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The Clinical Differentiation of Senile and Arteriosclerotic Psychoses.

The clinical diagnosis of senile and arteriosclerotic psychoses is discussed on the basis of observations in 60 anatomically verified cases.

Senile psychoses tend to occur at a later age and are apt to last longer than arteriosclerotic psychoses. In the former the course was gradually progressive; in the latter a sudden onset was common and the illness was sometimes brief and stormy.

As a rule, intellectual impairment was more pronounced in senile conditions. Acute states of confusion were typical of arteriosclerotic psychoses. Unless their manner of development is known, they cannot always be distinguished from the chronic confused states associated with senile psychoses. Depressive and hypochondriacal-like symptoms were often noted in arteriosclerotic disease; they were seldom a prominent feature of senile disorders. Marked fluctuations in the severity of the intellectual disturbances were common in the former, but did not occur in the latter.

Paranoid forms of senile psychosis resembled arteriosclerotic psychoses in that the intellectual functions were usually relatively well preserved, but outspoken and chronic paranoid syndromes were not encountered in arteriosclerotic disease.

Headache, dizziness and apoplectiform phenomena were observed in a considerable number of arteriosclerotic patients, though as a rule permanent neurologic changes did not occur early in the illness. Other characteristic features of this group were syncopal attacks, convulsive seizures, explosive emotional outbursts and clinical indications of cardiac disturbances.

Peripheral (radial) sclerosis was noted with equal frequency in the two psychoses. Hypertension was almost as common in senile patients as in arteriosclerotic patients.

Anatomically pure forms of senile or arteriosclerotic disease occurred less often than mixtures of the two processes, but clinical pictures of a mixed nature were not nearly as frequent as might be anticipated from the anatomic findings.

The mistakes in diagnosis uncovered by anatomic examination are discussed. A diagnosis of senile or arteriosclerotic disease, especially the latter, is made in too many rather than in too few cases. Toxic or symptomatic psychoses are a common source of error.

(Author's abstr.)

Encephalopathia Alcoholica.

An attempt has been made to evaluate critically the results of nutritional and, more particularly, vitamin studies, as they relate to the clinical syndromes of delirium tremens, the Korsakoff psychosis, Wernicke's syndrome and the various cerebral disorders that result from a deficiency of nicotinic acid. The manner in which chronic alcoholism may produce dietary insufficiency is briefly discussed. The authors emphasize the fact that while alcohol is apparently not immediately responsible for most of the observable damage, alcoholism is responsible for these changes. The effect of alcohol *per se* has still to be evaluated. The authors accentuate also the frequency with which these pictures merge one into the other, and the fact that vitamin therapy, to be

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effective, must be instituted before irreversible structural changes have occurred. As in vitamin deficiency diseases occurring elsewhere, those affecting the nervous system are usually the result of multiple rather than single deficiencies. A well balanced diet should therefore always supplement treatment with the specifically indicated vitamin or vitamins. (Authors' abstr.)

Brain Metabolism. VIII. The Effects of Electric Shock and Some Newer Drugs.

1. Adrenalin, caffeine and cocaine, in high concentrations in the dextrose substrate, inhibit oxygen uptake, whereas nicotine has no such effect.

2. Picrotoxin, prostigmine and tyramine when injected subcutaneously into the rat tend to increase brain oxygen-uptake in plain Ringer's-phosphate.

3. Dextrose has a protective action against the depressive effect of drugs.

4. Electrical convulsions inhibit oxygen uptake; this effect is greater after repeated shocks and is more marked in the dextrose substrate.

5. Electrical convulsions and to a greater extent, insulin, interfere with oxygen uptake, in contrast to metrazol and camphor, which are metabolic stimulants. The availability or utilizability of dextrose by the brain may account for this difference. (Authors' abstr.)

Prognostic Criteria in Schizophrenia.

The prognosis is most favorable when the duration of illness is short; the type of onset acute; exogenic precipitating factors, obvious; the element of confusion and the presence of atypical symptoms (especially manic-depressive symptoms), prominent in the early symptomatology; and when process symptoms are minimal or absent. Conversely, when these conditions are reversed, the prognosis is least favorable. This group of criteria is of the greatest import in the assessment of prognosis in schizophrenia.

In general, the catatonic subtype offers the best prognosis and paranoids the worst, with simple and hebephrenic subtypes in an intermediate position. Acute cases which are so atypical as not to allow classification under any of these four subtypes are especially favorable prognostically. A progressive hospital course is ominous, whereas improvement or fluctuation is indicative of a more favorable outcome. (Authors' abstr.)

Electroencephalography in Schizophrenia.

1. The EEG tracings from 500 schizophrenic patients are compared with the records from 215 normal controls. A significantly greater percentage of borderline and abnormal records was found in the schizophrenic patients.

2. A greater percentage of the abnormal records in the schizophrenic patients was found in the catatonic-hebephrenic group.

3. There is nothing consistent or diagnostic in the character of the EEG tracings of schizophrenic patients, a great variety of records being obtained. Rapid activity is common, but is not of diagnostic significance because similar activity is found in other neuropsychiatric disorders, particularly in the manic-depressives, agitated depressions of middle age and in neurosyphilitics.

4. There is no significant relationship between schizophrenia and epilepsy on the basis of the EEG patterns. (Authors' abstr.)

Electroencephalographic Studies of Corticohypothalamic Relations in Schizophrenia.

Using hypothalamic and cortical leads electroencephalographic studies in schizophrenics give the following preliminary results:

1. Reaction to external cold is deficient in that no reactive hyperthermia results and little electrical activity in hypothalamus or cortex can be observed. The normal or neurotic shows a definite reaction in both leads.

2. Adrenalin injected intravenously causes violent subjective and objective reactions, but intramuscularly causes little subjective and usually no objective effect on hypothalamus or cortex in schizophrenics. The intramuscular injection, however, causes a prompt and striking effect in normals and neurotics electrically as well as subjectively demonstrable.

3. Electrical stimulation of the hypothalamus of schizophrenics caused minimal or no reaction in that structure demonstrable by the EEG or secondarily in the cortex. Normal and neurotics showed a profound excitation of hypothalamus and cortex, frequently with prolonged "condensor" effect, and usually subjective responses as well.

4. Verbal stimuli when applied to schizophrenics elicited no excitation in either lead, whereas excitability from both has been recorded in neurotic subjects.

5. Sodium amytal injected intravenously showed a typical hypothalamic and cortical response, more striking in the hypothalamus, which further study may correlate with the "amytal interview" effect, and be related to the selective action of barbiturates.

6. Speculations have been presented regarding the fundamental deficiency of dynamic action of the hypothalamus in schizophrenia, and its possible secondary effects in the production of the essential psychosis in the psychomotor system. (Authors' abstr.)

The Treatment of Childhood Schizophrenia by Metrazol Shock Modified by β-erythroidin.

The method of modification of the metrazol seizure by beta-erythroidin hydrochloride used

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by other investigators is here applied to the treatment of schizophrenic children, in whom the incidence of fractures sustained by unmodified metrazol convulsions appears high.

The physiological effect of the drug is discussed in its application to the diminution of strength of muscular contraction, and modification of the convulsion.

Modification of the seizures has resulted in preventing fractures in 9 of 10 cases, the tenth patient sustaining a fracture through a delayed, unmodified seizure. It is suggested that by avoidance of the use of prostigmine, the danger of this complication may be decreased. The safety of this method of modification is assured by careful technique of administration.

Clinical results show that 8 of 9 schizophrenic children treated (including two previously treated by unmodified shock) have shown improvement in response to metrazol therapy. Final evaluation of the efficacy of metrazol treatment in these cases must necessarily await further observations, to determine whether patients treated by this method show a better remission rate than the low figures for spontaneous remission indicate. (Author's abstr.)

Electroencephalographic Studies in Organic Psychoses.

Some correlation exists between the EEG findings and the clinical status of the patient in cases of general paresis, senile psychosis, psychosis with cerebral arteriosclerosis and alcoholic psychosis, Korsakoff type. This relationship is by no means an absolute one. There are cases, acute as well as chronic, in which clinically the patient shows psychotic manifestations and the EEG is normal. In acute cases of organic psychosis where the mental disorder develops rather rapidly, often slow waves can be observed, especially in patients who suffer from marked confusional states, and in cases in which the consciousness is narrowed. In acute cases with only moderate mental impairment, the EEG findings are often normal or borderline. In chronic cases some parallelism exists between the degree of deterioration and the EEG findings. Slow waves can be demonstrated in many cases. They are, however, patients of long-standing mental deterioration who did not show noticeable corticoelectric changes. It is possible that if an organic process is very slow in developing, findings are missing for some time because the brain has had an opportunity to adapt itself to the damage. Another possibility is that in some of these cases the structural changes are not so much cortical, but are localized in sub-cortical associative areas, producing inconspicuous EEG findings. The authors found that the electroencephalogram has a distinct diagnostic value in organic psychoses if definite slow waves from one to five per second were present, because in such cases a definite mental impairment was demonstrable, but if the electroencephalogram was normal this did not exclude the presence of an organic psychosis. Due to the fact that no absolute correlation exists between the electroencephalographic findings and clinical observations, the diagnostic value of this method is limited at present until we shall be able to work out a closer correlation between organic psychotic processes and corticoelectric phenomena. (Authors' abstr.)

Electroencephalograms of Manic-depressive Patients.

The EEGs of 81 manic-depressive patients of whom 52 were diagnosed as manic-depressive depressed (MDD), 22 as manic-depressive manic (MDM), and 7 as manic-depressive mixed (MD mixed) were recorded and analysed. Findings reveal that there is very little change in the EEGs when patients diagnosed as MDD or MDM shift from one phase to another except when the level of consciousness is altered.

Those patients whose EEGs show erratic slow wave disturbances are those whose behavior is unpredictable, regardless of diagnosis.

The majority of the MDD group have A or MS patterns with alpha frequencies of 10 cycles or slower. The MDM majority of the MDM group have MF patterns with alpha frequencies 10 cycles or faster.

Previous studies show a correlation between passive, dependent types of individuals and A patterns, and between aggressive, active, independent individuals and MF or B patterns.

It is the author's belief that if a person with an A pattern should develop MD psychosis he will be more apt to be diagnosed as MDD. If he has an MF or B pattern composed of fast frequencies he will be more apt to be diagnosed as MDM or as "agitated depression."

The EEGs have not yet yielded data concerning the factors responsible for the shifts in the manic-depressive phases when the level of consciousness is still unimpaired.

(Author's abstr.)

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Psychoelectric Phenomena: Electroencephalograms. New Interpretations of Alpha and Beta Waves.

