulbring, E., and Chou, T. C. [*Brit. J. Pharmacol.*, **4**, 29–32 (1949).]

The antitubocurarine (I) activity of a series of compounds related to prostigmine (II) was compared with their inhibitory action on true cholinesterase (III) of dog caudate nucleus and on the pseudocholinesterase of horse serum. There is a significant correlation between (I) activity and inhibitory action on true (III). This supports the view that it is the true (III) which is responsible for the destruction of acetylcholine at the site of its physiological action. Substitution of the Me groups on the quaternary N of (II) by Et at first increases both the (I) and the anti-(III) activity, but the peak is reached at the diethyl compound, and the activity of the triethyl compound is again much less. A strongly basic N radical in the phenolic moiety of the (II) molecule is indispensable not only for strong (I) activity, but also for a strong anti-(III) activity.

H. O. SINGHER (Chem. Abstr.).

Rate of Penetration of Electrolytes into Nerve Fibers. Rothenberg, Mortimer A., and Feld, Emily A. [*J. Biol. Chem.*, **172**, 345–6 (1948).]

The permeability of surface membranes to K, Na and Ca and their rate of penetration have been investigated with giant nerve fibers of squid exposed to artificial sea water in which normally occurring ions were replaced individually by radio-active K^{42}, Na^{24} or Ca^{45}, in equimolar concentrations. Concentration of each ion in the axoplasm increased, K^+, which showed high concentration in axoplasm, penetrating within 60 minutes to a concentration double that outside the cell; Na^+ (low in exoplasm) penetrated rapidly for 15–20 minutes, then decreased, the amount inside the cell rising to 25–30 per cent. that outside; Ca^{++} (low in axoplasm) penetrated to a concentration 60–80 per cent. that outside. The ratio of the concentration of the 3 ions (C_i) compared to that outside (C_o) is a function of time of exposure. Results confirm that permeability requires an active process in surface membranes. JOHN P. CRISPELL (Chem. Abstr.).

Carbonic Anhydrase in the Central Nervous System of the Developing Fetus. Ashby, Winifred, and Butler, Ellen. [*J. Biol. Chem.*, **175**, 425–32 (1948); *cf. C. A.*, **42**, 5105*h*.]

The central nervous system of the normal fetus of cattle exhibited a progressive rostral increase in carbonic anhydrase (I). (I) was absent from the cerebrum until shortly before birth; at birth the adult pattern of (I) was established. A similar increase of (I) occurred in the human fetus except that no (I) was found in the pallium of the full term infant. This finding is discussed.

JOHN HEARON (Chem. Abstr.).

Requirement for Diphosphopyridine Nucleotide in the Aerobic Metabolism of Pyruvate by Brain Tissue. Larner, Joseph, Jandorf, Bernard J., and Summerson, Wm. H. [*J. Biol. Chem.*, **178**, 373–82 (1949).]

Diphosphopyridine nucleotide (DPN) was demonstrated to be an essential co-factor in the oxidation of pyruvic acid by H_2O-homogenized mouse brain. Other co-factors were inorganic $PO_4^{---}Mg^{++}$, adenosine triphosphate, fumarate,

and cytochrome c. The DPN requirement became apparent only after thorough homogenization of the tissue. When it was homogenized in 1–3 per cent. nicotinamide solutions instead of H_2O, it was unnecessary to add DPN. Since highly homogenized brain preparations had a higher DPNase activity than coarse homogenates, it is likely that intracellular DPNase is released or activated by thorough disintegration, thus promoting the decomposition of DPN present in the tissue. Nicotinamide apparently inhibited the DPNase activity.

ERICH HEFTMANN (Chem. Abstr.).

Presence of Histamine in Central Nervous System Extracts. Cicardo, V. H., and Stoppani, A. O. M. [*Nature*, **163**, 365 (1949).]

Histamine is the main agent responsible for the depressor effect produced when alcohol or aqueous extracts of ox- sheep, dog or rabbit brain are injected into laboratory animals. In dogs benadryl reduced the depressor effect induced by the extracts and small doses of histamine to the same degree. Atropine did not show this phenomenon. This histamine equivalent of the extracts was determined by comparison of the effect on arterial blood pressure of varying concentrations of extract with varying concentrations of histamine solutions. Acetone and CCl_3COOH extracts gave similar results. Et_2O extracts were inactive. Further fractionation was carried out after purification of the extracts with basic lead acetate. The active portion was precipitated with phosphotungstic acid or Ag-Ba hydroxide, and the depressor effect of this portion was also inhibited by benadryl.