Alberti's interpretation is that in the cortical ganglion cells there is a metabolic function, apparently regenerative, related to attention, and represented by the alpha waves. This is interrupted by stimulation, when a collateral (beta) process appears; less is known of the beta than of the alpha process. After excitation has continued for a certain time, the vegetative process reappears, to replace the energy expended. This is reflected by scattered alpha waves in a beta field and transient lapses of attention. The facts that the alpha process cannot be suspended for more than 12–14 seconds, is independent of mental activity and continues during sleep and narcosis are proofs of its vegetative nature. As, however, the entire cortex is never completely passive at any one time, small and irregular beta waves may be imposed on alpha waves during physical and mental rest. M. E. MORSE (Psychol. Abstr.).

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Regulation of the Treatment of Epilepsy by Synchronized Recording of Respiration and Brain Waves.

The method of synchronizing brain wave and respiration recording originally described by Cobb, Sargant and Schwab provides a quantitative check on changes in patients under various therapeutic conditions. Drugs may be more rapidly and accurately adjusted, so that the maximum anticonvulsant effects may be reached. Epileptic patients may be differentiated from normal subjects in that they have a low tolerance to convulsants. In testing patients the simplest and safest means of inducing convulsions is alkalosis. This is readily produced by hyperventilation. It is essential to measure the amount of this over-breathing in liters per minute if any sort of accuracy is to be expected in determining the tolerance of subjects to alkalosis. The basal metabolism machine affords a simple, direct and reliable means, and with the slight modifications mentioned in this and in a previous paper, is practical in its application to this measurement.

The brain wave record should be quantitated if it is to be used to measure the effect of specific amounts of hyperventilation on subjects. Spectral analysis of strips of record by the Grass-Gibbs analyzer is not feasible for most laboratories. Counting the number of abnormal waves gives only part of the answer. Amplitude and frequency must be considered. The authors have found that the delta index of Hoagland is ideal for this purpose.

The minute respiratory volume and the delta index when plotted together show clearly the effect of changes in breathing on the brain wave pattern. Such charts give an indication of the resistance of the subject to the alkalosis induced by over-breathing. The amount of this resistance can be increased by anticonvulsant drugs. This gives, therefore, a quantitative method of following the success or failure of drugs or of combinations of drugs and diet on the epilepsy of the patient.

Again, it must be emphasized that such laboratory procedures must not supersede, but must supplement, the usual clinical supervision and handling of patients. (Authors' abstr.)

Amyotrophic Lateral Sclerosis.

Forty-two cases of amyotrophic lateral sclerosis were studied histo-pathologically with reference to the origin and extent of the lesion of the upper motor neuron. Disease of the upper motor neuron originated in the giant pyramidal cells of Betz in only about one-third of the cases, while in the remaining two-thirds the pyramidal tracts became involved in the pons, the medulla oblongata or the spinal cord. The duration of the illness had no influence on the extent of involvement of the giant pyramidal cells of Betz.

The causative agent of amyotrophic lateral sclerosis has a predilection for attacking the upper motor neuron either at its point of origin (the motor cortex) or anywhere along its course, but

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chiefly in the brain-stem and the spinal cord. As further proof that the pyramidal pathways become involved anywhere along their course is the concomitant implication of other ventrolateral tracts of the spinal cord, for example, the spinothalamic and the spinocerebellar, in 21, or one-half, of the cases.

From the point of view of the lesion of the upper motor neuron, amyotrophic lateral sclerosis can be classified anatomically into forms originating in the cortex, the peduncle, the brain-stem and the spinal cord. Pathologically, 36 cases belong to the degenerative and 6 to the inflammatory type. (Author's abstr.)

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Vascular Pattern of Lesions of Multiple Sclerosis.

Sixty lesions from 5 cases of multiple sclerosis were studied by serial sections and their relation to the vascular system noted. The veins in the lesions were also compared with a group of 25 veins selected at random in the normal-appearing part of the brain in the same cases.

It was found that the lesions in the 5 cases studied were uniform so far as the number from each case would allow an opinion. When all 60 lesions were grouped together it was found that 20 were without a central vein, that 20 were oriented about a normal-appearing vein, and that only 9 were about a vein in which a thrombus could be found. In 2 lesions the vein was collapsed, and in 9 it appeared distended. In 3 of the 9 lesions which had a thrombosed vessel the thrombus was completely outside the lesion; in 3 others the thrombus was partly inside and partly outside the lesion, and in only 3 could it be said that the thrombus coincided with the lesion. The occurrence of a thrombus in one of these was questionable, as the actual site of the thrombus was missing and its presence was inferred from the state of the vessel proximally and distally.

No positive correlation was found between the lesions with a thrombus and the peculiarities of size or shape of the lesion, the degree of demyelination or the phagocytic cellular response. No positive correlation was found between the size or shape of the lesion and the presence or absence of a vein except that for some unexplained reason 4 out of the 5 irregular lesions were without central veins. When a vein was found within an ellipsoid or a cylindric lesion it was almost invariably oriented along the long axis of the lesion. There was a strong positive correlation between the perivascular cellular reaction of the central vein and the presence of a thrombus wholly or partly within the lesion.

The view that the demyelination in this disease is entirely unrelated to the vascular system does not seem tenable in the light of the foregoing observations. The view that the vascular changes, particularly thromboses, are an essential part of the pathogenesis of the lesion plaque

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seems equally untenable unless one accepts the assumption that venous thrombosis can disappear without trace, and at the same time exist long enough to cause permanent demyelination.

It is tentatively suggested that the thrombi observed are the result of the local absorption of thromboplastic substances released by the rapid breakdown of the myelin sheaths.

(Authors' abstr.)

Biochemical Disturbances in Mental Disorders. 1. Anti-insulin Effect of Blood in Cases of Schizophrenia.

Substantiation of a biochemical disturbance in schizophrenia has been presented. This disturbance indicates the presence of an anti-insulin substance in the blood of schizophrenic patients.

This disturbance or failure of the organism to adjust this disturbance may be one of the etiologic factors in schizophrenia.

A blood-sugar curve has been plotted which under the experimental conditions outlined is possibly significant for one form of schizophrenia and may be used as a biologic test.

A subdivision of schizophrenia into two pathogenically different forms is indicated.

(Authors' abstr.)

Vasospasm and Focal Cerebral Ischemia.

The effects of mechanical and electrical stimulation on the pial vessels of 30 cats, 1 dog and 4 monkeys have been studied.

The pial vessels in cats contract vigorously on mechanical or electrical stimulation.

The dog's pial vessels are not as sensitive to this type of stimulation as the cat's; the monkey's are less so than the dog's.

Such evidence as there is suggests that these spasms are not dependent on a neurovascular mechanism. They resemble closely the local spasms in peripheral vessels described by Lewis and others as muscular, and not neuromuscular. They are localized to the site of stimulation and are not propagated, as are the neurovascular reactions described by Ricker and Chase in the mesenteric vessels of rabbits.

Mechanical stretch of the vessel wall appeared to be an adequate stimulus to produce vasoconstriction. This is direct evidence that the tone of a pial vessel, at least in the cat, may be partly maintained by a purely local mechanism, i.e. endovascular pressure (or stretch).

The effect of acetylcholine, amyl nitrate and carbon dioxide on constrictions in pial vessels (produced by local stimulation) was studied.

A method is described whereby temporary focal cerebral ischemia resulting from vasospasm may be produced in animals.

Diminished blood flow in the cortex, as a result of vasoconstriction of pial vessels, has been demonstrated by thermoelectric blood flow recording. Areas of focal cerebral ischemia, following constriction of pial vessels by electrical or mechanical stimulation, have been shown to be present by intravital staining. It has been demonstrated that these areas of focal ischemia are frequently wedge-shaped, and that this is due to the anatomic distribution of the cortical vessels.

No clinical evidence was found that temporary focal cerebral ischemia resulting from vasospasm in the temporal, parietal or occipital cortex of cats recovered from ether anesthesia gave rise to any epileptiform neuronal discharge. That subclinical neuronal discharges occurred as the result of ischemia could not be ruled out with the methods employed.

That electrical stimulation causes focal cerebral ischemia through spasm of vessels should be taken into consideration in certain physiologic problems in which the effect of electrical stimulation on cortical nerve cells is being studied.

A theoretic discussion of the findings with regard to their applicability to human vessels is presented, especially in relation to their possible significance in electrical stimulation of the brain at operation, in electric shock and in trauma to the head. (Author's abstr.)

Avian Thiamine Deficiency.

Using dietary methods described before and staining methods which demonstrate the neurofibrillar structure of the neuron, the following observations were confirmed: (1) The first neuronal histologic change in thiamine-deficient pigeons is degeneration of the distal part of the axon, and changes in myelin are secondary to this; (2) degeneration proceeds centralward and is accompanied by slight shrinkage or sclerosis of the cell body; (3) the large nerve fibers degenerate first and the medium later, the small fibers usually remaining intact; and (4) opisthotonos may not be attended by any definite neurologic lesions. In the present study the early axonal changes were shown to consist of thickening of the neurofibrils and generalized swelling, followed by the formation of varicosities, in the distal parts of cell processes. This is followed by fragmentation.

Closer study of opisthotonos in acutely deficient birds revealed that it may be preceded by lateral rotation of the head similar to that which results when one labyrinth (utricle) is destroyed in a normal bird. This is succeeded by phasic extension of the neck and then the head. This may be accompanied by very slight or no degenerative changes in the vestibular system or elsewhere in very acutely deficient birds, but in pigeons with moderately acute deficiency degeneration occurs in the sensorial epitheliums of the labyrinth, especially of the posterior crista, and in

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the central endings of the vestibular nerve fibers, especially in the lateral part of the nucleus magnocellularis. It is concluded that opisthotonos is due to a selective release of the central vestibular centers from labyrinthine control. Because of its clinical characteristic enopisthotonos is thought to be due to still further impairment of the vestibular nerves.

In chronically deficient pigeons without opisthotonus all central and peripheral terminations of the vestibular nerves are moderately degenerated; no preponderant site of degeneration is present.

In both acutely and chronically deficient pigeons many basket terminations and moss fibers in the cerebellum degenerated, and in several mildly chronic (subacute) preparations this was associated with staggering (cerebellar ataxia). This is quite distinct from "locomotor ataxia" which occurs in very chronic experiments as the result of degeneration of the very large proprioceptive nerve fibers in the peripheral nerves. Also, in both acute and chronic experiments inconsistent degeneration was present in the optic lobes, the nucleus rotundus of the thalamus, the reticular formation and the secondary vestibulocochlear centers, and in chronic experiments, in the cell bodies and peripheral nerves of the third and fourth cranial nerves and elsewhere.

The following generalization may be made: The neurologic manifestations of an acute or a chronic deficiency in thiamine are accompanied first by impaired function and then by degeneration of the primary neurons of the proprioceptive nervous system and the central terminations of the optic nerves. Changes occur next in internuncial neurons of these same systems. Finally, after prolongation of the deficiency, the efferent nervous system becomes affected.

On the basis of curative experiments, neurons affected histologically by thiamine lack were divided into those which (1) return to normal when thiamine is given and (2) those which continue to degenerate. It appeared that many of those suffering from early degenerative changes belong definitely to the first group. Although fibers which had fragmented probably continue to degenerate, evidences of regeneration were seen in the cerebellar basket terminations.

Present observations indicate that brain hemorrhages in thiamine-deficient pigeons were secondary to changes in the axons and cell bodies. These hemorrhages appear to have no relation to the clinical behavior. (Author's abstr.)

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Cerebral Electrical Changes in Experimental Concussion.

Local and general cerebral trauma has been produced in cats under light anaesthesia. Continuous electro-encephalograms were recorded throughout the experiments from points in both hemispheres by means of electrodes fixed into the skull, in contact with the dura mater. Records of cardiac and respiratory changes were also made and alteration in reflex activity and spontaneous movement were noted.

Blows resulting in concussion always caused an instantaneous and generalized diminution in the cerebral electrical activity as recorded in the electro-encephalogram, which persisted after reflex activity had returned. There was then a delayed appearance of abnormally slow waves similar to those seen after concussion in man. Reflex activity returned before these abnormally slow waves were observed.

The cerebral electrical changes caused by local trauma were limited to the injured area, but they were similar to the generalized changes seen in concussion.

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No relationship was found between the cerebral electrical changes and the alterations in the cardio-respiratory mechanism or in reflex activity.

It is concluded that:

(1) Concussion is associated with diminution or cessation of the electrical activity of the whole cerebral hemispheres, as recorded in the electro-encephalogram.

(2) The delayed appearance of abnormally slow waves of cerebral electrical potential change may represent a stage in recovery from concussion.

(3) The reversible changes caused by local and general cerebral trauma are identical.

(4) Concussion is the direct result of mechanical violence to cerebral cells. It is not dependent upon secondary changes such as oedema, anoxia or hypoglycaemia for its initiation.

(Authors' abstr.)

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Electroencephalographic Individual Differences and Their Constancy. I. *During Sleep.*

Alpha rhythms were recorded in 20 male adult subjects, 5 representing each of the 4 types known as rare, mixed, subdominant, and dominant alpha, (1) under the standard waking condition of minimal activity, (2) during all-night sleep, and (3) during psychological activity of diverse types. The fact that this population was heterogeneous with respect to waking alpha index (the percentage of time an alpha rhythm was present on the record) prior to sleep, but homogeneous with respect to all sleep rhythms, and not heterogeneous as before sleep as regards waking alpha index after sleep, is taken to signify that individual differences in regard to alpha characteristics are not to be ascribed merely to a fixed inborn cortical organization.

M. J. ZIGLER (Psychol. Abstr.).

Converting a Spinal CR into a Reflex.

Dogs between 2 months and 3 years old were conditioned to respond with a flexion of one semitendinosus muscle to a slight shock to the tip of the tail. The response was extinguished, then reconditioned. Then the spinal cord was transected just above the level at which the 3rd lumbar roots emerge from the dura, and all roots posterior to the transection were severed except the ipsilateral 2nd or 3rd sacral dorsal and 7th lumbar dorsal and ventral, and the contralateral 6th and 7th lumbar ventral. As the two contralateral roots were severed one after the other, the CR progressively exhibited the character of a spinal reflex, i.e. it was impossible to extinguish it. The conversion of the spinal CR into a reflex is assumed to depend on the surgical removal of an inhibitory influence which normally is exerted upon the response mechanism through the contralateral ventral roots. M. J. ZIGLER (Psychol. Abstr.).

Electroencephalographic Individual Differences and Their Constancy. II. During Waking.

Samples of electroencephalic records 50 cm. in length were taken from the left motor and left occipital area of 4 subjects, who were selected as typifying the 4 groups which are often distinguished in terms of the percentage of time alpha waves are exhibited on the record. Samples were secured under experimental conditions involving a variety of psychological activities (resting, reading aloud and silent, talking, rotation, etc.), and statistical methods of analysis of variance and covariance were employed to determine whether individual differences in the alpha index of the electroencephalograms suffer changes and tend to disappear during various types of mental activity. In some instances in combination with visual stimulation, inter-individual differences were found to be reduced to a magnitude not significantly in excess of intra-individual differences. Even where heterogeneity was exhibited, it was found to be an expression of a significantly different distribution of alpha indices from that evinced in the resting condition. M. J. ZIGLER (Psychol. Abstr.).

Experimental Data on Different Neural Mechanisms for Learned and Unlearned Responses.

Dogs subjected to many consecutive hours of massed training by the familiar buzz-shock conditioning method were found temporarily to fall asleep to the conditioned stimulus but to remain awake to the unconditioned stimulus. Animals conditioned after injections of a depressing drug (nembutal) also temporarily fail to respond to a well-established conditioned stimulus, while the unconditioned stimulus is still effective. It was found that an unconditioned shock of one rear foot occasioned a response by the shocked foot combined with a tendency to lift

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the contralateral front foot; however, this contralateral effect tends not to express itself in the conditioned response. These observations are interpreted to signify that the neural mechanisms for learned (conditioned) and unlearned (unconditioned) flexion responses are different. Although the conventional view that the cerebral cortex is the neural basis for learning is here evaluated as correct in part, the emphasis in this study is that learning in the case of flexion conditioning depends upon an extremely complicated integration among nerve centers, including the cerebral cortex, the basal ganglia, portions of the medulla oblongata, the nucleus dorsalis of the spinal cord, and other relevant nerve centers. M. J. ZIGLER (Psychol. Abstr.).

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Two Cases of Myelodysplasia.

Myelodysplasia was first described by Alfred Fuchs in 1909 as a congenital state of defective development of the spinal cord due to incomplete closure of the embryonic neural tube. The principal symptoms are trophic and sensory disturbances of the legs, generally on one side. The syndrome is often mistaken for spina bifida or syringomyelia. Two cases are presented.
S. M. COLEMAN.

A Study of Migraine.

From the evidence presented it is concluded that the migraine syndrome is not pathological, but is a hyperfunctioning of the effort syndrome. This hyperfunctioning tends to increase the acidity of the system, as indicated by the increased elimination of acid by the kidneys. The C/N quotient in urine samples increases a very considerable amount during severe headaches. A stasis of the kidneys occurs during the migraine episode, along with a general stasis of the abdominal organs.

It is suggested that the tendency to migraine is due to the inheritance of an imbalance in the sympathetico-adrenal mechanism; migraine attacks follow periods of great uncertainty, while periods without mental strain are also periods free from headaches.

The evidence indicates that creatinine, urea, uric acid, the acetone bodies, reducing sugars, the phenols and probably ammonia play no specific role in the migraine syndrome.
S. M. COLEMAN.

Complications in Metrazol Treatment.

Besides psychomotor phenomena, such as homicidal, excited or destructive behaviour, psychotic exacerbations and neurological involvement, which is rare, there are a number of somatic complications. These include spinal fracture, dislocations, pulmonary involvement, thrombosis of veins and mouth injuries. Psychomotor releases and psychotic exacerbations are not uncommon and may endanger the patient and those about him. For the above reasons the practice of giving the treatment at home is deprecated. Metrazol is contraindicated in paranoid patients. S. M. COLEMAN.

Angioma Simplex of the Pons.

Two instances of solitary angioma simplex of the pons are reported with histological findings. The relative frequency of such conditions and their association with like malformations in the liver are stressed. S. M. COLEMAN.

J. NEUROL. PSYCHIAT.

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A Congenital Form of Amaurotic Family Idiocy.

An unusual form of lipoidosis is described in an 18-day-old female infant, dying from bilateral haemorrhages into the renal pyramids. At post-mortem the brain was found to be considerably reduced in size and weight (87 gm.) and there was retarded convolutional development in the frontal lobes. Microscopically the nerve cells showed swelling of the amaurotic family idiocy type, the intracellular lipoid being resistive to solvents. Numerous atypical nerve cells showing extreme distension with this material were present throughout the cerebral and cerebellar cortices. Lipoid abnormalities were conspicuous in the glial cells. Myelinization was retarded throughout the nervous system, and heavy deposits of extracellular doubly-refractile crystals of cholesterol esters were present in the white matter of the hemispheres and cerebellum. There was a severe olivocerebellar atrophy.

Microscopical examination of the viscera showed evidence of lipoid storage in the reticulo-endothelial system, the histochemical reactions of the deposited substance resembling those of the nerve cell granules.

Several points of difference between this condition and Tay-Sachs' disease are discussed, and it is concluded that the case is a hitherto undescribed variety of lipoidosis for which the name "congenital amaurotic family idiocy" is suggested. (Authors' abstr.)

The Significance of an Abnormal Electroencephalogram.

The electroencephalograms of 900 subjects, divided into groups of highly selected normals, normals, psychoneurotics, epileptics, and subjects of a post-traumatic syndrome have been examined. The percentage abnormality has been determined, the subjects being at rest and having over-breathed, upon the basis of arbitrary norms which have been described.

The abnormality rate varied from 5 per cent. in the highly selected normal group to 60 per cent. in the epileptic. 75 per cent. of the subjects with an abnormal response to over-breathing showed abnormality in the resting EEG. The percentage abnormality in epilepsy ranged from 55 in subjects of *grand mal* to 90 in patients with more than one type of fit. It is suggested that a non-specific diagnosis of epilepsy should be made upon the abnormalities seen in the EEG at rest and after over-breathing, but that the recognition of the specific type of fit should be based on clinical observation.

On the basis of the present results and those of other workers it is concluded that:

(1) An abnormal EEG. in an otherwise normal subject is strong evidence of an inborn constitutional abnormality involving the central nervous system.

(2) This abnormality appears to be non-specific and may manifest itself in the subject or his offspring as a behaviour disturbance which may be psychoneurotic, psychopathic, psychotic, or epileptic in type.

(3) On the basis of a norm which excludes 10 per cent. of apparently normal subjects, 40 per cent. of epileptics have a normal EEG. between fits, but the incidence of abnormality in epileptics showing more than one type of clinical fit was 90 per cent. The significance of this figure is discussed in relation to the findings of other investigators. (Author's abstr.)

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The Acoustic Area of the Monkey (Macaca mulatta).