J. P. DANEHY (Chem. Abstr.).

Chemical Topography of the Trace Elements in the Brain of Man, according to Spectroscopic Analysis. Voinar, A. O., and Rusanov, A. K. [*Biokhimiya*, **14**, 102–6 (1949).]

The amount and distribution in the brain of the following trace elements (elements which form less than 1 per cent. of the ash) have been determined by their emission spectra: Ag, Al, Bi, Cr, Cu, Mn, Mo, Ni, Pb, Si, Sn, Ti, V, and Zn. Sections of the brain which differ in morphological structure and physiological function possess a different chemical composition of the trace elements.

H. PRIESTLEY (Chem. Abstr.).

Anaerobic Leakage of Potassium from Brain. Dixon, K. C. [*Biochem. J.*, **44**, 187–90 (1949).]

Considerable amounts of K leak into the extracellular fluid from the brain cells when their supply of O_2 and glucose is cut off. Since enzymic activity in the cells depends upon the high concentration of K, its loss may be responsible for the damage. The effect of the glucose is probably associated with its use in glycolysis. At any rate, inhibition of glycolysis by NaF causes a similar leakage of K from the brain cells, as it also does in the red blood cells.

S. MORGULIS (Chem. Abstr.).

Cytochrome c in Human Brain. Buscaino, Giuseppe Andrea. [*Acta Neurol. (Naples)*, **3**, 584–96 (1949).]

By the Fujita method cytochrome c was determined in 41 human brains; it was maximum (up to 22·85 mgm. per cent.) in the cerebral cortex, followed by the 3rd frontal, the striatum, thalamus, substantia nigra, postrolandic, calcarine, etc. Old subjects showed rather increased values. Brain patients showed in the various zones a lower concentration than normal subjects. In some specific diseases, as progressive paralysis, schizophrenia, and parkinsonism, the lower concentration of cytochrome c was found only in certain zones of the brain.

C. SCANDURA (Chem. Abstr.).

Biochemical Changes in the Blood Caused by Emotions. Cotugno, Vito. [*Arch sci. Med.*, **87**, 201–6 (1949).]

Emotions cause modifications in the glucose tolerance and in K and polypeptides in the blood. The modifications are related to the orientation of the autonomic system of the person.

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

Cerebral Constituents in Relation to Blood Gases. Gurdjian, E. S., Webster, J. E., Stone, W. E., and Kopala, J. [*Am. J. Physiol.*, **156**, 149–57 (1949); *cf. C. A.*, **43**, 3088*c*.]

Cerebral tissue from morphinized dogs was obtained for chemical analysis by freezing *in situ* with liquid air. Lactic acid, glucose, glycogen and acid-soluble P compounds were determined. Blood samples obtained immediately prior to the freezing were analysed for lactic acid, glucose, O, CO_2 and pH, and the tensions of O and of CO_2 were calculated. The effects of variations in the respired gas mixtures were investigated. Variation of arterial CO_2 tension in the presence of adequate O caused no significant changes in the cerebral constituents studied. Hypoxia induced an increase of lactic acid and a breakdown of phosphocreatine in the brain. These changes were diminished when the associated acapnia was counteracted by adding CO_2 to the respired gas mixture. The cerebral adenosine triphosphate remained undecomposed during hypoxia until the extreme limit of tolerance was reached. At this point the phosphocreatine was almost completely decomposed and decomposition of adenosine triphosphate began. The cerebral glucose remained in the normal range during hypoxia. The glycogen values were normal. Changes of CO_2 tension did not play a major role in the regulation of the electro-corticogram either in the presence or in the absence of adequate O.

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

Influence of Atomic Decomposition Products on the Functional Condition of the Nervous System. Vasil'ev, L. L. [*Nauch. Byull. Leningrad. Gosudarst Univ., No.* **18**, 16–18 (1947).]

Alpha-particles (from polonium source) impacting on the sciatic nerve preparation (frog specimen) at 3–4 mm. distance, with stimulation of the nerve being produced by an induction coil (100 cycles per sec.), show an initial drop of stimulation threshold, followed by a rise to a steady level which maintains itself for 1–2 hours, after which a rapid decline sets in and the tetanic effects decline rapidly; the initial contractions become more prominent, followed by rapidly developing parabiosis. The action of the exposure to alpha radiation is similar to the action of ultraviolet irradiation, and is complementary to the action of other parabiotic agents; thus 0·5 hour exposure followed by immersion in isotonic KCl produces parabiosis in 9–10 minutes (instead of normal 12–15 minutes).