By the method of recording cortical response to click stimulation, the acoustic cortex of the monkey has been mapped. The area responsive to clicks is the portion of the superior face of the superior temporal gyrus included in the angle between the posterior and medial borders of the gyrus. It varies in extent from 8 to 12 mm., and is consistent from one animal to another. (Authors' abstr.)

The Effect of Section of the Medial Lemniscus on Proprioceptive Functions in Chimpanzees and Monkeys.

1. Section of the medial lemniscus in monkeys and chimpanzees did not produce an enduring loss of proprioceptive skill in the ability of trained animals to discriminate weights.

2. Primary section of the superior cerebellar peduncle carrying dentato-thalamic fibers produced no significant alteration in weight discrimination.

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3. When lesions of the medial lemniscus also involved the crossed dentato-rubro-thalamic tract, there was a marked permanent loss in general proprioceptive function and the ability to discriminate weights.

4. It is suggested that the cerebellar projections to the thalamus may subserve the recovery of proprioceptive function following interruption of the medial lemniscus.

(Authors' abstr.)

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*Retrograde Amnesia and Thixotropy. *Roosen, R.* 374

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Retrograde Amnesia and Thixotropy.

Thixotropy is the quality, which many colloids have, of undergoing transient liquefaction under mechanical influences, especially shaking. According to students of colloids, the cell contents can be influenced thixotropically. Roosen presents a hypothesis of the mechanical production of retrograde amnesia, particularly after concussion, in this manner. In addition to other injuries due to concussion, the cortical cells are changed transiently into a more fluid state, which creates an energic situation more favorable to ekphoria of older engrams than to manifestation of memory traces of the immediate past. Normal conditions return when the thixotropic effect has passed off. Nerve cell fluidity occurs also, but less strikingly and strongly, when oxidative processes are insufficient and acid metabolic products accumulate. This may also produce retrograde amnesia. M. E. MORSE (Psychol. Abstr.).

NEUROBIOL.

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The Transfer of Training.

Seven different interpretations are examined, and 3 (from the 167 experiments between 1890 and 1935) are appraised: Sleight's on memory, Thorndike's on thinking power, and Cox's on manual tasks. The significant conclusions from these that are corroborated by other experiments are: (1) The individual's awareness of the usable common element is effective in producing transfer. (2) The use of specifics, instead of such generalities as Latin and arithmetic, is needed in studying the problem of transfer. (3) Transfer takes place on the ideational, not on the neuromuscular level, and training can be generalized only by being explicit. The theoretical issues of the problem are: (1) By which process is transfer effected? "Identical elements" is ruled out because of confusion of meaning of each term; "generalization" is accepted because it reduces an unknown process to one with which we are already familiar. (2) In which manner do explicit generalizations produce transfer? The individuality of mind precludes the adoption of results from factor analyses; introspective reports are the most fertile approach. The educational inference is that the child should be regarded as an entering member of society; individual needs must be related to the demands of societal patterns.

H. MOORE (Psychol. Abstr.).

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A New Application of Conchology.

In this paper the suitability of shell collecting, as a form of occupational therapy for certain border-line psychiatric patients, is discussed. There is an interesting introduction to conchology and a useful bibliography. S. M. COLEMAN.

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1. Biochemistry, Pathology, etc.

Studies in Nicotinic Acid Metabolism. I. *Fate of Nicotinic Acid in Man. Sarett, Herbert P., Huff, Jesse W., Perlzweig, Wm. A., Stenhouse, Molly, and Forth, Rachel.* [*J. Nutrition*, **23**, 23–34 (1942); *cf. C.A.*, **35**, 777[9].]

Trigonelline and nicotinuric acid are the two known end-products of nicotinic acid metabolism found in human urine. For a study of normal trigonelline excretion in man, a diet free of coffee, peas, beans and other legumes is necessary. On such a diet 3 men excreted 9–13 mgm. of trigonelline daily. The normal daily excretion of acid-hydrolyzable nicotinic acid derivatives (I) by 36 subjects on different diets and under fasting conditions was 0·7–2·2 mgm. This level was unaffected by smoking. There is a constant loss of at least 10 mgm. of known (I) in the urine daily, regardless of diet. Of 200-mgm. oral doses of nicotinic acid, 25–90 mgm. could be accounted for by the increase in urinary trigonelline and nicotinuric acid. Trigonelline accounted for 55–84 per cent. of this increase. The feeding of glycine and choline with 200-mgm. doses of nicotinic acid did not change the proportions of trigonelline and nicotinuric acid excreted. Of 200-mgm. doses of nicotinamide, 36–60 mgm. could be accounted for by the increase in urinary trigonelline. There was no increased excretion in the acid-hydrolyzable nicotinic acid fractions. Trigonelline is not utilized by man when taken orally and is excreted almost completely as such. Nicotinuric acid when ingested is excreted to the extent of about 60 per cent. unchanged, and without an accompanying increase in trigonelline. When injected intravenously the recovery in the urine ranged from 94 to 97 per cent. After 6 daily doses of 500 mgm. of nicotinamide the urinary excretion of (I) and trigonelline in 6 subjects accounted for 27–42 per cent. of the ingested dose, compared to 5–21 per cent. which could be accounted for after the first day. Over 90 per cent. of this added excretion was in the form of trigonelline. Of the remaining (I) fraction, 65–80 per cent. was nicotinuric acid. The extra excretion of (I) and trigonelline after a test dose of nicotinamide may serve as an indication of the nutritional status of an individual with respect to nicotinic acid.

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II. *Fate of Nicotinic Acid in Normal and in Blacktongue Dogs. Sarett, Herbert P., and Stenhouse, Molly.* [*Ibid.*, 35–45.]

A study of the excretion of (I) and trigonelline in dogs at different levels of nicotinic acid intake shows that trigonelline and nicotinuric acid are the main end-products in urine. On a low-nicotinic acid diet or in blacktongue several 25-mgm. doses are required before the dog will excrete part of the dose. The urinary excretion of trigonelline falls to 1·1–2·7 mgm. per day on a low-nicotinic acid diet, and to 0·1 mgm. in the blacktongue dog. On a high nicotinic acid diet of 1·1 mgm. per kgm. dogs excrete a total of about 7 mgm. of the 12–15 mgm. that they ingest daily. Over 90 per cent. of this is in the form of trigonelline. After saturation with nicotinic acid, doses of 100 mgm. of this compound or nicotinamide are completely excreted in the form of trigonelline (75–94 per cent.) and nicotinuric acid (6–25 per cent.). The addition of glycine and choline does not influence the proportions excreted as nicotinuric acid or as trigonelline. Neither trigonelline nor nicotinuric acid can be utilized by the dog.

C. R. FELLERS (Chem. Abstr.).

Nicotinic Acid and Coenzyme Content of the Tissues of Normal and Blacktongue Dogs. Dann, W. J., and Handler, Philip. [*J. Nutrition*, **22**, 409–14 (1941); *cf. C.A.*, **34**, 2041[3].]

There is a significant decrease from the normal in the coenzyme content of the liver and muscle of blacktongue dogs. As it seems unlikely that the loss of coenzymes I and II from the body is the direct cause of the onset of pellagra or blacktongue and of death from these diseases, the question of the immediate cause of death remains unsolved. Observation of the experimental animals suggests that dehydration or the toxic reactions of the inflammation after the invasion of the alimentary mucous membranes may be the cause. C. R. FELLERS (Chem. Abstr.).

Pellagra and Mental Disease. Löfvendahl, Hillevi. [*Acta Med. Scand.*, **108**, 455–65 (1941).]

In 58 out of a total of 85 insane patients examined, the urinary nicotinic acid excretion was at about the lowest normal range (1·2 mgm.), while 20 patients excreted no nicotinic acid. In some instances treatment with nicotinic amide gave good results.

S. MORGULIS (Chem. Abstr.).

The Chemical Diagnosis of Pellagra (Nicotinic Acid Deficiency). Field, Henry, jun., Melnick, Daniel, Robinson, William D., and Wilkinson, Charles F., jun. [*J. Clin. Investigation*, **20**, 379–86 (1941).]

Previous work on the value of the determination of nicotinic acid compounds in urine or blood as a measure of nicotinic acid nutrition is conflicting and inconclusive. In the present report no differences were observed in the basal blood or plasma nicotinic acid contents of normal and deficient persons. Five types of blood nicotinic or nicotinamide tolerance curves showed no differences between normal and deficient subjects. An average but not a constant decrease in urinary excretion of substances reacting like nicotinamide was observed in deficient persons. A more marked and consistent decrease in excretion of trigonelline was found. With an accurately standardized experimental technique the urinary excretion of trigonelline may be a measure of nicotinic acid nutrition. J. B. BROWN (Chem. Abstr.).

Changes in the Nervous System and the Striated Muscles of the Adult Rat in Avitaminosis E. Monnier, Marcel. [*Z. Vitaminforsch.*, **11**, 235–58 (1941).]

Adult rats on a vitamin E free ration develop neurological symptoms in 10 months, a complete syndrome in 13–14 months, and may suffer spontaneous death in 14–23 months. The manifestations are ataxia, spastic paresis, amyotrophy, disturbed co-ordination, decreased general and special sensitivity, psychomotor and vegetative disorders. Histological changes include degeneration of the dorsal tracts of the cord, sclerosis of the ganglion cells of the ventral horn and the vegetative cells of the lateral column, slight demyelinization of the spinal roots, peripheral nerves and their endings in the muscles. Changes in the muscle (diffuse myodegeneration) appear by the tenth month. The syndrome involves both nerve and muscle, and is related to the pictures of tabes, chronic poliomyelitis, polyradiculoneuritis, myelopathic and myopathic progressive muscular atrophy, and amyotrophic lateral sclerosis.

JOSEPH S. HEPBURN (Chem. Abstr.).

Experimental Brain Tumors. (I) Tumors Produced with Methylcholanthrene. Zimmerman, H. M., and Arnold, Hildegarde. [*Cancer Research*, **1**, 919–38 (1941).]

Pellets of 20-methylcholanthrene were implanted in the cerebral meninges, the right cerebral hemisphere and the cerebellum of 103 male C3H mice. In all, 48 tumors were produced: 25 gliomas, 13 sarcomas, 7 mixed sarcomas and gliomas and 3 unclassified.

FELIX SAUNDERS (Chem. Abstr.).

Experimental Demyelination of the Central Nervous System. II. Respiratory Enzyme Systems of the Brain in Poisoning with Cyanide and with Azide. Wyndham, R. A. [*Australian J. Exptl. Biol. Med. Sci.*, **19**, 243–8 (1941); *cf. C.A.* **35**, 1511[5].]

Measurements of succinic dehydrogenase, cytochrome oxidase and catalase were made in rabbit brain tissue in vitro. Incubation of the tissues with CN produces no irreversible effect on the first two. Tissue from rabbits poisoned with CN showed no variations in enzyme activity

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related to intensity of CN dosage. In monkeys CN and azide produced no change in succinic dehydrogenase or catalase activities; with prolonged treatment there was a diminished cytochrome oxidase activity, chiefly in the grey matter. Conclusion: There is no evidence that demyelination in the cerebral hemispheres of monkeys is due to permanent inactivation of the respiratory enzymes. EDWARD EAGLE (Chem. Abstr.).

Action of Electrolytes on the Metabolism of Brain. *Noyons, A. K. M., and van Goor, H.* [*Acta Brevia Neerland. Physiol., Pharmacol., Microbiol.*, **11**, 59–65 (1941).]

The effect of the ions NH, K, Na, Li, Ca, Mg and Ba on the metabolism and R.Q. of guinea-pig brain was studied. The first four ions increase the R.Q. and the last three decrease it. The effect is compared with that of these ions on gelatin membranes and is caused by alteration of the permeability of the brain cells. M. L. C. BERNHEIM (Chem. Abstr.).

Glycolysis and Phosphorylation in Brain Extracts. *Ochoa, Severo.* [*J. Biol. Chem.*, **141**, 245–51 (1941); *cf. Geiger, C.A.*, **34**, 5517⁹.]

In the presence of Mg ions, inorganic phosphate, adenosine triphosphate and cozymase, dilute rat brain extracts form lactic acid at a high rate (30–40 mgm. in 90 minutes per gm. of fresh brain) from added glucose and hexosemono- or diphosphate. Dismutation between triose phosphate and pyruvic acid is markedly accelerated in the presence of a phosphate acceptor such as creatine or glucose; in this case inorganic phosphate is esterified with formation of either phosphocreatine or hexose diphosphate. In aqueous extracts of dry Me_2CO powders of brain, adenosine triphosphatase is completely inhibited by fluoride and the labile phosphate of adenosine triphosphate is transferred to glucose without liberation of inorganic phosphate (hexokinase reaction). A. P. LOTHROP (Chem. Abstr.).

Conditioned Reflex Hypoglycaemia, Glucosuria and Hyperglycaemia. *Savchenko, V. A.* [*Bull. biol. méd. exptl. U.R.S.S.*, **9**, 361–3 (1940) (*in English*).]

Insulin (1) and adrenaline (2) were injected into dogs as unconditioned stimuli while at the same time a loud whistle was blown as a conditioned stimulus. The reaction observed was the change in blood sugar. The injection of 0·5 units of (1), together with the use of the whistle, on four separate days, with intervals of 2–3 days, gave decreases in blood sugar in 90 minutes of 64 to 38, 93 to 34, 97 to 13 and 110 to 21 mgm. per cent. respectively.

Two days later an "empty" injection, without administration of (1), but accompanied by the whistle, caused a decrease in blood sugar from 91 to 11 mgm. per cent. in 90 minutes. The injection of (2) together with the use of the whistle caused the appearance of sugar in the urine. After six injections of (2) a positive sugar test could be obtained by the use of the conditioned stimulus alone. S. A. KARJALA (Chem. Abstr.).

Blood Cholesterol in Asphyxia. The Cerebral Origin of Cholesterinaemia. *Petyunin, I. A.* [*Bull. biol. méd. exptl. U.R.S.S.*, **9**, 466–7 (1940) (*in English*).]

Cholesterol (1) determinations were made in the arterial blood and that flowing from the brain of dogs during progressively increasing anoxaemia caused by diminution of the lumen of the trachea as a result of a gradually increasing pressure of 3 hours. A rapid rise in (1) generally occurred, and the venous blood was invariably higher in (1) than was arterial blood by 10–64 mgm. per cent. This indicates an increased mobilization of (1) from the lipoid depot of the brain as a result of the decreased O supply to the brain. The (1) level begins to fall 1–2 hours after the beginning of the experiment and at the same time the (1) level of arterial blood becomes higher than that of venous blood, which indicates retention of (1) by the brain. A drop in (1) values to normal or to 9–35 mgm. per cent. below normal during the experimental period was accompanied by profound changes in the nerve cells and neuroglia cells of the brain.

S. A. KARJALA (Chem. Abstr.).

Carbohydrate Metabolism in Experimental Epilepsy. *Fedorov, I. I.* [*Bull. biol. méd. exptl. U.R.S.S.*, **9**, 372–5 (1940) (*in German*).]

The experimental development of epilepsy in dogs, cats and rats by subarachnoidal injection of 0·2 ml. of 2 per cent. gallic acid, by intraperitoneal injection of 8–10 ml. of 2 per cent. oil of camphor, or by means of an electric current, causes a hyperglycaemia which reaches a maximum in 1–5 hours, falls to normal in approximately 25 hours and then gives rise to a marked hypoglycaemia. The source of the hyperglycaemia was found to be the rapid mobilization and breakdown of glycogen by the liver. A marked increase in blood lactic acid occurs during epilepsy, which remains considerably above normal for over 25 hours.

S. A. KARJALA (Chem. Abstr.).

The Phosphorus Fraction in Blood and Muscles in Experimental Epilepsy. *Averbukh, B. M.* [*Bull. biol. méd. exptl. U.R.S.S.*, **9**, 380–2 (1940) (*in German*).]

Experimental epilepsy induced in cats by camphor or bile caused an increase in inorganic P from 5·0 to 13·8 mgm. per cent. (average of 13 experiments). An average of 10 experiments gave increases of orthophosphoric acid from 4·86 to 12·3 mgm. per cent. in the blood and from 27·2 to 40·3 in the hind muscles and from 27·2 to 59·3 mgm. per cent. in the fore muscles respec-

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tively. Creatinephosphoric acid increased from 28·4 to 31·6 in the hind and decreased to 6·97 mgm. per cent. in the fore muscles. In each case the fore muscles were exercised while the hind muscles were at rest. S. A. KARJALA (Chem. Abstr.).

Peculiarities in the Development of Cholesterinaemia in Experimental Mechanical Jaundice. The Cerebral Origin of Cholesterinaemia. Petyunin, I. A. [*Bull. biol. méd. exptl. U.R.S.S.*, **9**, 463–5 (1940) (*in English*).]

Ligation of the common bile duct in 6 dogs caused a 3- to 4-fold increase in blood cholesterol (1) in 8–12 days. The level returned to normal in 4 dogs in 16, 30, 36 and 51 days respectively, while in 2 cases the level fell to 55–65 mgm. per cent. (50–75 per cent. below normal) with resultant death. This decrease cannot be due to improved elimination of (1), since it was accompanied by acholia and an increase in blood bilirubin to 39 mgm. per cent. During maximal cholesterinaemia the blood from the jugular vein had 100 mgm. per cent. or more (1) than the blood from the femoral artery. During the decrease in blood (1) this difference is less marked, and frequently the venous blood flowing from the brain was lower in (1) than was the arterial blood. The lethal cases showed marked degenerative changes in the cells of the cerebral cortex and the neuroglia. S. A. KARJALA (Chem. Abstr.).

Chemical Studies in Delirium Tremens. Cohn, Clarence. [*J. Lab. Clin. Med.*, **27**, 63–7 (1941).]

The following chemical determinations were made on the blood and spinal fluid of male patients with the diagnosis of delirium tremens before and after treatment: blood sugar, blood urea N, hemoglobin, hematocrit values, serum protein, serum chloride, total base, CO combining power, available fluid by the method utilizing NaCNS and spinal fluid chloride, protein, sugar and total base. The clinical evidence and the results of these determinations indicated the presence of dehydration. Before treatment, haemoconcentration, hypochloraemia, lowered alkaline reserve and some evidence of a decrease in total base were found. The volume of fluid available for the solution of thiocyanate was below the normal. The treatment, which resulted in recovery of the patient and caused restoration of fluid and salt, varied, but in general it consisted of NaCl, fluids, thiamine and nicotinic acid administered orally and parenterally. Blood lactic acid, pyruvate and acetoacetic acid determinations were made on one case. The results, which were only slightly above normal, together with the chloride, bicarbonate and protein determinations left an apparent anion deficit that suggested the presence of other organic anions. Changes in the spinal fluid suggested the occurrence of injury to the central nervous system in certain patients. H. W. ROBINSON (Chem. Abstr.).

Specific Pulmonary Alterations in Tuberous Sclerosis. Vejlens, Gert. [*Acta Path. Microbiol. Scand.*, **18**, 317–30 (1941).]

Tuberous sclerosis of the brain may cause a peculiar cystic pulmonary degeneration, with a myomatous proliferation of the septa of the cysts which is tumour-like. This is designated "tuberous sclerosis of the lungs." The genesis of the disease is not clear, but co-present alterations in the histology of the endocrine glands suggest that it is due to a disturbance of the equilibrium of the endocrine system. It is congenital. JOHN T. MYERS (Chem. Abstr.).

The Determination of Cerebrosides. Brand, Florence C., and Sperry, Warren M. [*J. Biol. Chem.*, **141**, 545–53 (1941).]

Difficulties are encountered in the hydrolysis of cerebrosides when carried out according to published procedures for their determination. It is satisfactorily accomplished by the use of 3 N HCl and heating for 45 minutes. The galactose is determinated by the method of Miller and Van Slyke (*C.A.*, **30**, 6407) by direct titration with ceric sulphate. Recovery of galactose added to cerebrosides averaged 99·8 = 2·23 per cent., and of cerebrosides added to brain lipide extracts 98·0 = 1·87 per cent. A. P. LOTHROP (Chem. Abstr.).

Further Biochemical Studies in Mongolism. Bixby, Emily M. [*Am. J. Mental Deficiency*, **45**, 201–6 (1940); *cf. C.A.*, **34**, 6352[1].]

No definite deviations from normal in the concentration of Cl, Na, protein, N.P.N., glucose or cholesterol in the blood or blood serum were observed in Mongoloids from 3 to 26 years old. Hypercholesterolemia was found in a mother of 3 children all of whom were Mongols, and a causal relationship is suggested. Bixby's previous finding of a delayed glycemic response in Mongols in the glucose-tolerance test is confirmed. WARREN M. SPERRY (Chem. Abstr.).

Changes in Blood Urea Following Hypothalamic Lesions. Miller, H. R., Krueger, E., Alper, J. M., and Spiegel, E. A. [*Proc. Soc. Exptl. Biol. Med.*, **48**, 478–91 (1941).]

In cats, lesions of the anterior hypothalamic area and preoptic region caused a rise in blood urea. No change occurred in the level of other blood constituents. In 3 cats surviving beyond 18 days the blood-urea level returned to normal. L. E. GILSON (Chem. Abstr.).