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

Carotid Sinus Discharge and the Adrenals. Arterenol as a Chemical Mediator of Sympathetic Nerve Stimulation and Hormone of the Adrenal Medulla. Holz, Peter, and Schumann, Hans Joachim. [*Arch. exptl. Path. Pharmakol.*, **206**, 49–64 (1949).]

In cats and dogs clamping both carotids provokes carotid sinus discharge, resulting in rise of blood pressure and contraction of the spleen, but no change in blood sugar and no inhibition of intestinal contractions. These effects are not those of adrenaline, but are like those of noradrenaline which presumably originates in the adrenal medulla.

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

Microchemical Studies of the Nervous System. VI. Water and the Sulphur-, Phosphorus-, and Nitrogen-containing Fractions of the Brain of the Newborn and Adult Mouse. May, Raoul Michel. [*Rev. can. biol.*, **7**, 642–61 (1948); *cf. C. A.*, **43**, 4308*c*.]

The methods of chemical analysis previously applied to degenerating brains and nerves (*C. A.*, **25**, 1562; **35**, 3301[9]; **42**, 5486*d*) were applied to the study of the brain cells of newborn, 7-day, 14-day, and adult albino mice. There was a decrease in water content from 86·93 in the newborn to 77·41 per cent. in the adult. On the dry basis total S decreased from 1·043 at birth to 0·719 per cent. in the adult; lipoid S decreased from 0·183 to 0·116, with a temporary increase to 0·233 per cent. at 7 days; protein S increased from 0·300 to 0·371 per cent. On the dry basis total P increased from 1·892 at birth to 1·949 at 7 days, and dropped to 1·396 per cent. in the adult; lipoid P increased from 0·549 at birth to 0·799 per cent in the adult; protein P decreased from 0·378 to 0·184 per cent. Total N decreased from 11·32 at birth to 9·67 per cent. in the adult; protein N, from 8·89 to 7·39 per cent.; lipoid N increased during the first 2 weeks from 0·76 to 1·26, and fell to

0·70 per cent. in the adult. There is an increase in phosphatides during the post-natal myelinization. In all cases there was a decrease in water-soluble fractions during growth, in water-soluble S from 0·641 to 0·210, in water-soluble P from 0·895 to 0·357, in water-soluble N from 2·13 to 1·24 per cent. The results are also given as percentage of the fresh brain and as absolute weight during the growth of the brain. These figures, compared with those obtained in the study of nerve degeneration, show that in the latter process there is a differentiation of certain cellular constituents, and in addition there are special chemical transformations that are different from a simple reversion to the embryonic state. Quantitative analyses of the constituents of the nerve cells reveal variations which shed more light on their age, and consequently on their adaptive possibilities at a given stage of their growth, than the modifications revealed by presently known cytological techniques. A. PAPINEAU-COUTURE (Chem. Abstr.).

Cell Reactions in the Hypothalamus following Overloading of the Antidiuretic Function. Hillarp, Nils Åke. [*Acta Endocrinol.*, **2**, 33–43 (1949).]

Cytological changes in the cells of the hypothalamus of rats have been studied during acute and chronic overloading of the antidiuretic function. Acute effects were obtained by intravenous or subcutaneous injections of 5 per cent. NaCl, and produced a distinct chromatolytic reaction in the nerve cells of the supraoptic nucleus and in the magnocellular portion of the paraventricular nucleus. Chronic effects were produced by giving NaCl in drinking-water as follows: 1·5 per cent. NaCl for one week, 2·0 per cent. and 2·5 per cent. for two weeks. Changes in the cells included cellular hypertrophy, an increase in the Nissl-substance, consisting of large coarsely granular aggregations in the periphery of the cell, and an increase in size of the nucleoli in both the supraoptic nucleus and the magnocellular portion of the paraventricular nucleus. ELVA G. SHIPLEY (Chem. Abstr.).

The Localization of Hypothalamic Centers Controlling the Gonadotropic Function of the Hypophysis. Hillarp, Nils Åke. [*Acta Endocrinol.*, **2**, 11–23 (1949).]

Brain lesions of different sizes were made in various regions of the hypothalamus in female rats. From the results it appears the center for luteinizing hormone (LH) secretion is in the anterior hypothalamus, anterior and ventral to the paraventricular nucleus. Lesions in these areas produced follicles but no corpora lutea, and a condition of constant estrus. Lesions caudal to the paraventricular nucleus in the form of small bilateral basal injuries produce the same disturbance, indicating a fiber system originating in the anterior hypothalamus. Complete inhibition of LH does not seem to occur with destruction of the centre or the fiber tract.
ELVA G. SHIPLEY (Chem. Abstr.).

Psychotic States: Correlations of Biochemical and Histologic Changes in the Brain. Barondes, R. de Rohan, Kuhns, Ralph H., Levine, David I., and Strachan, C. E. [*Military Surgeon*, **104**, 278–85 (1949).]

A review attempting to correlate metabolic disturbances of glycogen, sulfhydryl, creatinine, lipides, glutamic acid, enzymes, etc., to psychotic and parapsychotic states. B. LUSTIG (Chem. Abstr.).

Neuronal Changes Associated with Tetany of Alkalosis and Hypocalcemia. Koenig, Harold, and Koenig, Ruth. [*Proc. Physiol. Soc. Philadelphia, Jan.* 18, 1949; *Am. J. Med. Sci.*, **217**, 466 (1949).]

Hypocalcemic tetany decreased the cytoplasmic basophilia, and alkalotic tetany increased it in the large motor neurons of the spinal cord and brain-stem of cats.
MARION HORN PESKIN (Chem. Abstr.).

3. Pharmacology and Treatment.

Anticonvulsant Drugs: Mechanisms of Action and Methods of Assay. Goodman, L. S., Toman, J. E. P., and Swinyard, E. A. [*Arch. intern. Pharmacodynamie*, **78**, 144–62 (1949).]

The action of the anti-epileptic drugs is discussed with data on 4 methods of assay in rats. M. L. C. BERNHEIM (Chem. Abstr.).

Vasomotor Drugs on the Convulsant Threshold in Rodents with and without Diphenylhydantoin. Hanzlik, P. J., Cutting, W. C., Hoskins, Dean, Hanzlik, Harold-Barnes, E. W., and Doherty, E. W. [*Stanford Med. Bull.*, **6**, 47–53 (1948).]

Ergotamine, ergonovine and fluid extract of ergot (I) consistently raised the threshold for electric convulsions in rabbits whether or not they had received intravenously 25 mgm. diphenylhydantoin (II)/kgm. body-weight 2 hours previously. (I) also had this effect on rats whether or not they had received 20–40 mgm. (II) gastrically 2–4 hours in advance. The actions of epinephrine, ephedrine, tyramine and amphetamine (III) on rabbits, and of (III) on rats were more variable, but these drugs usually raised the convulsive threshold moderately or negligibly in animals not treated with (II) and decreased the anticonvulsant activity of (II). $NaNO_2$, papaverine, and aminophylline, especially the latter, reduced the thresholds in treated and untreated rabbits. The threshold in these animals was not appreciably affected by quinine, cinchophen, azosulfamide, parathyroid solution, or Ca gluconate. MARSHALL E. DEUTSCH (Chem. Abstr.).

Effect of Convulsant and Anticonvulsant Agents on the Activity of Carbonic Anhydrase. Torda, Clara, and Wolff, Harold G. [*J. Pharmacol. Exptl. Therap.*, **95**, 444–7 (1949).]

Known methods for determining the activity of carbonic anhydrase from human blood *in vitro* are described. Low concentrations (0·0004 *M* or less) of convulsant drugs (acetylcholine, camphor, DDT, digitoxin, metrazole, picrotoxin, scilliroside, strychnine, ouabain) decreased the activity and anticonvulsants (hydantoin, methylphenylethylhydantoin, phenobarbital) increased it.

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

Antagonism between Metrazole and Anticonvulsants Demonstrated by the Rabbit Electroencephalogram. Bertrand, Ivan, Quivy, D., and Gayet-Hallion, Th. [*Compt. rend. soc. Biol.*, **142**, 1357–60 (1948).]

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

Anticonvulsive Action and Molecular Structure of Some Heterocyclic Pentagonal Compounds. V. Duration of the Anticonvulsive Action of Dimethyldithiohydantoin. Hazard, Rene, Cheymol, Jean, Chabrier, Pierre, and Smarzewska, Klaudia. [*Compt. rend.*, **228**, 958–60 (1949); *cf. C. A.*, **43**, 2324*a*.]