Static and Some Other Reactions in Cats after Removal of the Neopallium. Lobach, L. A. M., and Diercks, A. L. [*Arch. néerl. Physiol.*, **25**, 212–218 (1941).]

After removal of the cerebral cortex the Stutz reflex is more readily elicited and more pronouncedly manifested in cats than under normal conditions. Also, there are marked differences

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in independence of the reactions which play weighty roles in the statics and movements of these animals. C. P. STONE (Psychol. Abstr.).

Effects of Reduced Atmospheric Pressures on the Electroencephalogram. Lyman, R. S., Carlson, W. A., and Benson, O. O., jun. [*J. aviat. Med.*, **12**, 115–125 (1941).]

Brain waves were obtained during pseudo-ascents to high altitudes in a decompression chamber. Little change except for periods of general flattening of the brain waves were seen during the ascent as the subject continued to inhale oxygen through a mask. "Changes in the brain waves occurred after taking off the oxygen mask at 18,000 ft. simulated altitude. These changes seldom had sharp times of onset or ending and were somewhat variable in constancy and degree in any one individual. One subject with some (epileptoid) abnormalities in his electro-encephalogram and one deteriorated epileptic showed no greater difference between brain waves at ground level and at 18,000 and 15,000 ft. respectively—before, during or after removal of the oxygen mask—than occurred in quite healthy subjects."

C. PFAFFMANN (Psychol. Abstr.).

Cerebrospinal Fluid Protein Values Determined by Tyrosine Equivalent Method. Marron, T. U. [*Am. J. Med. Sci.*, **202**, 330–3 (1941).]

The average value of spinal fluid protein in 100 normal persons was found to be 32 mgm. per cent., with an upper limit of 46 mgm. per cent., by use of the method of Johnston and Gibson (*C.A.*, **32**, 2557[8]). Males averaged 33 and females 28 mgm. per cent. The ranges for pathological values agree well with generally accepted figures. Body dehydration does not appreciably affect the spinal fluid protein concentration. FERRIN B. MORELAND (Chem. Abstr.).

Spinal Fluid Changes Following Cerebrovascular Accidents. Brown, Charles H. [*Med. Record*, **153**, 198–200 (1941).]

The spinal fluid was studied in 75 patients following cerebrovascular accidents. Nine of the spinal fluids were bloody or xanthochromic. Five cases showed an increased cell count after cerebral hemorrhage; the cells present were chiefly lymphocytes. The Pandy reaction was negative in 26 cases, borderline in 16 and positive in 9; the positive and borderline reactions are probably dependent upon an increase of, or a change in the type of, protein following the accident. The mean protein value in 51 cases (excluding those with syphilis or bloody spinal fluid) was 62·6 mgm. per 100 c.c.; values as high as 180 mgm. per 100 c.c. were found in 3 cases. The mean sugar value in 51 cases was 74·6 mgm. per 100 c c. Changes in the Lange and mastic reactions can often be expected after a cerebrovascular accident. These probably depend on an increase of, or a change in, the nature of the protein associated with softening and degenerating nervous tissue. Neither these changes nor the increase in protein are indicative of syphilis unless there is further evidence from the blood or spinal fluid serological reactions.

RUTH BERGGREN (Chem. Abstr.).

Obstetrical and Gynaecological Study on the Amount of Vitamin C in the Cerebrospinal Fluid. I. *Obstetrical Investigation on the Amount of Vitamin C in the Cerebrospinal Fluid. Wakabayasi, K., Kikuti, T., Adati, H., Takenaka, T., and Takeguti, M.* [*Japan. J. Obstet. Gynecol.*, **24**, No. 1, 20–1 (1941).]

The vitamin C (1) in the cerebrospinal fluid determinated by the method of Jazler and Niederberger (*C.A.*, **30**, 6404[9]) was lower in pregnant women (3rd to 10th month) than in non-pregnant patients. (1) in primiparas was slightly lower, though not greatly different from (1) in multiparas. (1) did not increase or decrease definitely as the pregnancy advanced. (1) was highest in winter, lowest in summer and about the same in the fall and spring; i.e. (1) was low and did not vary considerably between April and October, then increased rapidly in November, and remained high until February. This rise in (1) can probably be attributed to the increased consumption of vegetables and fruits, especially oranges, which are abundant in the fall and winter. There was no correlation between (1) and the age of the pregnant women.

II. *Gynaecological Investigation on the Amount of Vitamin C in the Cerebrospinal Fluid.* [*Ibid*].

22-whole (1) was low when the uterine myoma or ovarian cyst was large, i.e. some relation apparently exists between (1) and the size of the tumour, although the reason for this is not known. In uterine cancer (1) diminished as the degree of infiltration increased. No definite relation was noted between (1) and the age of the patient. (1) in nulliparas did not differ greatly from that in multiparas, although in nulliparas with uterine cancer or ovarian cyst (1) was higher than in multiparas with these pathological conditions. (1) was comparatively low from the summer to the middle of the fall, reaching a minimum in September; (1) increased quite rapidly in November and remained high during December, January and February. This agrees with the observations above. RUTH BERGGREN (Chem. Abstr.).

Determination of Iron in the Cerebrospinal Fluid. Vonkennel, J., and Tilling, Th. [*Klin. Wochschr.*, **19**, 177–81 (1940).]

The Fe in the cerebrospinal fluid (1) was determinated by the following method, a slight modification of the Heilmeyer procedure (*cf.* Heilmeyer and Plötner, *C.A.*, **31**, 5426[3]): To 2 c.c. (1) in a test tube add 1 c.c. HCl, shake well and let stand 10 minutes. Add 2 c.c. 20 per cent

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CCl_3CO_2H, shake vigorously and let stand 10 minutes longer. Centrifuge 15 minutes, then filter through an Fe-free filter paper (Schleicher and Schüll No. 589, diameter 7 cm.). To 1 c.c. filtrate (= 0·4 c.c. 1) in a graduated test tube add p-nitrophenol, neutralize with NH_3 and acidify slightly with 0·5 NH_2SO_4. Add 1 drop hydroquinone solution and 1 drop o-phenanthroline solution. Let the colour develop and determine photometrically. The average of 70 determinations of Fe in (1) in various diseases was 32γ per cent. (limits 15–51γ per cent.). No difference in the Fe contents was found between lumbar and suboccipital (1). The average Fe content was 32γ per cent. in normal (1) and 28γ per cent. in pathological (1) in chronic or subacute infections. This difference is probably accidental. The variation in Fe content was the same in both instances. The (1) Fe was at the most about one quarter of the serum Fe. In 4 out of 5 patients with brain tumours the (1) Fe was relatively low (15–24γ per cent.), while in 4 cases of acute meningitis and poliomyelitis it was relatively high (35–44γ per cent.). The (1) Fe was 33γ per cent. (15–46γ per cent.) in men and 29γ per cent. (15–48γ per cent.) in women. This is in distinct contrast to the serum Fe, which is markedly higher in men. During more than 1 month the (1) Fe showed very little variation (29–38γ per cent.) in 1 case of serous meningitis. In spite of the marked decrease in serum Fe in malaria, the (1) Fe remained constant. With an intact blood-1 barrier, the (1) Fe is independent of the serum Fe. The (1) Fe was normal (32γ per cent.), however, in 1 case of paralysis, in which a disturbance of the blood-(1) barrier was assumed. RUTH BERGGREN (Chem. Abstr.).

Injection of Heavy Water into the Cerebrospinal Fluid Space. *Stern, Karl, and Dancey, T. E.* [*Proc. Soc. Exptl. Biol. Med.*, **48**, 619–20 (1941).]

In catatonic patients replacement of 10–22 c.c. of spinal fluid by D_2O saline solution produced a marked cellular reaction in the fluid but no appreciable change in the psychiatric or neurological picture, nor in the pulse rate, temperature, respiration, blood pressure, basal metabolic rate, blood cholesterol, glucose tolerance, or kidney function.

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

Heat Flocculation Reaction of Cerebrospinal Fluid of Patients with Anterior Acute Poliomyelitis. *Perelli, Corrado.* [*Minerva med.*, **1**, 363–5 (1941).]

A study of 13 cases by the $CaCl_2$-heat flocculation method of Cortese (*Boll. ist. sieroterap. milan.*, **9**, 1940), of which 4 were controls. The method is valuable for the diagnosis, and perhaps will give information valuable in the treatment of poliomyelitis. Details of the method are given. WM. F. BRUCE (Chem. Abstr.).

Cerebrospinal Fluid Findings in Swineherd's Meningitis. *Wehrlin, H.* [*Helv. Med. Acta*, **6**, 605–9 (1939).]

The typical changes in the cerebrospinal fluid are apparent in the second stage of the fever and are characterized by increase of lymphocytes. The albumin and pressure change are not characteristic and may be lacking altogether. In the first stage of light cases the fluid usually shows no change, while in severe cases the albumin findings are strongly pathological, but there is only an insignificant increase of cells. REBECCA YABLONSKY (Chem. Abstr.).

The Influence of Growth and Myelination on the Deposition and Metabolism of Lipides in the Brain. *Waelsch, Heinrich, Sperry, Warren M., and Stoyanoff, V. A.* [*J. Biol. Chem.*, **140**, 885–97 (1941); *cf. C.A.*, **34**, 6684′.]

Up to 40 days of age, growth is quantitatively a more important factor than myelination in the deposition of fat acids in rat brain. The amount of unsaponifiable lipides deposited in the process of myelination is equal to that deposited as a result of growth at about 20 days of age and exceeds it thereafter. The rate of regeneration of lipides in the brain is highest during the period immediately following birth, when the demand for lipides for new deposition is also greatest. The young rats were given D_2O for 4 days before being killed. The proportion of H atoms derived from body H_2O in the unsaponifiable lipides and fat acids, as measured by the D concentration, was highest in the youngest animals (4 days old) and declined steadily up to 40 days of age. This is in large part due to the fact that with increasing age the absolute amount of lipides deposited in a 4-day period comprises a smaller and smaller proportion of the total lipides even during myelination, and also that there is a decrease in the rate of lipide regeneration in the brain with increasing age. A. P. LOTHROP (Chem. Abstr.).

Demonstration of Myelolytic Substances in Disseminated Sclerosis. *Weil, Arthur, and Heilbrunn, Gert.* [*Proc. Soc. Exptl. Biol. Med.*, **48**, 233–7 (1941).]

A substance which acted destructively on the myelin sheaths of rat spinal cord was found in urines of patients with disseminated sclerosis. It was more frequently observed during periods of exacerbation than during periods of remission. Similar myelolytic substances were also observed in cases of post-encephalitic parkinsonism, but they were absent in syphilis and in other diseases of the central nervous system. The myelolytic substance was isolated from the concentrated urine by extraction with dioxane. The method is described.