Intravenous injections of 25 mgm. pentetrazole per kgm. produce epilepsy-like crises, which, however, can be prevented by injections of 50 mgm./kgm. of Ca salt of dimethyldithiohydantoin (I). In experiment (*a*), a mixture of pentetrazole and (I) was injected intravenously into 4 rabbits. In (*b*) the pentetrazole injection was followed within 48 to 51 seconds by (I). In (*c*) (I) was injected first, followed by pentetrazole from 1 minute to 4 hours later. The best protection against convulsions was provided by (*c*) with an interval of 15 minutes between injections. This protective effect of (I) lasted several hours. D. A. M. (Chem. Abstr.).

Curare-like Actions of Tri(diethylaminoethoxy) Benzene Triethyliodide. Mushin, Wm. W., Wien, R., Mason, D. F. J., and Langston, G. T. [*Lancet*, **256**, 726–8 (1949); *cf. W., Arch. intern. Pharmacodynamie*, **77**, 96 (1948); *J. Physiol. (London)*, **107**, 44 (1948).]

Flaxedil (I) in doses of 40–70 mgm. (about 1 mgm./kgm. of body-weight), injected intravenously in conscious persons, produced complete paralysis of the abdominal and forearm muscles without affecting the diaphragm or pulmonary ventilation. Recovery was full and complete within 30 minutes. The paralysis induced by (I) was rapidly neutralized by intravenous prostigmine.

BARBARA R. MURRAY (Chem. Abstr.).

Comparison of Decamethonium Iodide (C10) *with* d-*Tubocurarine in Controlling Electrically Induced Convulsions. Hobson, J. A., and Prescott, F.* [*Lancet*, **256**, 819–20 (1949).]

C10 (I) (0·08 mgm./kgm. of body-weight) and thiopentone (bistrimethylammonium pentane diiodide) (C5) were administered to 40 patients undergoing electroconvulsion therapy; at other times 20 of the patients received *d*-tubo-

curarine (0·3 mgm./kgm. of body-weight) (II). (I) was considered preferable to (II), since it did not produce histamine-like reactions, and the curarization produced by (I) passed off more rapidly. BARBARA R. MURRAY (Chem. Abstr.).

Curare and Curare-like Substances. Pharmacology and Clinical Use. Mussgnug, Gunther. [*Pharmazie*, **4**, 17–20 (1949).] H. A. WEGNAR (Chem. Abstr.).

The Action of Synthetic Curarizing Compounds on Skeletal Muscle and Sympathetic Ganglia, both Normal and Denervated. Bulbring, Edith, and Depierre, France. [*Brit. J. Pharmacol.*, **4**, 22–8 (1949).]

The action of β-diethylaminoethoxy benzene ethiodide (I) 1, 3-bis (β-diethylaminoethoxy) benzene diethiodide (II), and 1, 2, 3-tris (β-diethylaminoethoxy) benzene thriethiodide (III) was studied. On normal skeletal muscle (III) has a strong curarizing action against both electric stimulation of nerve to muscle and intra-arterial injections of acetylcholine (IV); (I) is one-fiftieth to one-twelfth as strong. This ratio of activity is reversed on the sympathetic ganglion whether it is stimulated through its preganglionic nerve or by intra-arterial injection of (IV), (I) being 50–100 times stronger than (III). On denervated skeletal muscle, stimulated by (IV), the ratio of activities of (I) and (III) is the reverse of that in normal muscle, provided that the doses of the curarizing agents are small. Large doses of (I) have a sensitizing action on denervated muscle; as the dose is increased the diminution of the muscle response to (IV) gives way to an augmentation. This effect has also been observed with *d*-tubocurarine (V). (I) and (V) both have a stimulant action by themselves on the normal as well as on the denervated sympathetic ganglion and on denervated muscle. A 50 per cent. inhibition of true cholinesterase (VI) of dog caudate nucleus was caused by (II) (10^{-3} M). (I) and (III) do not inhibit (VI) in this concentration. The muscle response to (IV) was depressed by (II) only in doses of about 1 mgm., and (II) was intermediate in potency between (I) and (III). H. O. SINGHER (Chem. Abstr.).

Effect of Extracellular Electrolyte Depletion on Brain Electrolyte Pattern and Electroshock Seizure Threshold. Swinyard, Ewart A. [*Am. J. Physiol.*, **156**, 163–9 (1949).]