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

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Pathological Variations in Blood and Spinal Fluid Pyruvic Acid. Bueding, Ernest, Wortis, Herman, Stern, Marvin, and Esteronne, Dorothy. [*J. Clin. Investigation*, **21**, 85–9 (1942).]

Normal blood pyruvic acid levels, determined as described previously (*C.A.*, **35**, 7481[5]), ranged from 0·77 to 1·23 mgm. per cent. (average 1·02). Hyperpyruvaemia was noted in cases of " acute " peripheral neuropathy and in about two-thirds of the cases associated with hyperpyrexia. The diagnostic significance of the determination is discussed.

J. B. BROWN (Chem. Abstr.).

Peripheral Nervous Lesions Produced in the Pigeon by Alimentary Disequilibrium Caused by Galactose and Lactic Acid. Bertrand, Ivan, and Lecoq, Raoul. [*Bull. sci. pharmacol.*, **48**, 251–60 (1941).]

A diet containing 66 per cent. galactose causes in the pigeon nervous lesions leading to a fragmentation of the cylinder axis. If a diet containing 10 per cent. lactic acid is given, the same destruction of the cylinder axis appears concomitantly with a drop in the alkali reserve. The symptoms are milder if sucrose is added to the diet. The picture differs fundamentally from the nervous changes produced by vitamin B deficiency. A. E. MEYER (Chem. Abstr.).

The Possibilities of the Passage of Substances from the Blood to the Central Nervous System. Broman, Torc. [*Acta Psychiat. Neurol.*, **16**, 1–25 (1941).]

Acid dyes and certain toxins usually fail to pass from the blood into the central nervous system. This is not entirely a permeability effect, and indicates the presence of a " blood-brain-barrier " localized in the walls of the cerebral vessels. Positively charged dyes and certain drugs are able to pass, but bile pigments are not. The existence of a barrier between the blood and the cerebrospinal fluid is more doubtful. M. L. C. BERNHEIM (Chem. Abstr.).

Convulsive Mechanism in Idiopathic Hypoparathyroidism. McQuarrie, Irvine, Hansen, A. E., and Ziegler, M. R. [*J. Clin. Endocrinol.*, **1**, 789–98 (1941); *cf. C.A.*, **35**, 6648[8].]

The convulsive reactivity of a patient suffering from idiopathic hypoparathyroidism was investigated under a variety of experimental conditions. Prolonged use of a diet with a low mineral content increased the convulsive tendency. A diet with a relatively high mineral content, particularly as regards Ca and P, decreased the convulsive reactivity, even when the serum Ca and inorganic P showed no significant increases over values found during low-mineral diet periods. During periods of low mineral intake typical convulsions could be induced at will by sustained, post-pituitary antidiuresis and by the intravenous administration of typhoid vaccine without significant changes in the serum Ca and inorganic P. On the other hand, during periods of high mineral intake convulsions could not be induced by these procedures, even when the levels of serum Ca and inorganic P were essentially the same as those for the low-mineral periods. Active tetany and even convulsions could be produced by short periods of voluntary hyperventilation during the period of low, but not during the period of adequate, mineral intake. Administration of parathyroid extract for several days preceding and during a prolonged period of pitressin antidiuresis prevented induction of convulsions by these procedures, even when the mineral content of the diet was low. Artificial fever during a period of low mineral intake did not provoke tetany, but resulted in an excessive loss of water by way of the skin. Fever cannot be regarded as the factor responsible for the increase in convulsive tendency which follows the development of acute infection in the tetanic subject. Addition of sufficient $NaHCO_3$ to the high mineral diet to give its ash the same preponderance of alkali over acid as that calculated for the low mineral diet did not lower its protective value measurably. The increase in convulsive reactivity resulting from pitressin antidiuresis during periods of low mineral intake was much less marked when sufficient NaCl was given to prevent or greatly lessen the degree of dilution of the extracellular body fluids caused by the extra water retention period. Conclusion: Other factors, in addition to a diminished concentration of Ca ions in the extracellular body fluids, are involved in the causation of convulsions in hypoparathyroidism. The possible nature of such factors is discussed. FELIX SAUNDERS (Chem. Abstr.).

2. Pharmacology and Treatment.

The Effects of Insulin, Metrazole and Electric Shock on Blood Pyruvate, Lactate and Glucose. Elliott, K. A. C., Rivers, T. D., Elliott, F. H., and Platt, B. [*J. Lab. Clin. Med.*, **26**, 1928–34 (1941).]

Neither insulin in small or large doses nor metrazole nor electric shock treatments have any specific effect on the level of blood pyruvate and probably none on the lactate level in human subjects. The concentration of these substances bore no relation to the blood-sugar level nor to the condition of brain function. Blood pyruvate and lactate rose and fell together, and the rise seemed to be the result only of muscular activity. In metrazole and electric shock the rise of these constituents was very high. Curare decreased the effect of metrazole convulsions on blood lactate, pyruvate and glucose. Changes in blood pyruvate were less marked than those of lactate. Injections of large doses of vitamin B_1 into patients in insulin stupor produced no definite effect on blood pyruvate or glucose during the experimental period.

HOWARD W. ROBINSON (Chem. Abstr.).

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Effect of Metrazole on the Blood Pressure of Man and Dog. *Woodbury, R. A., Hamilton, W. F., Cleckly, H. M., and Volpitto, Perry P.* [*J. Pharmacol.*, **73**, 431–40 (1941).]

In man convulsant doses of metrazole increase arterial pressure by an average maximum of 100 mm. Hg. Vasoconstriction plays only a minor part. The main rise is limited to the duration of, and is produced by, the contractions of the skeletal muscles. The sympathetic and parasympathetic nervous systems are excited and their effects on arterial pressure and heart rate are mingled. In unanaesthetized dogs subconvulsant and convulsant doses produce large prolonged elevations in arterial pressure. In contrast to the effect in man, these are produced by vasoconstriction. The convulsions themselves increase arterial pressure only 20–30 mm. Hg. There is little effect on the pressure gradient through the pulmonary circulation.

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

Changes in Susceptibility to the Convulsant Action of Metrazole. *Sacks, Jacob, and Glaser, Nathan M.* [*J. Pharmacol.*, **73**, 289–95 (1941).]

Rats can be made tolerant to the convulsant action of metrazole by giving injections of adequate doses on alternate days. Animals in which such a tolerance has been developed have a latent increased susceptibility produced by the convulsions. A series of metrazole convulsions at such intervals that tolerance does not develop leads to a state of increased susceptibility to the convulsant action of the drug. Tolerance can be produced in such susceptible animals by giving adequate doses on alternate days. L. E. Gilson (Chem. Abstr.).

Clinical Experimental Observations on the Intracarotid Injection of Cardiazole and Pernocton. *Yasukoti, G.* [*Hukuoka Acta Med.*, **33**, 1135–44 (*in Japanese*) (*in German*, 85) (1940).]

A general convulsion which was typically epileptic and bilateral appeared constantly immediately after the intracarotid injection of 50 mgm. metrazole (30 cases). The injection of 100 mgm. metrazole into the vertebral artery during surgical operation for lymphadenitis never induced the typical epileptic convulsion but only tonic rigidity of the whole body with nystagmus and fibrillary twitchings (3 cases). Injection of 50 mgm. pernocton into the right common carotid resulted in a transitory contralateral, i.e. central, disturbance in the power of motility and perception which was abnormal only in the eye muscles; internal ophthalmoplegia was ipsilateral and external ophthalmoplegia was never evident. Consciousness was not affected (10 cases). Upon injection of pernocton into the left common carotid transitory aphasic symptoms also appeared (10 cases). Ruth Berggren (Chem. Abstr.).

Metabolism in Perfused Dog Head during Sodium Pentobarbital Depression and Metrazole Stimulation. *Handley, C. A., Sweeney, H. Morrow, and Brookman, Bruce T.* [*Proc. Soc. Exptl. Biol. Med.*, **48**, 670–2 (1941).]

During Na pentobarbital depression there is a measurable reduction in both O and glucose utilization. Metrazole administered during this depression restores the utilization of both to about the control levels or beyond. L. E. Gilson (Chem. Abstr.).

Inhibition of Brain Oxidations by a Convulsant Barbiturate. *Fuhrman, F. A., Martin, A. W., and Dille, J. M.* [*Science*, **94**, 421–2 (1941).]

Na 1, 3-dimethylbutylethyl barbiturate (1) inhibits glucose oxidation of rat brain slices. The concentrations are 1 and 2 mgm. per cent. for convulsant and lethal doses respectively. A comparable inhibition of rat brain oxidation is observed with equivalent concentrations of Na pentobarbital, but when administered to intact animals in doses equivalent to those of (1) it produces depression. Felix Saunders (Chem. Abstr.).

The Effect of Certain Antiseptics on the Respiration of Brain Tissues in vitro. *Manifold, M. C.* [*Brit. J. Exptl. Path.*, **22**, 111–25 (1941).]

The glucose and pyruvic acid oxidation systems of brain are inhibited by the acridine derivatives. Acriflavine is the most effective, followed in order by azochloramide, soluseptasine, proflavine sulphate and 2, 7-diaminoacridine HCl. These results agree with those obtained by histological methods, and show that the effect on enzyme systems is a useful method for investigating the problems of antiseptics. M. L. C. Bernheim (Chem. Abstr.).

Treatment of Post-encephalitic Parkinson's Syndrome with Ascorbic Acid. *Wirth, J.* [*Deut. med. Wochschr.*, **48**, 1328–30 (1940).]

Parkinsonism is being successfully treated with ascorbic acid. It prevents tissue anoxia due to deficiency of oxygen in the blood by accelerating oxido-reduction.

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

Sodium Diphenylhydantoin and Experimental Epilepsy. (1) *Action on the Convulsions Produced by Sudden Chilling or Direct Chemical Excitation of the Spinal Cord of the Frog.* / *Moussatché, H.* [*Rev. brasil. biol.* (*Rio de Janeiro*), **1**, 301–4 (1941); *Science*, **95**, 106 (1942).]

Injection of 0·10–0·15 gm./kgm. into the ventral lymph sac prevents the production of epileptic convulsions by the above agencies. Other spinal reflexes and general nervous activity are not affected. L. E. Gilson (Chem. Abstr.).

Carbohydrate Metabolism in Narcosis. *Fedorov, I. I.* [*Bull. biol. méd. exptl. U.R.S.S.*, **9**, 368–71 (1940) (*in German*).]