Data are presented showing the distribution of water, Na, K, and Cl in the plasma and brains subjected to acute extracellular electrolyte depletion and subsequent replacement and the relation of these alterations to concomitant changes in electroshock threshold. The electroshock threshold is not closely correlated with total brain water or the calculated intracellular fluid volume; it is independent of the volume of extracellular fluid and of the intracellular K concentration. The electroshock seizure threshold is more closely correlated with extracellular Na and Cl concentration than with other factors examined.

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

The Action of Autonomic Drugs on Elasmobranch and Teleost Involuntary Muscle. Dreyer, N. B. [*Arch. intern. Pharmacodynamie*, **78**, 63–6 (1949).]

Elasmobranch smooth muscle is not affected by pilocarpine. Acetylcholine causes contraction unaffected by atropine. Adrenaline, ergotoxine and ergotamine are powerful stimulants, but ephedrine and amphetamine are without action. In teleosts, parasympathomimetic drugs are inhibited by atropine, and adrenaline causes relaxation. M. L. C. BERNHEIM (Chem. Abstr.).

Central Inhibitory Effects of Carbon Dioxide. (II) Macacus rhesus. Stein, S. N., and Pollock, G. H. [*Proc. Soc. Exptl. Biol. Med.*, **70**, 290–1 (1949).]

Monkeys with screw-in electrodes fitted to the head were immobilized with dihydro-β-erythroidine and treated with various concentrations of CO_2–O_2 mixture for different periods of time; then they were stimulated with threshold and super-threshold voltages. Concentrations of 20 per cent. or more of CO_2 prevented convulsions when inhaled for 3 minutes.

(III) Man. Pollock, G. H., Stein, S. N., and Gyarfas, K. [*Ibid.*, 291–2.]

CO_2–O_2 mixtures were inhaled before a super-threshold shock current was applied for 0·5 seconds. With 30 per cent. CO_2 30 seconds of inhalation sufficed

to prevent convulsions; with 15–20 per cent. mixtures slightly longer period of inhalation were required.

(*IV*) *Convulsive Phenomena. Gyarfas, K., Pollock, G. H., and Stein, S. N.* [*Ibid.*, 292–3.] L. E. GILSON (Chem. Abstr.).

Central Impairment of Sympathetic Reflexes by 8-Aminoquinolines. Moe, Gordon K., Peralta, Braulio and Seevers, Maurice H. [*J. Pharmacol. Exptl. Therap.*, **95**, 407–14 (1949).]

Pamaquine, pentaquine and isopentaquine chronically administered to dogs produce impairment of sympathetic cardiovascular reflexes, but not of parasympathetic or respiratory reflexes. The first 2 compounds cause similar effects in monkeys. It is believed that medullary cell groups are destroyed. 8-(2-(2-piperidyl)-isopropylamino)-6-methoxyquinoline (SN 13, 140) did not produce the above effects. L. E. GILSON (Chem. Abstr.).

Central Actions of Myanesin. Nicolau, Jose del Castillo. [*Arch. med. exptl. (Madrid)*, **11**, 363–85 (1948).]

Experiments on decerebrate cats show a nonspecific inhibition of spinal reflexes by myanesin (I). Flexion and crossed extension are inhibited, the latter profoundly, but the knee-jerk is unaffected. The tonic cervical reflex induced by vertical flexion of the head is inhibited to the same degree as is crossed extension. Strychnine and (I) quantitatively antagonize one another, and eserine also antagonizes (I), especially with respect to crossed extension. The powerful inhibitory action of (I) on muscular tonus can be shown on normal, lightly anesthetized and decerebrate animals. (I) acts by specifically inhibiting internuncial neurons.
MARSHALL E. DEUTSCH (Chem. Abstr.).

Some of the Effects of Myanesin in Psychiatric Patients. Schlau, Louis S., and Unna, Klaus R. [*J. Am. Med. Assoc.*, **140**, 672–3 (1949).]

Myanesin or tolserol (3-o-toloxy-1, 2-propanediol) is a muscle-relaxing drug which does not affect voluntary muscle power, is not hypnotic, and does not affect responsiveness to external stimulation. It allays anxiety without clouding consciousness. S. MORGULIS (Chem. Abstr.).

Detoxication of Barbiturates of Short Action. Donatelli, L. [*Boll. soc. ital. biol. sper.*, **24**, 461–2 (1948).]

After injection in rabbits, evipan, narconumal and eunarcon (methylated on N) were found most easily detoxicated, followed closely by pentothal; slower detoxication was noted for rectidon and pernocton (bromoallylated).
M. ELLIOTT (Chem. Abstr.).

Influence of Volatile Anesthetics and Barbiturates on Rate of Gastric Passage and Intestinal Absorption of Glucose Solutions in Rats. Cordier, D., and Chanel, J. [*Compt. rend. soc. biol.*, **142**, 1120–2 (1948).]

Et_2O, $CHCl_3$, phenobarbital and pentothal retarded passage through the stomach and absorption in the intestine. L. E. GILSON (Chem. Abstr.).

Some Pharmacological Properties of Sodium Ethyl 3, 3-Dimethylallylbarbiturate. Taylor, N. B. G., and Noble, R. L. [*Nature*, **163**, 447 (1949).]

Sodium ethyl 3, 3-dimethylallylbarbiturate (I) produces stimulation of gastric and salivary secretion of central origin when administered to dogs and cats. (I) possesses an activity opposite to that of the ordinary hypnotic barbiturates. It produces an increase in blood pressure (even on intravenous administration), immediate marked stimulation of depth and frequency of respiration, and sharp increase in body temperature. Overdose in unanesthetized animals is associated with restlessness, muscular contractions, and violent convulsions leading to death, which may be due to respiratory muscle spasm. Sympathetic overactivity may occur. The LD_{50} (cats, rabbits) is 6·7 mgm./kgm., intraperitoneally or intravenously, with 3–4 mgm./kgm. producing symptoms. Under pentobarbital (II) anesthesia cats will tolerate 50 mgm./kgm. given over 4 hours, and 5 mgm./kgm.

produces a blood pressure rise which returns rapidly to normal. Isolated gut in Ringer's solution is stimulated, then narcotized, when concentrations of 0·01 per cent. (I) are reached. During the narcosis there is no response to added acetylcholine or histamine. The use of (I) as an antidote to overcome the undesirable effects of ordinary barbiturates is suggested. Preliminary work indicates that an anesthetic mixture of (I) and (II) may be given rapidly to rabbits by the intravenous route without producing respiratory paralysis.

JOHN P. CRISPELL (Chem. Abstr.).

A Study of Mescaline in Human Subjects. Salomon, Kurt, Gabrio, Beverly Wescott, and Thale, Thomas. [*J. Pharmacol. Exptl. Therap.*, **95**, 455–9 (1949).]

Five schizophrenic and one neurotic patient were given 200–400 mgm. mescaline sulfate (I) orally, and the urinary excretion of methoxyl groups was followed quantitatively for 18 hours (method described). (I) was identified in the urine, and most of the methoxy groups were probably present as (I). Methoxyl groups belonging to breakdown products were found in a resin-like residue of the urine extract. Total methoxyl recovered in 18 hours in the urine was 9–39 per cent. of the amounts ingested. Some was still being excreted at the end of the 18-hour period. L. E. GILSON (Chem. Abstr.).

Asperula Odorata—Sweet Woodruff. Kreitmair, H. [*Pharmazie*, **4**, 140–1 (1949).]

Brief review of the pharmacology of coumarin and results of sedative and hypnotic studies in mice. H. A. WEGNER (Chem. Abstr.).

Urethan-induced Damage of the Central Nervous System. Klima, R., and Wengraf, G. [*Wien. med. Wochschr.*, **99**, 109–12 (1949).]

Among 25 patients with blood diseases (leukemia, lymphogranulomatosis, polycythemia) successfully treated with urethan, 8 exhibited psychic and neurological disorders caused by urethan-induced damage to the ganglion cell apparatus of the brain and spinal cord. In 3 autopsies extensive disseminated degenerative foci were found. ERICH HIRSCHBERG (Chem. Abstr.).

A Suicide by Oral Ingestion of 2, 4-Dinitrophenol. Duquenois, Pierre. [*Ann. med. legale, criminol., police sci., med. sociale et toxicol.*, **30**, 86–91 (1949).]

Ingestion of 1·5 gm. dinitrophenol caused death in about 24 hours. The progressive symptoms of the poisoning are described. Post-mortem analyses showed the presence of aminophenols as well as 2, 4-dinitrophenol in the viscera and urine.

ERICH HIRSCHBERG (Chem. Abstr.).