The administration *per os* of 2 gm./kgm. body weight of glucose in 10 per cent. solution to

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rabbits, and the intravenous injection into dogs and cats during Et_2O narcosis causes a strong hyperglycaemia until the narcosis terminates, after which the blood-sugar values return to normal. In cases of high blood-sugar values during the first hour of narcosis the difference in sugar values between arterial and venous blood decreases and eventually becomes negative, venous blood sugar being higher than arterial sugar. Blood lactic acid increases during narcosis either with or without sugar administration. The intravenous injection of 5 units/kgm. body weight of insulin into dogs and rabbits during Et_2O narcosis caused no decrease in blood sugar until the narcosis terminated. The injection of adrenaline during narcosis caused a greater hyperglucaemia than under normal conditions. The lactic acid values were similar to those found after sugar administration in narcosis. S. A. KARJALA (Chem. Abstr.).

A Statistical Study of Temperature in Hypoglycemic Coma. Tietz, Esther Bogen, Nadler, Cecile, and Klemperer, Edith. [*J. Lab. Clin. Med.*, **27**, 11–15 (1941); *cf. C.A.*, **34**, 3370⁴.]

The mean rectal temperatures of schizophrenic patients during insulin coma over a period of 45 days show a trend upward in patients showing recovery, but no rise in those patients unimproved by the treatments. This rise in temperature is not due to the decrease in the insulin dosage required to produce shock. H. W. ROBINSON (Chem. Abstr.).

Effect of Acetylcholine on the Spinal Cord. Beritov, I. S., and Bakuradze, A. [*J. Physiol.* (*U.S.S.R.*), **28**, 3–16 (*in English*, 16–17) (1940); *cf. C.A.*, **34**, 1428; **35**, 7544⁷.

Acetylcholine (1) 1 : 500 heightens reflex reactions by increasing the excitability of cell elements of the cord, and at lower concentrations inhibits the reflexes by activating the dendrites. (1) does not excite motor reactions while nerve impulses do, and it augments excitability and elicits a general inhibition, which does not last over one minute. After a brief interruption these phenomena cannot be reproduced for several minutes. This indicates that (1) is not a transmitter of excitation in the central nervous system, nor does it cause inhibition in the latter.

Effect of Mechanical Stimulation on the Spinal Cord. [*Ibid.*, 18–23 (*in English*, 24).]

Mechanical stimulation has the same effect on the spinal cord as stimulation with (1). T. LAANES (Chem. Abstr.).

Action of Drugs Beneficial in Myasthenia Gravis. (1) Effect of Prostigmine and Guanidine on Serum and Muscle Potassium. Thompson, Vincent, and Tice, Arnold. [*J. Pharmacol.* **73**, 455–62 (1941).]

Prostigmine and guanidine produce a decrease in serum K in dogs and rats and an increase in muscle K in rats. The signs of prostigmine intoxication in dogs parallel more closely the depression of serum K than they do the cholinesterase inhibition. The serum K response to prostigmine in patients with myasthenia is not constant. The duration of relief symptoms follows more closely the change in serum K (whether raised or lowered) than it does the decrease in cholinesterase activity. L. E. GILSON (Chem. Abstr.).

Hypnotics and Blood-sugar-regulating Centers. Rosenthal, F. E. [*J. Pharmacol.*, **73**, 446–54 (1941).]

Picrotoxin hyperglucemia is regularly suppressed by many aliphatic hypnotics, such as chloral hydrate, paraldehyde, barbiturates and chloretone, because of a depressant action on the blood-sugar-regulating centres. Urethan even in large doses cannot annul the central glycogenolytic action of picrotoxin. Hypnotic action can occur without the sugar centers being involved. Depression of the sugar centers by hypnotics can take place independently of any action on the sleep mechanism. They can be depressed by small doses of hypnotics which do not produce sleep, and depression of the sugar centers can outlast awakening by many hours. Barbiturates have a stronger depressing action on the sugar centers than chloral hydrate and paraldehyde. Rabbit experiments are described. L. E. GILSON (Chem. Abstr.).

Effect of Calcium and Strontium Salts on the Action of Sodium Pentobarbital. Cole, Versa V. [*J. Pharmacol.*, **73**, 335–42 (1941); *cf. C.A.*, **35**, 18666, 7.

Both Ca and Sr acetate increased the duration of Na pentobarbital anesthesia in rats when injected intraperitoneally. Ca acetate increased the duration of anesthesia when given intravenously; Sr acetate had little or no effect. Ca acetate was more toxic than Sr acetate. Na pentobarbital decreased the toxicity of Sr acetate, but Sr acetate did not decrease the toxicity of Na pentobarbital. L. E. GILSON (Chem. Abstr.).

An Analysis of the Action of Bulbocapnine in Spinal Animals. Ward, James W., and Kolb, Lawrence C. [*J. Pharmacol.*, **73**, 375–82 (1941).]

Bulbocapnine affects primarily the afferent portions of the nervous system or some synapse in the cord. On the neuro-muscular activity of spinal cats it produced increase in tone in the flexor and extensor muscles of the hind legs. The extensors were primarily affected. In the unilaterally deafferented spinal cat bulbocapnine caused resistance to passive flexion but little or no resistance to passive traction. No changes in resistance to passive movements of the hind legs was produced in the animal with spinal cord removed or in the spinal animal with bilateral dorsal root section. L. E. GILSON (Chem. Abstr.).

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A Method for the Rapid Isolation and Spectrographic Measurement of Cocaine from Brain Tissue *Strait, L. A., Aird, A. B., and Weiss, S.* [*J. Pharmacol.*, **73**, 363–74 (1941).]

After extraction and separation from interfering substances the cocaine is determined through its ultraviolet absorption at 227mμ.; as little as 0·05 mgm. in 12 gm. brain tissue can be determined with satisfactory accuracy. L. E. GILSON (Chem. Abstr.).

The Inhibition of Brain Oxidations by a Convulsant Barbiturate. Fuhrman, F. A., Martin, A. W., and Dille, J. M. [*Science*, **94**, 421 (1941).]

Some hold to the view that narcosis is essentially an inhibition of brain glucose oxidation, as it has been shown that barbiturates and other narcotics depress the *in vitro* oxidation of glucose by brain slices; thus they differentiate narcotics from tissue poisons which inhibit oxidations in general. In chemical structure sodium 1, 3-dimethyl-butyl-ethyl barbiturate differs from sodium pentobarbital but little, but in contrast to the depressant action of pentobarbital it produces convulsions in warm-blooded animals. In view of the inhibitory action of pentobarbital on glucose oxidation by brain slices it seemed possible to employ this closely related compound, with its strikingly different action in the intact animal, to test this theory of narcosis. The results of the authors' experiments show that sodium 1, 3-dimethyl-butyl-ethyl barbiturate is effective in producing inhibition of glucose oxidation of rat brain slices. Both compounds inhibit brain glucose oxidation, but upon the intact animal the effect of one is to produce convulsions whereas the other acts as a depressant; the results obtained are thus not explained by the theory of narcosis. F. A. MOTE (Psychol. Abstr.).

The Effect of Thiamine on the Residual Neural Disturbances of Treated Pernicious Anemia. Zillhardt, J. C., MacLean, Keith, and Murphy, William P. [*Ann. Internal Med.*, **15**, 33–43 (1941).]

The administration of thiamine chloride seems to have a beneficial effect on those residual neural symptoms of pernicious anemia that remain stationary in spite of intensive and persistent anti-pernicious anemia therapy. Intramuscular administration of 3,000 international units of thiamine twice a week was more effective than 950 units per mouth twice daily. The maximum final effect occurred within two months and further administration had no effect.

JOHN T. MYERS (Chem. Abstr.).

The Effect of Vitamin B Complex on the Residual Neural Disturbances of Treated Pernicious Anemia. Zillhardt, J. C., Howard, Isabel, and Murphy, William P. [*Ann. Internal. Med.*, **15**, 44 (1941).]

Vitamin B complex did not have the same beneficial effect as did thiamine chloride.

JOHN T. MYERS (Chem. Abstr.).

The Influence of the Direct Injection of Tetanus Antitoxin into the Cerebrospinal Fluid on the Development of Tetanus. Shtern, L. S., Yarmochkevich, A. I., Cherechnev, I. A., and Gromakovskaya, M. M. [*Bull. biol. méd. exptl. U.R.S.S.*, **9**, 355–7 (1940) (*in French*).]

The injection of tetanus antitoxin into the general circulation after the injection of toxin has no effect if the symptoms of tetanus have appeared. This failure to neutralize the toxin is due to the inability of the antibodies to penetrate the haemato-encephalic barrier. The injection into dogs of 1·5–3 times the amount required to neutralize twice the lethal dose of toxin *in vitro* was partially successful if the injection was made into the lateral cerebral ventricles immediately after the onset of tetanus, and completely successful if the injection was made simultaneously into the general circulation and the cerebral ventricles. When the injections were made 16–30 hours after the appearance of the first symptoms, all the animals died in 8–26 days. The injection of antitoxin into the general circulation immediately after the symptoms of tetanus appear, followed by injections into the cerebral ventricles 7 hours later, led to complete survival of the animals, but if the last injection is made 22 hours after the appearance of the symptoms all the animals die. S. A. KARJALA (Chem. Abstr.).

Ethyl Alcohol Therapy in Intoxications with Barbiturates and with Alcohol. Carratalá, Rogelio E. [*Semana méd.* (*Buenos Aires*), **2**, 1274–80 (1941).]

The intravenous M.L.D. was determinated in the dog for barbital as 0·5 gm. per kgm., phenobarbital 0·3, amytal 0·09, somniphene 0·24, dial 0·18 gm. The corresponding figures in the rabbit were 0·55, 0·15, 0·08, 0·3, 0·12; coma developed within 15 to 35 minutes. If the animals received an intravenous injection of 2 c.c. of 30 per cent. EtOH preceding the poisoning, the onset of the coma was delayed. Death was prevented by the alcohol in about 50 per cent. of the cases. Although EtOH has ultimately a narcotic and paralysing action similar to that of the barbiturates the small doses produce only the initial exciting effect. The result, however, is inferior to that obtained with heart stimulants and analeptics. The EtOH treatment in cases of human intoxication with barbiturates was disappointing The method of treatment with strychnine, picrotoxin and cardiazole is described. Chronic alcoholism in dogs produced by adding increasing doses of EtOH to the food was treated with daily intravenous injections of 3 gm. per kgm. of EtOH as 30 per cent. solution. After treatment the dogs refused food containing EtOH; before they had preferred it. Treatment of 14 patients with severe alcoholism was carried out with 60 to 80 c.c. daily of a 1 : 2 mixture of EtOH (95 per cent.) and 30 per cent. glucose. The symptoms were not relieved but the craving for EtOH subsided.

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

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