Effects of a Quaternary Base Capable of Blocking Functions of the Autonomic Nervous System. Longino, F. H., Chittum, J. R., and Grimson, K. S. [*Proc. Soc. Exptl. Biol. Med.*, **70**, 467–75 (1949).]

1, 1-diethyl-2, 6-dimethylpiperidinium bromide, previously described as a ganglionic blocking agent, has an inhibitory or blocking action on functions mediated by the sympathetic and parasympathetic divisions of the autonomic nervous system. In dog and man it reduces blood pressure and prevents several vasomotor reflexes, but does not prevent the vasopressor action of adrenaline. It causes reduction of tone of the stomach and small intestine and abolishes their motor activity, reduces gastric acidity, and causes pupillary dilation, loss of accommodation, and dryness of mucous membranes. Urecholine reverses its effect on gastric motility, and neostigmine appears to reverse all of its effects.

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

Effect of Autonomic Blocking Agents on Sweat Secretion in Cats. Patton, Harry D. [*Proc. Soc. Exptl. Biol. Med.*, **70**, 412–14 (1949).]

Footpad sweat secretion in cats is not blocked by dibenamine, even with doses 3–4 times as great as block sweating in man. Miosis, relaxation of the nictitating membrane and paralysis of the pilomotor system indicated that the dosage was adequate to block known adrenergic systems. Atropine consistently produced complete and irreversible sudomotor paralysis. Apparently the sudomotor inner-

vation in cats is entirely cholinergic and possesses no adrenergic component, as reported in man. L. E. GILSON (Chem. Abstr.).

Pharmacological Studies on the Causative Agent of Canine Hysteria. Radomski, Jack L., and Woodard, Geoffrey. [*J. Pharmacol. Exptl. Therap.*, **95**, 429–37 (1949); *cf. C. A.*, **42**, 7845*c*.]

Commercial wheat gluten (80 per cent. protein) treated with 6 gm./kgm. of NCl_3 (agene) was used. Dogs were fed 0·05–2·0 gm./kgm./day. The dose-time curve for production of running fits resembled a hyperbola. The threshold daily dose for cumulative effect was 0·07 gm./kgm./day in dogs (pure bred Dalmatians). The acute toxic dose was 3·5 gm./kgm. for dogs and 10 gm./kgm. for rabbits. The previous administration of agene-treated gluten (I) in doses sufficient to produce ataxia lowered the threshold of reaction to metrazole. This reaction had the characteristics of both canine hysteria and metrazole convulsions. Hydration from forced water intake did not provoke fits in dogs given subeffective doses of (I). Acidosis and alkalosis from administration of NH_4Cl and Na citrate had no effect on production of canine hysteria by (I). Trimethadione and diphenylhydantoin did not suppress canine hysteria, and diphenylhydantoin appeared to potentiate the production of convulsions and death by (I). Daily administration of phenobarbital antagonized the production of canine hysteria.

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

New Data on the Mechanism of Action of Sympathomimetic Substances, in particular Aleudrine. And'yan, A., Lissak, K., and Martin, I. [*Fiziol. Zhur. S.S.S.R.*, **34**, 727–38 (1948).]

Anesthetized cats with both suprarenals tied off were given adrenaline, tyramine, ephedrine ergotamine, sympathol, adrianol and aleudrine. Action on nictitating membrane and isolated heart was unchanged or enhanced by denervation; aleudrine, in contrast to other sympathomimetic substances, did not contract the nictitating membrane, but relaxed it. Ephedrine caused complete miosis. Ephedrine and tyramine, on the basis of action on the nictitating membrane and heart, act as myotropic rather than neurotropic agents. Positive inotropic and chronotropic action of aleudrine on isolated heart of cat or frog is 10 times stronger than that of adrenaline; no difference in their action on rat heart was observed. Aleudrine has no action on the peripheral circulatory system of a frog, but has a relaxing effect on the virgin cat uterus, which is 10 times stronger than that of adrenaline. G. M. KOSOLAPOFF (Chem. Abstr.).

Duration of Alcohol Action and Chronaxia. Mouriquand, G., Edel, V., and Chighizola, R. [*Bull. acad. natl. med.*, **133**, 107–12 (1949).]

The chronaxia is lowered in alcohol intoxication. Repeated doses have an additive effect. Return to normal is slower than the elimination from the blood. The nervous system remains for some time sensitized to renewed alcoholic action.

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